DIAGNOSTIC AND STATISTICAL MANUAL OF MENTAL DISORDERS

FIFTH EDITION TEXT REVISION

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AMERICAN PSYCHIATRIC ASSOCIATION

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American Psychiatric Association
800 Maine Avenue SW
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Maritza Rubio-Stipec, Sc.D.
David Shaffer, M.D.
Andrew E. Skodol II, M.D.
Susan E. Swedo, M.D.
B. Timothy Walsh, M.D.
Philip Wang, M.D., Dr.P.H. *(2007–2012)*
William M. Womack, M.D.
Kimberly A. Yonkers, M.D.
Kenneth J. Zucker, Ph.D.
Norman Sartorius, M.D., Ph.D., *Consultant*

APA Division of Research Staff on DSM-5

Darrel A. Regier, M.D., M.P.H., *Director, Division of Research*
William E. Narrow, M.D., M.P.H., *Associate Director*
Emily A. Kuhl, Ph.D., *Senior Science Writer; Staff Text Editor*
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Lisa H. Greiner, M.S.S.A., *DSM-5 Field Trials Project Manager*
Eve K. Moscicki, Sc.D., M.P.H., *Director, Practice Research Network*
S. Janet Kuramoto, Ph.D., M.H.S., *Senior Scientific Research Associate, Practice Research Network*

Amy Porfiri, M.B.A. *Director of Finance and Administration*
Jennifer J. Shupinka, *Assistant Director, DSM Operations*
Seung-Hee Hong, *DSM Senior Research Associate*
Anne R. Hiller, *DSM Research Associate*
Alison S. Beale, *DSM Research Associate*
Spencer R. Case, *DSM Research Associate*

Joyce C. West, Ph.D., M.P.P., *Health Policy Research Director, Practice Research Network*

Farifteh F. Duffy, Ph.D., *Quality Care Research Director, Practice Research Network*

Lisa M. Countis, *Field Operations Manager, Practice Research Network*

Christopher M. Reynolds, *Executive Assistant*

APA Office of the Medical Director

James H. Scully Jr., M.D.
Medical Director and CEO

Editorial and Coding Consultants

Michael B. First, M.D.

Maria N. Ward, M.Ed., RHIT, CCS-P

DSM-5 Work Groups

ADHD and Disruptive Behavior Disorders

David Shaffer, M.D.
Chair

F. Xavier Castellanos, M.D.
Co-Chair

Paul J. Frick, Ph.D., *Text Coordinator*

Glorisa Canino, Ph.D.

Terrie E. Moffitt, Ph.D.

Joel T. Nigg, Ph.D.

Luis Augusto Rohde, M.D., Sc.D.

Rosemary Tannock, Ph.D.

Eric A. Taylor, M.B.

Richard Todd, Ph.D., M.D. *(d. 2008)*

Anxiety, Obsessive-Compulsive Spectrum, Posttraumatic, and Dissociative Disorders

KATHARINE A. PHILLIPS, M.D.
Chair

Michelle G. Craske, Ph.D., *Text Coordinator*
J. Gavin Andrews, M.D.
Susan M. Bögels, Ph.D.
Matthew J. Friedman, M.D., Ph.D.
Eric Hollander, M.D. *(2007–2009)*
Roberto Lewis-Fernández, M.D., M.T.S.
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Murray B. Stein, M.D.
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Hans-Ulrich Wittchen, Ph.D.

Childhood and Adolescent Disorders

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Chair

Ronald E. Dahl, M.D.
E. Jane Costello, Ph.D. *(2007–2009)*
Regina Smith James, M.D.
Rachel G. Klein, Ph.D.
James F. Leckman, M.D.
Ellen Leibenluft, M.D.
Judith H. L. Rapoport, M.D.
Charles H. Zeanah, M.D.

Eating Disorders

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Chair

Stephen A. Wonderlich, Ph.D., *Text Coordinator*
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Rachel Bryant-Waugh, M.D.
Hans W. Hoek, M.D., Ph.D.
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Marsha D. Marcus, Ph.D.
James E. Mitchell, M.D.
Ruth H. Striegel-Moore, Ph.D.
G. Terence Wilson, Ph.D.
Barbara E. Wolfe, Ph.D., A.P.R.N.

Mood Disorders

JAN A. FAWCETT, M.D.
Chair

Ellen Frank, Ph.D., *Text Coordinator*
Jules Angst, M.D. *(2007–2008)*
William H. Coryell, M.D.
Lori L. Davis, M.D.
Raymond J. DePaulo, M.D.
Sir David Goldberg, M.D.
James S. Jackson, Ph.D.
Kenneth S. Kendler, M.D. *(2007–2010)*
Mario Maj, M.D., Ph.D.
Husseini K. Manji, M.D. *(2007–2008)*
Michael R. Phillips, M.D.

Trisha Suppes, M.D., Ph.D.
Carlos A. Zarate, M.D.

Neurocognitive Disorders

DILIP V. JESTE, M.D. (2007–2011)
Chair Emeritus

DAN G. BLAZER, M.D., PH.D., M.P.H.
Chair

RONALD C. PETERSEN, M.D., PH.D.
Co-Chair

Mary Ganguli, M.D., M.P.H., *Text Coordinator*
Deborah Blacker, M.D., Sc.D.
Warachal Faison, M.D. *(2007–2008)*
Igor Grant, M.D.
Eric J. Lenze, M.D.
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Perminder S. Sachdev, M.D., Ph.D.

Neurodevelopmental Disorders

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Gillian Baird, M.A., M.B., B.Chir., *Text Coordinator*
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Rosemary Tannock, Ph.D.
Fred Volkmar, M.D. *(2007–2009)*
Amy M. Wetherby, Ph.D.
Harry H. Wright, M.D.

Personality and Personality Disorders[1]

ANDREW E. SKODOL, M.D.
Chair

JOHN M. OLDHAM, M.D.
Co-Chair

Robert F. Krueger, Ph.D., *Text Coordinator*
Renato D. Alarcón, M.D., M.P.H.
Carl C. Bell, M.D.
Donna S. Bender, Ph.D.
Lee Anna Clark, Ph.D.
W. John Livesley, M.D., Ph.D. *(2007–2012)*
Leslie C. Morey, Ph.D.
Larry J. Siever, M.D.
Roel Verheul, Ph.D. *(2008–2012)*

Psychotic Disorders

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Chair

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Wolfgang Gaebel, M.D.
Raquel E. Gur, M.D., Ph.D.
Stephan H. Heckers, M.D.
Dolores Malaspina, M.D., M.S.P.H.
Michael J. Owen, M.D., Ph.D.

Susan K. Schultz, M.D.
Rajiv Tandon, M.D.
Ming T. Tsuang, M.D., Ph.D.
Jim van Os, M.D.

Sexual and Gender Identity Disorders

KENNETH J. ZUCKER, PH.D.
Chair

Lori Brotto, Ph.D., *Text Coordinator*
Irving M. Binik, Ph.D.
Ray M. Blanchard, Ph.D.
Peggy T. Cohen-Kettenis, Ph.D.
Jack Drescher, M.D.
Cynthia A. Graham, Ph.D.
Martin P. Kafka, M.D.
Richard B. Krueger, M.D.
Niklas Långström, M.D., Ph.D.
Heino F.L. Meyer-Bahlburg, Dr. rer. nat.
Friedemann Pfäfflin, M.D.
Robert Taylor Segraves, M.D., Ph.D.

Sleep-Wake Disorders

CHARLES F. REYNOLDS III, M.D.
Chair

Ruth M. O'Hara, Ph.D., *Text Coordinator*
Charles M. Morin, Ph.D.
Allan I. Pack, Ph.D.
Kathy P. Parker, Ph.D., R.N.
Susan Redline, M.D., M.P.H.
Dieter Riemann, Ph.D.

Somatic Symptom Disorders

JOEL E. DIMSDALE, M.D.
Chair

James L. Levenson, M.D., *Text Coordinator*
Arthur J. Barsky III, M.D.
Francis Creed, M.D.
Nancy Frasure-Smith, Ph.D. *(2007–2011)*
Michael R. Irwin, M.D.
Francis J. Keefe, Ph.D. *(2007–2011)*
Sing Lee, M.D.
Michael Sharpe, M.D.
Lawson R. Wulsin, M.D.

Substance-Related Disorders

CHARLES P. O'BRIEN, M.D., PH.D.
Chair

THOMAS J. CROWLEY, M.D.
Co-Chair

Wilson M. Compton, M.D., M.P.E., *Text Coordinator*
Marc Auriacombe, M.D.
Guilherme L. G. Borges, M.D., Dr.Sc.
Kathleen K. Bucholz, Ph.D.
Alan J. Budney, Ph.D.
Bridget F. Grant, Ph.D., Ph.D.
Deborah S. Hasin, Ph.D.
Thomas R. Kosten, M.D. *(2007–2008)*
Walter Ling, M.D.
Spero M. Manson, Ph.D. *(2007-2008)*
A. Thomas McLellan, Ph.D. *(2007–2008)*
Nancy M. Petry, Ph.D.
Marc A. Schuckit, M.D.
Wim van den Brink, M.D., Ph.D. *(2007–2008)*

DSM-5 Study Groups

Diagnostic Spectra and DSM/ICD Harmonization

STEVEN E. HYMAN, M.D.
Chair (2007–2012)

William T. Carpenter Jr., M.D.
Wilson M. Compton, M.D., M.P.E.
Jan A. Fawcett, M.D.
Helena C. Kraemer, Ph.D.
David J. Kupfer, M.D.
William E. Narrow, M.D., M.P.H.
Charles P. O'Brien, M.D., Ph.D.
John M. Oldham, M.D.
Katharine A. Phillips, M.D.
Darrel A. Regier, M.D., M.P.H.

Lifespan Developmental Approaches

ERIC J. LENZE, M.D.
Chair

SUSAN K. SCHULTZ, M.D.
Chair Emeritus

DANIEL S. PINE, M.D.
Chair Emeritus

Dan G. Blazer, M.D., Ph.D., M.P.H.
F. Xavier Castellanos, M.D.
Wilson M. Compton, M.D., M.P.E.
Daniel T. Mamah, M.D., M.P.E.
Andrew E. Skodol II, M.D.
Susan E. Swedo, M.D.

Gender and Cross-Cultural Issues

KIMBERLY A. YONKERS, M.D.
Chair

ROBERTO LEWIS-FERNÁNDEZ, M.D., M.T.S.
Co-Chair, Cross-Cultural Issues

Renato D. Alarcón, M.D., M.P.H.
Diana E. Clarke, Ph.D., M.Sc.
Javier I. Escobar, M.D., M.Sc.
Ellen Frank, Ph.D.
James S. Jackson, Ph.D.
Spero M. Manson, Ph.D. *(2007–2008)*
James P. McNulty, A.B., Sc.B.
Leslie C. Morey, Ph.D.
William E. Narrow, M.D., M.P.H.
Roger Peele, M.D.
Philip Wang, M.D., Dr.P.H. *(2007–2012)*
William M. Womack, M.D.
Kenneth J. Zucker, Ph.D.

Psychiatric/General Medical Interface

LAWSON R. WULSIN, M.D.
Chair

Ronald E. Dahl, M.D.
Joel E. Dimsdale, M.D.
Javier I. Escobar, M.D., M.Sc.
Dilip V. Jeste, M.D. *(2007–2011)*
Walter E. Kaufmann, M.D.
Richard E. Kreipe, M.D.
Ronald C. Petersen, M.D., Ph.D.
Charles F. Reynolds III, M.D.
Robert Taylor Segraves, M.D., Ph.D.

B. Timothy Walsh, M.D.

xx

Impairment and Disability

JANE S. PAULSEN, PH.D.
Chair

J. Gavin Andrews, M.D.
Glorisa Canino, Ph.D.
Lee Anna Clark, Ph.D.
Diana E. Clarke, Ph.D., M.Sc.
Michelle G. Craske, Ph.D.
Hans W. Hoek, M.D., Ph.D.
Helena C. Kraemer, Ph.D.
William E. Narrow, M.D., M.P.H.
David Shaffer, M.D.

Diagnostic Assessment Instruments

JACK D. BURKE JR., M.D., M.P.H.
Chair

Lee Anna Clark, Ph.D.
Diana E. Clarke, Ph.D., M.Sc.
Bridget F. Grant, Ph.D., Ph.D.
Helena C. Kraemer, Ph.D.
William E. Narrow, M.D., M.P.H.
David Shaffer, M.D.

DSM-5 Research Group

WILLIAM E. NARROW, M.D., M.P.H.
Chair

Jack D. Burke Jr., M.D., M.P.H.
Diana E. Clarke, Ph.D., M.Sc.

Helena C. Kraemer, Ph.D.
David J. Kupfer, M.D.
Darrel A. Regier, M.D., M.P.H.
David Shaffer, M.D.

Course Specifiers and Glossary

WOLFGANG GAEBEL, M.D.
Chair

Ellen Frank, Ph.D.
Charles P. O'Brien, M.D., Ph.D.
Norman Sartorius, M.D., Ph.D., *Consultant*
Susan K. Schultz, M.D.
Dan J. Stein, M.D., Ph.D.
Eric A. Taylor, M.B.
David J. Kupfer, M.D.
Darrel A. Regier, M.D., M.P.H.

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[1]The members of the Personality and Personality Disorders Work Group are responsible for the alternative DSM-5 model for personality disorders that is included in Section III. The Section II personality disorders criteria and text (with updating of the text) are retained from DSM-IV-TR.

Preface to DSM-5-TR

The American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition, Text Revision (DSM-5-TR), is the first published revision to DSM-5. This revised manual integrates the original published DSM-5 diagnostic criteria with modifications (mostly for clarity) for over 70 disorders, comprehensively updated descriptive text accompanying each of the DSM disorders based on reviews of the literature since the publication of DSM-5, and the addition of a new diagnosis, prolonged grief disorder, and symptom codes for reporting suicidal and nonsuicidal self-injurious behavior. These changes differ from the scope of the prior text revision, DSM-IV-TR, in which the updates were confined almost exclusively to the text, leaving the diagnostic criteria virtually unchanged. This edition also integrates all prior online updates made to DSM-5 after its publication in 2013, in response to usage, specific scientific advances, and ICD-10-CM coding adjustments through an iterative revision process. Consequently, DSM-5-TR is the product of three separate revision processes, each one overseen by separate (but overlapping) groups of experts: the development of the original DSM-5 diagnostic criteria and text by the DSM-5 Task Force, published in 2013; updates to the DSM-5 diagnostic criteria and text by the DSM Steering Committee, which has overseen the iterative revision process; and fully updated text overseen by the Revision Subcommittee.

The clinical and research understanding of mental disorders continues to advance. As a result, most of the DSM-5-TR disorder texts have had at least some revision since the 9 years from original publication in DSM-5, with the overwhelming majority having had significant revisions. Sections of the text that were most extensively updated were Prevalence, Risk and Prognostic Factors, Culture-Related Diagnostic Issues, Sex- and Gender-

Related Diagnostic Issues, Association With Suicidal Thoughts or Behavior, and Comorbidity. Also, for the first time ever, the entire DSM text has been reviewed and revised by a Work Group on Ethnoracial Equity and Inclusion to ensure appropriate attention to risk factors such as the experience of racism and discrimination, as well as to the use of non-stigmatizing language. For future periodic DSM-5-TR coding and other updates, see www.dsm5.org.

For benefit of reference in this manual, "DSM" refers generally to DSM as an entity, not specifying a particular edition (e.g., "Clinical training and experience are needed to use DSM for determining a clinical diagnosis."). "DSM-5" refers to the entire set of currently approved criteria sets, disorders, other conditions, and content officially published in May 2013. "DSM-5-TR" refers to approved text in this current volume. Although the scope of the text revision did not include conceptual changes to the criteria sets or to other DSM-5 constructs, the need to make changes in certain diagnostic criteria sets for the purpose of clarification became apparent in conjunction with the text updates made across the book. Because the conceptual construct of criteria is unchanged, the criteria sets in DSM-5-TR that had their origins in DSM-5 are still referred to as "DSM-5-criteria." The new diagnostic entity prolonged grief disorder is referred to as a DSM-5-TR disorder, because of its addition in this volume.

The development of DSM-5-TR was a tremendous team effort. We are especially indebted to the tireless efforts of Wilson M. Compton, M.D., M.P.E., and Daniel S. Pine, M.D., as DSM-5 Text Revision Subcommittee Vice Chairs, as well as the more than 200 experts from across our field who did the lion's share of the work in the preparation of the text revision. We would also like to thank Paul Appelbaum, M.D., Chair of the DSM Steering

Committee, along with the entire DSM Steering Committee, for their careful review of the text and criteria clarifications, and for making other helpful suggestions. Special gratitude goes to Ann M. Eng, DSM Managing Editor, for her timely shepherding of the DSM-5-TR development process from planning to completion and for her meticulous attention to detail, all critical to the success of this revision. We are grateful for the valuable

contributions and help of Nitin Gogtay, M.D., Chief of the American Psychiatric Association Division of Research and Deputy Medical Director; Diana E. Clarke, Ph.D., Managing Director of Research and Senior Research Statistician/Epidemiologist; and Lamyaa H. Yousif, M.D., Ph.D., M.Sc., Senior DSM Operations Manager and Research Associate. We are thankful for the leadership of John McDuffie, Publisher, American Psychiatric Association Publishing, and the work of the following editorial and production staff at American Psychiatric Association Publishing in bringing this important work to fruition: Greg Kuny, Managing Editor, Books; Tammy Cordova, Graphic Design Manager; Andrew Wilson, Director of Production; Judy Castagna, Assistant Director of Production Services; Erika Parker, Acquisitions Editor; Alisa Riccardi, Senior Editor, Books; and Carrie Y. Farnham, Senior Editor, Books. Finally, we also recognize with appreciation Saul Levin, M.D., M.P.A., CEO and Medical Director of the American Psychiatric Association, for his advocacy and support of this comprehensive text revision.

Michael B. First, M.D.
Revision Subcommittee Co-Chair and DSM-5-TR Editor
Philip Wang, M.D., Dr.P.H.
Revision Subcommittee Co-Chair
November 5, 2021

Preface to DSM-5

The American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) is a classification of mental disorders with associated criteria designed to facilitate more reliable diagnoses of these disorders. With successive editions over the past 60 years, it has become a standard reference for clinical practice in the mental health field. Since a complete description of the underlying pathological processes is not possible for most mental disorders, it is important to emphasize that the current diagnostic criteria are the best available description of how mental disorders are expressed and can be recognized by trained clinicians. DSM is intended to serve as a practical, functional, and flexible guide for organizing information that can aid in the accurate diagnosis and treatment of mental disorders. It is a tool for clinicians, an essential educational resource for students and practitioners, and a reference for researchers in the field.

Although this edition of DSM was designed first and foremost to be a useful guide to clinical practice, as an official nomenclature it must be applicable in a wide diversity of contexts. DSM has been used by clinicians and researchers from different orientations (biological, psychodynamic, cognitive, behavioral, interpersonal, family/systems), all of whom strive for a common language to communicate the essential characteristics of mental disorders presented by their patients. The information is of value to all professionals associated with various aspects of mental health care, including psychiatrists, other physicians, psychologists, social workers, nurses, counselors, forensic and legal specialists, occupational and rehabilitation therapists, and other health professionals. The criteria are concise and explicit and intended to facilitate an objective assessment of symptom presentations in a variety of clinical settings—inpatient,

outpatient, partial hospital, consultation-liaison, clinical, private practice, and primary care—as well in general community epidemiological studies of mental disorders. DSM-5 is also a tool for collecting and communicating accurate public health statistics on mental disorder morbidity and mortality rates. Finally, the criteria and corresponding text serve as a textbook for students early in their profession who need a structured way to understand and diagnose mental disorders as well as for seasoned professionals encountering rare disorders for the first time. Fortunately, all of these uses are mutually compatible.

These diverse needs and interests were taken into consideration in planning DSM-5. The classification of disorders is harmonized with the World Health Organization's *International Classification of Diseases* (ICD), the official coding system used in the United States, so that the DSM criteria define disorders identified by ICD diagnostic names and code numbers. In DSM-5, both ICD-9-CM and ICD-10-CM codes (the latter scheduled for adoption in October 2015) are attached to the relevant disorders in the classification.

Although DSM-5 remains a categorical classification of separate disorders, we recognize that mental disorders do not always fit completely within the boundaries of a single disorder. Some symptom domains, such as depression and anxiety, involve multiple diagnostic categories and may reflect common underlying vulnerabilities for a larger group of disorders. In recognition of this reality, the disorders included in DSM-5 were reordered into a revised organizational structure meant to stimulate new clinical perspectives. This new structure corresponds with the organizational arrangement of disorders planned for ICD-11 scheduled for release in 2015. Other enhancements have been introduced to promote ease of use across all settings:

- **Representation of developmental issues related to diagnosis.** The change in chapter organization better reflects a lifespan approach, with disorders more frequently diagnosed in childhood (e.g., neurodevelopmental disorders) at the beginning of the manual and disorders more applicable to older adulthood (e.g., neurocognitive disorders) at the end of the manual.

Also, within the text, subheadings on development and course provide descriptions of how disorder presentations may change across the lifespan. Age-related factors specific to diagnosis (e.g., symptom presentation and prevalence differences in certain age groups) are also included in the text. For added emphasis, these age-related factors have been added to the criteria themselves where applicable (e.g., in the criteria sets for insomnia disorder and posttraumatic stress disorder, specific criteria describe how symptoms might be expressed in children). Likewise, gender and cultural issues have been integrated into the disorders where applicable.

- **Integration of scientific findings from the latest research in genetics and neuroimaging.** The revised chapter structure was informed by recent research in neuroscience and by emerging genetic linkages between diagnostic groups. Genetic and physiological risk factors, prognostic indicators, and some putative diagnostic markers are highlighted in the text. This new structure should improve clinicians' ability to identify diagnoses in a disorder spectrum based on common neurocircuitry, genetic vulnerability, and environmental exposures.
- **Consolidation of autistic disorder, Asperger's disorder, and pervasive developmental disorder into autism spectrum disorder.** Symptoms of these disorders represent a single continuum of mild to severe impairments in the two domains of social communication and restrictive repetitive behaviors/interests rather than being distinct disorders. This change is designed to improve the sensitivity and specificity of the criteria for the diagnosis of autism spectrum disorder and to identify more focused treatment targets for the specific impairments identified.
- **Streamlined classification of bipolar and depressive disorders.** Bipolar and depressive disorders are the most commonly diagnosed conditions in psychiatry. It was therefore important to streamline the presentation of these disorders to enhance both clinical and educational use. Rather than separating the definition of manic, hypomanic, and major depressive episodes from the definition of bipolar I disorder, bipolar II disorder, and major depressive disorder as in the previous edition, we included all of the component criteria within the respective criteria for each disorder. This approach will facilitate bedside diagnosis and treatment of these important disorders. Likewise, the explanatory notes for differentiating bereavement and major depressive disorder will provide far greater clinical guidance than was previously provided in the simple bereavement exclusion criterion. The new specifiers of anxious distress and mixed features are now fully described in the narrative on specifier variations that accompanies the criteria for these disorders.
- **Restructuring of substance use disorders for consistency and clarity.** The categories of substance abuse and substance dependence have been eliminated and replaced with an overarching new category of substance use disorders—with the specific substance used defining

the specific disorders. “Dependence” has been easily confused with the term “addiction” when, in fact, the tolerance and withdrawal that previously defined dependence are actually very normal responses to prescribed medications that affect the central nervous system and do not necessarily indicate the presence of an addiction. By revising and clarifying these criteria in DSM-5, we hope to alleviate some of the widespread misunderstanding about these issues.

- **Enhanced specificity for major and mild neurocognitive disorders.** Given the explosion in neuroscience, neuropsychology, and brain imaging over the past 20 years, it was critical to convey the current state-of-the-art in the diagnosis of specific types of disorders that were previously referred to as the “dementias” or organic brain diseases. Biological markers identified by imaging for vascular and traumatic brain disorders and

specific molecular genetic findings for rare variants of Alzheimer’s disease and Huntington’s disease have greatly advanced clinical diagnoses, and these disorders and others have now been separated into specific subtypes.

- **Transition in conceptualizing personality disorders.** Although the benefits of a more dimensional approach to personality disorders have been identified in previous editions, the transition from a categorical diagnostic system of individual disorders to one based on the relative distribution of personality traits has not been widely accepted. In DSM-5, the categorical personality disorders are virtually unchanged from the previous edition. However, an alternative “hybrid” model has been proposed in Section III to guide future research that separates interpersonal functioning assessments and the expression of pathological personality traits for six specific disorders. A more dimensional profile of personality trait expression is also proposed for a trait-specified approach.
- **Section III: new disorders and features.** A new section (Section III) has been added to highlight disorders that require further study but are not sufficiently well established to be a part of the official classification of mental disorders for routine clinical use. Dimensional measures of symptom severity in 13 symptom domains have also been incorporated to allow for the measurement of symptom levels of varying severity across all diagnostic groups. Likewise, the WHO Disability Assessment Schedule (WHODAS), a standard method for assessing global disability levels for mental disorders that is based on the International Classification of Functioning, Disability and Health (ICF) and is applicable in all of medicine, has been provided to replace the more limited Global Assessment of Functioning scale. It is our hope that as these measures are implemented over time, they will provide greater accuracy and flexibility in the

clinical description of individual symptomatic presentations and associated disability during diagnostic assessments.

- **Online enhancements.** DSM-5 features online supplemental information. Additional cross-cutting and diagnostic severity measures are available online (www.psychiatry.org/dsm5), linked to the relevant disorders. In addition, the Cultural Formulation Interview, Cultural Formulation Interview—Informant Version, and supplementary modules to the core Cultural Formulation Interview are also included online at www.psychiatry.org/dsm5.

These innovations were designed by the leading authorities on mental disorders in the world and were implemented on the basis of their expert review, public commentary, and independent peer review. The 13 work groups, under the direction of the DSM-5 Task Force, in conjunction with other review bodies and, eventually, the APA Board of Trustees, collectively represent the global expertise of the specialty. This effort was supported by an extensive base of advisors and by the professional staff of the APA Division of Research; the names of everyone involved are too numerous to mention here but are listed in the Appendix. We owe tremendous thanks to those who devoted countless hours and invaluable expertise to this effort to improve the diagnosis of mental disorders.

We would especially like to acknowledge the chairs, text coordinators, and members of the 13 work groups, listed in the front of the manual, who spent many hours in this volunteer effort to improve the scientific basis of clinical practice over a sustained 6-year period. Susan K. Schultz, M.D., who served as text editor, worked tirelessly with Emily A. Kuhl, Ph.D., senior science writer and DSM-5 staff text editor, to coordinate the efforts of the work groups into a cohesive whole. William E. Narrow, M.D., M.P.H., led the research group that developed the overall research strategy for DSM-5, including the field trials, that greatly enhanced the evidence base for this revision. In addition, we are grateful to those who contributed so much time to the independent review of the revision proposals, including Kenneth S. Kendler, M.D., and Robert Freedman, M.D., co-chairs of the Scientific Review Committee; John S. McIntyre, M.D., and Joel Yager, M.D., co-chairs of the Clinical and Public Health Committee; and Glenn Martin, M.D., chair of the APA Assembly

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David J. Kupfer, M.D.
DSM-5 Task Force Chair
Darrel A. Regier, M.D., M.P.H.
DSM-5 Task Force Vice-Chair
December 19, 2012

DSM-5-TR Classification

Before each disorder name, ICD-10-CM codes are provided. Blank lines indicate that the ICD-10-CM code depends on the applicable subtype, specifier, or class of substance. For periodic DSM-5-TR coding and other updates, see www.dsm5.org.

Following chapter titles and disorder names, page numbers for the corresponding text or criteria are included in parentheses.

Note for all mental disorders due to another medical condition: Insert the name of the etiological medical condition within the name of the mental disorder due to [the medical condition]. The code and name for the etiological medical condition should be listed first immediately before the mental disorder due to the medical condition.

Neurodevelopmental Disorders (35)

Intellectual Developmental Disorders (37)

___.__	Intellectual Developmental Disorder (Intellectual Disability) (37) *Specify* current severity:
F70	Mild
F71	Moderate
F72	Severe
F73	Profound
F88	Global Developmental Delay (46)
F79	Unspecified Intellectual Developmental Disorder (Intellectual Disability) (46)

Communication Disorders (46)

Neurodevelopmental Disorders (35)

F80.2 Language Disorder (47)

F80.0 Speech Sound Disorder (50)

F80.81 Childhood-Onset Fluency Disorder (Stuttering) (51)

Note: Later-onset cases are diagnosed as F98.5 adult-onset fluency disorder.

F80.82 Social (Pragmatic) Communication Disorder (54)

F80.9 Unspecified Communication Disorder (56)

Autism Spectrum Disorder (56)

F84.0 Autism Spectrum Disorder (56)

Specify current severity: Requiring very substantial support, Requiring substantial support, Requiring support

Specify if: With or without accompanying intellectual impairment, With or without accompanying language impairment

Specify if: Associated with a known genetic or other medical condition or environmental factor (**Coding note:** Use additional code to identify the associated genetic or other medical condition); Associated with a neurodevelopmental, mental, or behavioral problem

Specify if: With catatonia (use additional code F06.1)

Attention-Deficit/Hyperactivity Disorder (68)

___.__ Attention-Deficit/Hyperactivity Disorder (68)

Specify if: In partial remission

Specify current severity: Mild, Moderate, Severe

Specify whether:

F90.2 Combined presentation

F90.0 Predominantly inattentive presentation

F90.1 Predominantly hyperactive/impulsive presentation

F90.8 Other Specified Attention-Deficit/Hyperactivity Disorder (76)

F90.9 Unspecified Attention-Deficit/Hyperactivity Disorder (76)

Specific Learning Disorder (76)

___.__ Specific Learning Disorder (76)

Specify current severity: Mild, Moderate, Severe

Specify if:

F81.0 With impairment in reading (specify if with word reading accuracy, reading rate or fluency, reading comprehension)

F81.81 With impairment in written expression (specify if with spelling accuracy, grammar and punctuation accuracy, clarity or organization of written expression)

F81.2 With impairment in mathematics (specify if with number sense, memorization of arithmetic facts, accurate or fluent calculation, accurate math reasoning)

Motor Disorders (85)

F82 Developmental Coordination Disorder (85)

F98.4 Stereotypic Movement Disorder (89)

Specify if: With self-injurious behavior, Without self-injurious behavior

Specify if: Associated with a known genetic or other medical condition, neurodevelopmental disorder, or environmental factor

Specify current severity: Mild, Moderate, Severe

Tic Disorders

F95.2 Tourette's Disorder (93)

F95.1 Persistent (Chronic) Motor or Vocal Tic Disorder (93)

Specify if: With motor tics only, With vocal tics only

F95.0 Provisional Tic Disorder (93)

F95.8 Other Specified Tic Disorder (98)

F95.9 Unspecified Tic Disorder (98)

Other Neurodevelopmental Disorders (99)

F88 Other Specified Neurodevelopmental Disorder (99)

F89 Unspecified Neurodevelopmental Disorder (99)

Schizophrenia Spectrum and Other Psychotic Disorders (101)

The following specifiers apply to Schizophrenia Spectrum and Other Psychotic Disorders where indicated:

[a]*Specify* if: The following course specifiers are only to be used after a 1-year duration of the disorder: First episode, currently in acute episode; First episode, currently in partial remission; First episode, currently in full remission; Multiple episodes, currently in acute episode; Multiple episodes, currently in partial remission; Multiple episodes, currently in full remission; Continuous; Unspecified

[b]*Specify* if: With catatonia (use additional code F06.1)

[c]*Specify* current severity of delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, negative symptoms, impaired cognition, depression, and mania symptoms

F21 Schizotypal (Personality) Disorder (104)

F22 Delusional Disorder[a,c] (104)

Specify whether: Erotomanic type, Grandiose type, Jealous type, Persecutory type, Somatic type, Mixed type, Unspecified type

Specify if: With bizarre content

F23 Brief Psychotic Disorder[b,c] (108)

Specify if: With marked stressor(s), Without marked stressor(s), With peripartum onset

F20.81 Schizophreniform Disorder[b,c] (111)

Specify if: With good prognostic features, Without good prognostic features

F20.9 Schizophrenia[a,b,c] (113)

___.__ Schizoaffective Disorder[a,b,c] (121)

Specify whether:

F25.0 Bipolar type

F25.1 Depressive type

___.__ Substance/Medication-Induced Psychotic Disorder[c] (126)

Note: For applicable ICD-10-CM codes, refer to the substance classes under Substance-Related and Addictive Disorders for the specific substance/medication-induced psychotic disorder. See also the criteria set and corresponding recording procedures in the manual for more information.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given.

Specify if: With onset during intoxication, With onset during withdrawal, With onset after medication use

___.__ Psychotic Disorder Due to Another Medical Condition[c] (131)
Specify whether:

F06.2 With delusions

F06.0 With hallucinations

F06.1 Catatonia Associated With Another Mental Disorder (Catatonia Specifier) (135)

F06.1 Catatonic Disorder Due to Another Medical Condition (136)

F06.1 Unspecified Catatonia (137)
Note: Code first **R29.818** other symptoms involving nervous and musculoskeletal systems.

F28 Other Specified Schizophrenia Spectrum and Other Psychotic Disorder (138)

F29 Unspecified Schizophrenia Spectrum and Other Psychotic Disorder (138)

Bipolar and Related Disorders (139)

The following specifiers apply to Bipolar and Related Disorders where indicated:

[a]*Specify:* With anxious distress (*specify* current severity: mild, moderate, moderate-severe, severe); With mixed features; With rapid cycling; With melancholic features; With atypical features; With mood-congruent psychotic features; With mood-incongruent psychotic features; With catatonia (use additional code F06.1); With peripartum onset; With seasonal pattern

[b]*Specify:* With anxious distress (*specify* current severity: mild, moderate, moderate-severe, severe); With mixed features; With rapid cycling; With peripartum onset; With seasonal pattern

___.__ Bipolar I Disorder[a] (139)

____.__ Current or most recent episode manic

F31.11 Mild

F31.12 Moderate

F31.13 Severe

F31.2 With psychotic features

F31.73 In partial remission

F31.74 In full remission

F31.9 Unspecified

F31.0 Current or most recent episode hypomanic

F31.71 In partial remission

F31.72 In full remission

F31.9 Unspecified

___.__ Current or most recent episode depressed

F31.31 Mild

F31.32 Moderate

F31.4 Severe

F31.5 With psychotic features

F31.75 In partial remission

F31.76 In full remission

F31.9 Unspecified

F31.9 Current or most recent episode unspecified

F31.81 Bipolar II Disorder (150)

Specify current or most recent episode: Hypomanic[b], Depressed[a]

Specify course if full criteria for a mood episode are not currently met: In partial remission, In full remission

Specify severity if full criteria for a major depressive episode are currently met: Mild, Moderate, Severe

F34.0 Cyclothymic Disorder (159)

Specify if: With anxious distress (*specify* current severity: mild, moderate, moderate-severe, severe)

___.__ Substance/Medication-Induced Bipolar and Related Disorder (162)

Note: For applicable ICD-10-CM codes, refer to the substance classes under Substance-Related and Addictive Disorders for the specific substance/medication-induced bipolar and related disorder. See also the criteria set and corresponding recording procedures in the manual for more information.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given.

Specify if: With onset during intoxication, With onset during withdrawal, With onset after medication use

___.__ Bipolar and Related Disorder Due to Another Medical Condition (166)

Specify if:

F06.33 With manic features

F06.33 With manic- or hypomanic-like episode

F06.34 With mixed features

F31.89 Other Specified Bipolar and Related Disorder (168)

F31.9 Unspecified Bipolar and Related Disorder (169)

F39 Unspecified Mood Disorder (169)

Depressive Disorders (177)

F34.81 Disruptive Mood Dysregulation Disorder (178)

____.__ Major Depressive Disorder (183)

Specify: With anxious distress (*specify* current severity: mild, moderate, moderate-severe, severe); With mixed features; With melancholic features; With atypical features; With mood-congruent psychotic features; With mood-incongruent psychotic features; With catatonia (use additional code F06.1); With peripartum onset; With seasonal pattern

____.__ Single episode

F32.0 Mild

F32.1 Moderate

F32.2 Severe

F32.3 With psychotic features

F32.4 In partial remission

F32.5 In full remission

F32.9 Unspecified

____.__ Recurrent episode

F33.0 Mild

F33.1 Moderate

F33.2 Severe

F33.3 With psychotic features

F33.41 In partial remission

F33.42 In full remission

F33.9 Unspecified

F34.1 Persistent Depressive Disorder (193)

Specify: With anxious distress (*specify* current severity: mild, moderate, moderate-severe, severe); With atypical features

Specify if: In partial remission, In full remission

Specify if: Early onset, Late onset

Specify if: With pure dysthymic syndrome; With persistent major depressive episode; With intermittent major depressive episodes, with current episode; With intermittent major depressive episodes, without current episode

Specify current severity: Mild, Moderate, Severe

F32.81 Premenstrual Dysphoric Disorder (197)

___.__ Substance/Medication-Induced Depressive Disorder (201)

Note: For applicable ICD-10-CM codes, refer to the substance classes under Substance-Related and Addictive Disorders for the specific substance/medication-induced depressive disorder. See also the criteria set and corresponding recording procedures in the manual for more information.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given.

Specify if: With onset during intoxication, With onset during withdrawal, With onset after medication use

___.__ Depressive Disorder Due to Another Medical Condition (206)

Specify if:

F06.31 With depressive features

F06.32 With major depressive–like episode

F06.34 With mixed features

F32.89 Other Specified Depressive Disorder (209)

F32.A Unspecified Depressive Disorder (210)

F39 Unspecified Mood Disorder (210)

Anxiety Disorders (215)

F93.0 Separation Anxiety Disorder (217)

F94.0 Selective Mutism (222)

____.__ Specific Phobia (224)

Specify if:

F40.218 Animal

F40.228 Natural environment

____.__ Blood-injection-injury

F40.230 Fear of blood

F40.231 Fear of injections and transfusions

F40.232 Fear of other medical care

F40.233 Fear of injury

F40.248 Situational

F40.298 Other

F40.10 Social Anxiety Disorder (229)

Specify if: Performance only

F41.0 Panic Disorder (235)

____.__ Panic Attack Specifier (242)

F40.00 Agoraphobia (246)

F41.1 Generalized Anxiety Disorder (250)

___.__ Substance/Medication-Induced Anxiety Disorder (255)

Note: For applicable ICD-10-CM codes, refer to the substance classes under Substance-Related and Addictive Disorders for the specific substance/medication-induced anxiety disorder. See also the criteria set and corresponding recording procedures in the manual for more information.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given.

Specify if: With onset during intoxication, With onset during withdrawal, With onset after medication use

F06.4 Anxiety Disorder Due to Another Medical Condition (258)

F41.8 Other Specified Anxiety Disorder (261)

F41.9 Unspecified Anxiety Disorder (261)

Obsessive-Compulsive and Related Disorders (263)

The following specifier applies to Obsessive-Compulsive and Related Disorders where indicated:

[a]*Specify* if: With good or fair insight, With poor insight, With absent insight/delusional beliefs

F42.2 Obsessive-Compulsive Disorder[a] (265)

Specify if: Tic-related

F45.22 Body Dysmorphic Disorder[a] (271)

Specify if: With muscle dysmorphia

F42.3 Hoarding Disorder[a] (277)

Specify if: With excessive acquisition

F63.3 Trichotillomania (Hair-Pulling Disorder) (281)

F42.4 Excoriation (Skin-Picking) Disorder (284)

____.___ Substance/Medication-Induced Obsessive-Compulsive and Related Disorder (287)

Note: For applicable ICD-10-CM codes, refer to the substance classes under Substance-Related and Addictive Disorders for the specific substance/medication-induced obsessive-compulsive and related disorder. See also the criteria set and corresponding recording procedures in the manual for more information.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given.

Specify if: With onset during intoxication, With onset during withdrawal, With onset after medication use

F06.8 Obsessive-Compulsive and Related Disorder Due to Another Medical Condition (291)

Specify if: With obsessive-compulsive disorder–like symptoms, With appearance preoccupations, With hoarding symptoms, With hair-pulling symptoms, With skin-picking symptoms

F42.8 Other Specified Obsessive-Compulsive and Related Disorder (293)

F42.9 Unspecified Obsessive-Compulsive and Related Disorder (294)

Trauma- and Stressor-Related Disorders (295)

F94.1 Reactive Attachment Disorder (295)

Specify if: Persistent

Specify current severity: Severe

F94.2 Disinhibited Social Engagement Disorder (298)

Specify if: Persistent

Specify current severity: Severe

F43.10 Posttraumatic Stress Disorder (301)

Specify whether: With dissociative symptoms

Specify if: With delayed expression

____.__ Posttraumatic Stress Disorder in Individuals Older Than 6 Years (301)

____.__ Posttraumatic Stress Disorder in Children 6 Years and Younger (303)

F43.0 Acute Stress Disorder (313)

____.__ Adjustment Disorders (319)

Specify if: Acute, Persistent (chronic)

Specify whether:

F43.21 With depressed mood

F43.22 With anxiety

F43.23 With mixed anxiety and depressed mood

F43.24 With disturbance of conduct

F43.25 With mixed disturbance of emotions and conduct

F43.20 Unspecified

F43.8 Prolonged Grief Disorder (322)

F43.8 Other Specified Trauma- and Stressor-Related Disorder (327)

F43.9 Unspecified Trauma- and Stressor-Related Disorder (328)

Dissociative Disorders (329)

F44.81 Dissociative Identity Disorder (330)

F44.0 Dissociative Amnesia (337)

Specify if:

F44.1 With dissociative fugue

F48.1 Depersonalization/Derealization Disorder (343)

F44.89 Other Specified Dissociative Disorder (347)

F44.9 Unspecified Dissociative Disorder (348)

Somatic Symptom and Related Disorders (349)

F45.1 Somatic Symptom Disorder (351)

Specify if: With predominant pain

Specify if: Persistent

Specify current severity: Mild, Moderate, Severe

F45.21 Illness Anxiety Disorder (357)

Specify whether: Care-seeking type, Care-avoidant type

____.___ Functional Neurological Symptom Disorder (Conversion Disorder) (360)

Specify if: Acute episode, Persistent

Specify if: With psychological stressor (specify stressor), Without psychological stressor

Specify symptom type:

F44.4 With weakness or paralysis

F44.4 With abnormal movement

F44.4 With swallowing symptoms

F44.4 With speech symptom

F44.5 With attacks or seizures

F44.6 With anesthesia or sensory loss

F44.6 With special sensory symptom

F44.7 With mixed symptoms

F54 Psychological Factors Affecting Other Medical Conditions (364)

Specify current severity: Mild, Moderate, Severe, Extreme

____.___ Factitious Disorder (367)

Specify: Single episode, Recurrent episodes

F68.10 Factitious Disorder Imposed on Self

F68.A Factitious Disorder Imposed on Another

F45.8 Other Specified Somatic Symptom and Related Disorder (370)

F45.9 Unspecified Somatic Symptom and Related Disorder (370)

Feeding and Eating Disorders (371)

The following specifiers apply to Feeding and Eating Disorders where indicated:

[a]*Specify* if: In remission

[b]*Specify* if: In partial remission, In full remission

[c]*Specify* current severity: Mild, Moderate, Severe, Extreme

____.___ Pica[a] (371)

F98.3 In children

F50.89 In adults

F98.21 Rumination Disorder[a] (374)

F50.82 Avoidant/Restrictive Food Intake Disorder[a] (376)

____.__ Anorexia Nervosa[b,c] (381)

Specify whether:

F50.01 Restricting type

F50.02 Binge-eating/purging type

F50.2 Bulimia Nervosa[b,c] (387)

F50.81 Binge-Eating Disorder[b,c] (392)

F50.89 Other Specified Feeding or Eating Disorder (396)

F50.9 Unspecified Feeding or Eating Disorder (397)

Elimination Disorders (399)

F98.0 Enuresis (399)

Specify whether: Nocturnal only, Diurnal only, Nocturnal and diurnal

F98.1 Encopresis (402)

Specify whether: With constipation and overflow incontinence, Without constipation and overflow incontinence

____.__ Other Specified Elimination Disorder (405)

N39.498 With urinary symptoms

R15.9 With fecal symptoms

____.__ Unspecified Elimination Disorder (405)

R32 With urinary symptoms

R15.9 With fecal symptoms

Sleep-Wake Disorders (407)

The following specifiers apply to Sleep-Wake Disorders where indicated:

[a]*Specify* if: Episodic, Persistent, Recurrent

[b]*Specify* if: Acute, Subacute, Persistent

[c]*Specify* current severity: Mild, Moderate, Severe

F51.01 Insomnia Disorder[a] (409)

Specify if: With mental disorder, With medical condition, With another sleep disorder

F51.11 Hypersomnolence Disorder[b,c] (417)

Specify if: With mental disorder, With medical condition, With another sleep disorder

____.___ Narcolepsy[c] (422)

Specify whether:

G47.411 Narcolepsy with cataplexy or hypocretin deficiency (type 1)

G47.419 Narcolepsy without cataplexy and either without hypocretin deficiency or hypocretin unmeasured (type 2)

G47.421 Narcolepsy with cataplexy or hypocretin deficiency due to a medical condition

G47.429 Narcolepsy without cataplexy and without hypocretin deficiency due to a medical condition

Breathing-Related Sleep Disorders (429)

G47.33 Obstructive Sleep Apnea Hypopnea[c] (429)

____.___ Central Sleep Apnea (435)

Specify current severity

Specify whether:

G47.31 Idiopathic central sleep apnea

R06.3 Cheyne-Stokes breathing

G47.37 Central sleep apnea comorbid with opioid use

Note: First code opioid use disorder, if present.

____.___ Sleep-Related Hypoventilation (439)

Specify current severity

Specify whether:

G47.34 Idiopathic hypoventilation

G47.35 Congenital central alveolar hypoventilation

G47.36 Comorbid sleep-related hypoventilation

____.___ Circadian Rhythm Sleep-Wake Disorders[a] (443)

Specify whether:

G47.21 Delayed sleep phase type (444)

Specify if: Familial, Overlapping with non-24-hour sleep-wake type

G47.22 Advanced sleep phase type (446)

Specify if: Familial

G47.23 Irregular sleep-wake type (447)

G47.24 Non-24-hour sleep-wake type (448)

G47.26 Shift work type (450)

G47.20 Unspecified type

Parasomnias (451)

___.___ Non–Rapid Eye Movement Sleep Arousal Disorders (452)

Specify whether:

F51.3 Sleepwalking type

Specify if: With sleep-related eating, With sleep-related sexual behavior (sexsomnia)

F51.4 Sleep terror type

F51.5 Nightmare Disorder[b,c] (457)

Specify if: During sleep onset

Specify if: With mental disorder, With medical condition, With another sleep disorder

G47.52 Rapid Eye Movement Sleep Behavior Disorder (461)

G25.81 Restless Legs Syndrome (464)

___.__ Substance/Medication-Induced Sleep Disorder (468)

Note: For applicable ICD-10-CM codes, refer to the substance classes under Substance-Related and Addictive Disorders for the specific substance/medication-induced sleep disorder. See also the criteria set and corresponding recording procedures in the manual for more information.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given.

Specify whether: Insomnia type, Daytime sleepiness type, Parasomnia type, Mixed type

Specify if: With onset during intoxication, With onset during withdrawal, With onset after medication use

G47.09 Other Specified Insomnia Disorder (475)

G47.00 Unspecified Insomnia Disorder (475)

G47.19 Other Specified Hypersomnolence Disorder (475)

G47.10 Unspecified Hypersomnolence Disorder (476)

G47.8 Other Specified Sleep-Wake Disorder (476)

G47.9 Unspecified Sleep-Wake Disorder (476)

Sexual Dysfunctions (477)

The following specifiers apply to Sexual Dysfunctions where indicated:

[a]*Specify* whether: Lifelong, Acquired

[b]*Specify* whether: Generalized, Situational

[c]*Specify* current severity: Mild, Moderate, Severe

F52.32 Delayed Ejaculation[a,b,c] (478)

F52.21 Erectile Disorder[a,b,c] (481)

F52.31 Female Orgasmic Disorder[a,b,c] (485)

Specify if: Never experienced an orgasm under any situation

F52.22 Female Sexual Interest/Arousal Disorder[a,b,c] (489)

F52.6 Genito-Pelvic Pain/Penetration Disorder[a,c] (493)

F52.0 Male Hypoactive Sexual Desire Disorder[a,b,c] (498)

F52.4 Premature (Early) Ejaculation[a,b,c] (501)

___.__

Substance/Medication-Induced Sexual Dysfunction[c] (504)

Note: For applicable ICD-10-CM codes, refer to the substance classes under Substance-Related and Addictive Disorders for the specific substance/medication-induced sexual dysfunction. See also the criteria set and corresponding recording procedures in the manual for more information.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given.

Specify if: With onset during intoxication, With onset during withdrawal, With onset after medication use

F52.8 Other Specified Sexual Dysfunction (509)

F52.9 Unspecified Sexual Dysfunction (509)

Gender Dysphoria (511)

The following specifier and note apply to Gender Dysphoria where indicated:

[a]*Specify* if: With a disorder/difference of sex development

[b]**Note:** Code the disorder/difference of sex development if present, in addition to gender dysphoria.

___.___ Gender Dysphoria (512)

F64.2 Gender Dysphoria in Children[a,b]

F64.0 Gender Dysphoria in Adolescents and Adults[a,b]

Specify if: Posttransition

F64.8 Other Specified Gender Dysphoria (520)

F64.9 Unspecified Gender Dysphoria (520)

Disruptive, Impulse-Control, and Conduct Disorders (521)

F91.3 Oppositional Defiant Disorder (522)

Specify current severity: Mild, Moderate, Severe

Intermittent Explosive Disorder (527)

F63.81

___.___ Conduct Disorder (530)
Specify if: With limited prosocial emotions
Specify current severity: Mild, Moderate, Severe
Specify whether:

F91.1 Childhood-onset type

F91.2 Adolescent-onset type

F91.9 Unspecified onset

F60.2 Antisocial Personality Disorder (537)

F63.1 Pyromania (537)

F63.2 Kleptomania (539)

F91.8 Other Specified Disruptive, Impulse-Control, and Conduct Disorder (541)

F91.9 Unspecified Disruptive, Impulse-Control, and Conduct Disorder (541)

Substance-Related and Addictive Disorders (543)

Substance-Related Disorders (544)

Alcohol-Related Disorders (553)

___.__ Alcohol Use Disorder (553)
Specify if: In a controlled environment
Specify current severity/remission:

F10.10 Mild

F10.11 In early remission

F10.11 In sustained remission

F10.20 Moderate

F10.21 In early remission

F10.21 In sustained remission

F10.20 Severe

F10.21 In early remission

F10.21 In sustained remission

___.__ Alcohol Intoxication (561)

F10.120 With mild use disorder

F10.220 With moderate or severe use disorder

F10.920 Without use disorder

___.__ Alcohol Withdrawal (564)

Without perceptual disturbances

F10.130 With mild use disorder

F10.230 With moderate or severe use disorder

F10.930 Without use disorder

With perceptual disturbances

F10.132 With mild use disorder

F10.232 With moderate or severe use disorder

F10.932 Without use disorder

___.__ Alcohol-Induced Mental Disorders (567)

Note: Disorders are listed in their order of appearance in the manual.

[a]*Specify* With onset during intoxication, With onset during withdrawal

[b]*Specify* if: Acute, Persistent

[c]*Specify* if: Hyperactive, Hypoactive, Mixed level of activity

____.__ Alcohol-Induced Psychotic Disorder[a] (126)

F10.159 With mild use disorder

F10.259 With moderate or severe use disorder

F10.959 Without use disorder

____.__ Alcohol-Induced Bipolar and Related Disorder[a] (162)

F10.14 With mild use disorder

F10.24 With moderate or severe use disorder

F10.94 Without use disorder

___.__	Alcohol-Induced Depressive Disorder[a] (201)
F10.14	With mild use disorder
F10.24	With moderate or severe use disorder
F10.94	Without use disorder
___.__	Alcohol-Induced Anxiety Disorder[a] (255)
F10.180	With mild use disorder
F10.280	With moderate or severe use disorder
F10.980	Without use disorder
___.__	Alcohol-Induced Sleep Disorder[a] (468)
	Specify whether Insomnia type
F10.182	With mild use disorder

F10.282	With moderate or severe use disorder
F10.982	Without use disorder
___.__	Alcohol-Induced Sexual Dysfunction[a] (504)
	Specify if: Mild, Moderate, Severe
F10.181	With mild use disorder
F10.281	With moderate or severe use disorder
F10.981	Without use disorder
___.__	Alcohol Intoxication Delirium[b,c] (672)
F10.121	With mild use disorder
F10.221	With moderate or severe use disorder
F10.921	Without use disorder
___.__	Alcohol Withdrawal Delirium[b,c] (673)
F10.131	With mild use disorder
F10.231	With moderate or severe use disorder

F10.931 Without use disorder

____.__ Alcohol-Induced Major Neurocognitive Disorder (712)

Specify if: Persistent

____.__ Amnestic-confabulatory type

F10.26 With moderate or severe use disorder

F10.96 Without use disorder

____.__ Nonamnestic-confabulatory type

F10.27 With moderate or severe use disorder

F10.97 Without use disorder

____.__ Alcohol-Induced Mild Neurocognitive Disorder (712)

Specify if: Persistent

F10.188 With mild use disorder

F10.288 With moderate or severe use disorder

F10.988 Without use disorder

F10.99 Unspecified Alcohol-Related Disorder (568)

Caffeine-Related Disorders (569)

F15.920 Caffeine Intoxication (569)

F15.93 Caffeine Withdrawal (571)

____.__ Caffeine-Induced Mental Disorders (574)

Note: Disorders are listed in their order of appearance in the manual.

Specify With onset during intoxication, With onset during withdrawal, With onset after medication use. **Note:** When taken over the counter, substances in this class can also induce the relevant substance-induced mental disorder.

F15.980 Caffeine-Induced Anxiety Disorder (255)

F15.982 Caffeine-Induced Sleep Disorder (468)

Specify whether Insomnia type, Daytime sleepiness type, Mixed type

F15.99 Unspecified Caffeine-Related Disorder (574)

Cannabis-Related Disorders (575)

___.__ Cannabis Use Disorder (575)

Specify if: In a controlled environment

Specify current severity/remission:

F12.10 Mild

F12.11 In early remission

F12.11 In sustained remission

F12.20 Moderate

F12.21 In early remission

F12.21 In sustained remission

F12.20 Severe

F12.21 In early remission

F12.21 In sustained remission

___.__ Cannabis Intoxication (582)

Without perceptual disturbances

F12.120 With mild use disorder

F12.220 With moderate or severe use disorder

F12.920 Without use disorder

With perceptual disturbances

F12.122 With mild use disorder

F12.222 With moderate or severe use disorder

F12.922 Without use disorder

___.__ Cannabis Withdrawal (584)

F12.13 With mild use disorder

F12.23 With moderate or severe use disorder

F12.93 Without use disorder

____.___ Cannabis-Induced Mental Disorders (586)

Note: Disorders are listed in their order of appearance in the manual.

[a]*Specify* With onset during intoxication, With onset during withdrawal, With

onset after medication use. **Note:** When prescribed as medication, substances in this class can also induce the relevant substance-induced mental disorder.

[b]*Specify* if: Acute, Persistent

[c]*Specify* if: Hyperactive, Hypoactive, Mixed level of activity

____.__ Cannabis-Induced Psychotic Disorder[a] (126)

F12.159 With mild use disorder

F12.259 With moderate or severe use disorder

F12.959 Without use disorder

____.__ Cannabis-Induced Anxiety Disorder[a] (255)

F12.180 With mild use disorder

F12.280 With moderate or severe use disorder

F12.980 Without use disorder

____.__ Cannabis-Induced Sleep Disorder[a] (468)

Specify whether Insomnia type, Daytime sleepiness type, Mixed type

F12.188 With mild use disorder

F12.288 With moderate or severe use disorder

F12.988 Without use disorder

____.__ Cannabis Intoxication Delirium[b,c] (672)

F12.121 With mild use disorder

F12.221 With moderate or severe use disorder

F12.921 Without use disorder

F12.921 Pharmaceutical Cannabis Receptor Agonist–Induced Delirium[b,c] (674)

Note: When pharmaceutical cannabis receptor agonist medication taken as prescribed. The designation “taken as prescribed” is used to differentiate

medication-induced delirium from substance intoxication delirium.

F12.99 Unspecified Cannabis-Related Disorder (586)

Hallucinogen-Related Disorders (587)

___.__ Phencyclidine Use Disorder (587)
Specify if: In a controlled environment
Specify current severity/remission:

F16.10 Mild

F16.11 In early remission

F16.11 In sustained remission

F16.20 Moderate

F16.21 In early remission

F16.21 In sustained remission

F16.20 Severe

F16.21 In early remission

F16.21 In sustained remission

___.__ Other Hallucinogen Use Disorder (590)
Specify the particular hallucinogen
Specify if: In a controlled environment
Specify current severity/remission:

F16.10 Mild

F16.11 In early remission

F16.11 In sustained remission

F16.20 Moderate

F16.21 In early remission

F16.21 In sustained remission

F16.20 Severe

F16.21 In early remission

F16.21

In sustained remission

___.__ Phencyclidine Intoxication (594)

F16.120 With mild use disorder

F16.220 With moderate or severe use disorder

F16.920 Without use disorder

___.__ Other Hallucinogen Intoxication (596)

F16.120 With mild use disorder

F16.220 With moderate or severe use disorder

F16.920 Without use disorder

F16.983 Hallucinogen Persisting Perception Disorder (598)

___.__ Phencyclidine-Induced Mental Disorders (600)

Note: Disorders are listed in their order of appearance in the manual.

[a]*Specify* With onset during intoxication, With onset after medication use. **Note:** When prescribed as medication, substances in this class can also induce the relevant substance-induced mental disorder.

____.__ Phencyclidine-Induced Psychotic Disorder[a] (126)

F16.159 With mild use disorder

F16.259 With moderate or severe use disorder

F16.959 Without use disorder

____.__ Phencyclidine-Induced Bipolar and Related Disorder[a] (162)

F16.14 With mild use disorder

F16.24 With moderate or severe use disorder

F16.94 Without use disorder

____.__ Phencyclidine-Induced Depressive Disorder[a] (201)

F16.14 With mild use disorder

F16.24 With moderate or severe use disorder

F16.94 Without use disorder

Phencyclidine-Induced Anxiety Disorder[a] (255)

____.__ Phencyclidine-Induced Anxiety Disorder[a] (255)

With mild use disorder

F16.180

F16.280 With moderate or severe use disorder

F16.980 Without use disorder

____.__ Phencyclidine Intoxication Delirium (672)

Specify if: Acute, Persistent

Specify if: Hyperactive, Hypoactive, Mixed level of activity

F16.121 With mild use disorder

F16.221 With moderate or severe use disorder

F16.921 Without use disorder

____.__ Hallucinogen-Induced Mental Disorders (600)

Note: Disorders are listed in their order of appearance in the manual.

[a]*Specify* With onset during intoxication, With onset after medication use. **Note:** When prescribed as medication, substances in this class can also induce the relevant substance-induced mental disorder.

[b]*Specify* if: Acute, Persistent

[c]*Specify* if: Hyperactive, Hypoactive, Mixed level of activity

____.__ Other Hallucinogen–Induced Psychotic Disorder[a] (126)

F16.159 With mild use disorder

F16.259 With moderate or severe use disorder

F16.959 Without use disorder

____.__ Other Hallucinogen–Induced Bipolar and Related Disorder[a] (162)

F16.14 With mild use disorder

F16.24 With moderate or severe use disorder

F16.94 Without use disorder

____.__ Other Hallucinogen–Induced Depressive Disorder[a] (201)

F16.14 With mild use disorder

F16.24

With moderate or severe use disorder

F16.94 Without use disorder

___.__ Other Hallucinogen-Induced Anxiety Disorder[a] (255)

F16.180 With mild use disorder

F16.280 With moderate or severe use disorder

F16.980 Without use disorder

___.__ Other Hallucinogen Intoxication Delirium[b,c] (672)

F16.121 With mild use disorder

F16.221 With moderate or severe use disorder

F16.921 Without use disorder

F16.921 Ketamine or Other Hallucinogen–Induced Delirium[b,c] (674)

Note: When ketamine or other hallucinogen medication taken as prescribed. The designation "taken as prescribed" is used to differentiate medication-induced delirium from substance intoxication delirium.

F16.99 Unspecified Phencyclidine-Related Disorder (600)

F16.99 Unspecified Hallucinogen-Related Disorder (601)

Inhalant-Related Disorders (601)

___.__ Inhalant Use Disorder (601)
Specify the particular inhalant
Specify if: In a controlled environment
Specify current severity/remission:

F18.10 Mild

F18.11 In early remission

F18.11 In sustained remission

F18.20 Moderate

F18.21 In early remission

F18.21 In sustained remission

F18.20 Severe

F18.21 In early remission

F18.21 In sustained remission

___.__ Inhalant Intoxication (605)

F18.120 With mild use disorder

F18.220 With moderate or severe use disorder

F18.920 Without use disorder

____.__ Inhalant-Induced Mental Disorders (607)

Note: Disorders are listed in their order of appearance in the manual.

[a]*Specify* With onset during intoxication

____.__ Inhalant-Induced Psychotic Disorder[a] (126)

F18.159 With mild use disorder

F18.259 With moderate or severe use disorder

F18.959 Without use disorder

____.__ Inhalant-Induced Depressive Disorder[a] (201)

F18.14 With mild use disorder

F18.24 With moderate or severe use disorder

F18.94 Without use disorder

____.__ Inhalant-Induced Anxiety Disorder[a] (255)

F18.180 With mild use disorder

F18.280 With moderate or severe use disorder

F18.980 Without use disorder

____.__ Inhalant Intoxication Delirium (672)

Specify if: Acute, Persistent

Specify if: Hyperactive, Hypoactive, Mixed level of activity

F18.121 With mild use disorder

F18.221 With moderate or severe use disorder

F18.921 Without use disorder

____.__ Inhalant-Induced Major Neurocognitive Disorder (712)

Specify if: Persistent

F18.17 With mild use disorder

F18.27 With moderate or severe use disorder

F18.97 Without use disorder

____.__ Inhalant-Induced Mild Neurocognitive Disorder (712)

Specify if: Persistent

F18.188 With mild use disorder

F18.288 With moderate or severe use disorder

F18.988 Without use disorder

F18.99 Unspecified Inhalant-Related Disorder (608)

Opioid-Related Disorders (608)

___.__ Opioid Use Disorder (608)

Specify if: On maintenance therapy, In a controlled environment

Specify current severity/remission:

F11.10 Mild

F11.11 In early remission

F11.11 In sustained remission

F11.20 Moderate

F11.21 In early remission

F11.21 In sustained remission

F11.20 Severe

F11.21 In early remission

F11.21 In sustained remission

___.__ Opioid Intoxication (615)

Without perceptual disturbances

F11.120 With mild use disorder

F11.220 With moderate or severe use disorder

F11.920 Without use disorder

With perceptual disturbances

F11.122 With mild use disorder

F11.222 With moderate or severe use disorder

F11.922 Without use disorder

___.__ Opioid Withdrawal (617)

F11.13 With mild use disorder

F11.23 With moderate or severe use disorder

F11.93 Without use disorder

____.__ Opioid-Induced Mental Disorders (619)

Note: Disorders are listed in their order of appearance in the manual.

[a]*Specify* With onset during intoxication, With onset during withdrawal, With onset after medication use. **Note:** When prescribed as medication, substances in this class can also induce the relevant substance-induced mental disorder.

[b]*Specify* if: Acute, Persistent

[c]*Specify* if: Hyperactive, Hypoactive, Mixed level of activity

____.__ Opioid-Induced Depressive Disorder[a] (201)

F11.14 With mild use disorder

F11.24 With moderate or severe use disorder

F11.94 Without use disorder

____.__ Opioid-Induced Anxiety Disorder[a] (255)

F11.180

	With mild use disorder
F11.280	With moderate or severe use disorder
F11.980	Without use disorder
___.__	Opioid-Induced Sleep Disorder[a] (468)
	Specify whether Insomnia type, Daytime sleepiness type, Mixed type
F11.182	With mild use disorder
F11.282	With moderate or severe use disorder
F11.982	Without use disorder
___.__	Opioid-Induced Sexual Dysfunction[a] (504)
	Specify if: Mild, Moderate, Severe
F11.181	With mild use disorder
F11.281	With moderate or severe use disorder
F11.981	Without use disorder
___.__	Opioid Intoxication Delirium[b,c] (672)
F11.121	With mild use disorder
F11.221	With moderate or severe use disorder
F11.921	Without use disorder
___.__	Opioid Withdrawal Delirium[b,c] (673)
F11.188	With mild use disorder
F11.288	With moderate or severe use disorder
F11.988	Without use disorder
___.__	Opioid-Induced Delirium[b,c] (674)
	Note: The designation “taken as prescribed” is used to differentiate medication-induced delirium from substance intoxication delirium and substance withdrawal delirium.
F11.921	When opioid medication taken as prescribed (674)

F11.988 During withdrawal from opioid medication taken as prescribed (674)

F11.99 Unspecified Opioid-Related Disorder (619)

Sedative-, Hypnotic-, or Anxiolytic-Related Disorders (620)

___.__ Sedative, Hypnotic, or Anxiolytic Use Disorder (620)
Specify if: In a controlled environment
Specify current severity/remission:

F13.10 Mild

F13.11 In early remission

F13.11 In sustained remission

F13.20 Moderate

F13.21 In early remission

F13.21 In sustained remission

F13.20 Severe

F13.21 In early remission

F13.21 In sustained remission

___.__ Sedative, Hypnotic, or Anxiolytic Intoxication (626)

F13.120 With mild use disorder

F13.220 With moderate or severe use disorder

F13.920 Without use disorder

___.__ Sedative, Hypnotic, or Anxiolytic Withdrawal (628)

Without perceptual disturbances

F13.130 With mild use disorder

F13.230 With moderate or severe use disorder

F13.930 Without use disorder

With perceptual disturbances

F13.132 With mild use disorder

F13.232 With moderate or severe use disorder

F13.932 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Mental Disorders (631)

Note: Disorders are listed in their order of appearance in the manual.

[a]*Specify* With onset during intoxication, With onset during withdrawal, With onset after medication use. **Note:** When prescribed as medication, substances in this class can also induce the relevant substance-induced mental disorder.

[b]*Specify* if: Acute, Persistent

[c]*Specify* if: Hyperactive, Hypoactive, Mixed level of activity

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Psychotic Disorder[a] (126)

F13.159 With mild use disorder

F13.259 With moderate or severe use disorder

F13.959 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Bipolar and Related Disorder[a] (162)

F13.14 With mild use disorder

F13.24 With moderate or severe use disorder

F13.94 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Depressive Disorder[a] (201)

F13.14 With mild use disorder

F13.24 With moderate or severe use disorder

F13.94 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Anxiety Disorder[a] (255)

F13.180 With mild use disorder

F13.280 With moderate or severe use disorder

F13.980 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Sleep Disorder[a] (468)

Specify whether Insomnia type, Daytime sleepiness type, Parasomnia type, Mixed type

F13.182 With mild use disorder

F13.282 With moderate or severe use disorder

F13.982 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Sexual Dysfunction[a] (504)

Specify if: Mild, Moderate, Severe

F13.181 With mild use disorder

F13.281 With moderate or severe use disorder

F13.981 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic Intoxication Delirium[b,c] (672)

F13.121 With mild use disorder

F13.221 With moderate or severe use disorder

F13.921 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic Withdrawal Delirium[b,c] (673)

F13.131 With mild use disorder

F13.231 With moderate or severe use disorder

F13.931 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Delirium[b,c] (674)

Note: The designation “taken as prescribed” is used to differentiate medication-induced delirium from substance intoxication delirium and substance

withdrawal delirium.

F13.921 When sedative, hypnotic, or anxiolytic medication taken as prescribed (674)

F13.931 During withdrawal from sedative, hypnotic, or anxiolytic medication taken as prescribed (674)

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Major Neurocognitive Disorder (712)

Specify if: Persistent

F13.27 With moderate or severe use disorder

F13.97 Without use disorder

____.__ Sedative-, Hypnotic-, or Anxiolytic-Induced Mild Neurocognitive Disorder (712)

Specify if: Persistent

F13.188 With mild use disorder

F13.288 With moderate or severe use disorder

F13.988 Without use disorder

F13.99 Unspecified Sedative-, Hypnotic-, or Anxiolytic-Related Disorder (632)

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Stimulant-Related Disorders (632)

____.__ Stimulant Use Disorder (632)

Specify if: In a controlled environment

Specify current severity/remission:

____.__ Mild

F15.10 Amphetamine-type substance

F14.10 Cocaine

F15.10 Other or unspecified stimulant

____.__ Mild, In early remission

F15.11 Amphetamine-type substance

F14.11 Cocaine

F15.11 Other or unspecified stimulant

___.__ Mild, In sustained remission

F15.11 Amphetamine-type substance

F14.11 Cocaine

F15.11 Other or unspecified stimulant

___.__ Moderate

F15.20 Amphetamine-type substance

F14.20 Cocaine

F15.20 Other or unspecified stimulant

___.__ Moderate, In early remission

F15.21 Amphetamine-type substance

F14.21 Cocaine

F15.21 Other or unspecified stimulant

___.__ Moderate, In sustained remission

F15.21 Amphetamine-type substance

F14.21 Cocaine

F15.21 Other or unspecified stimulant

___.__ Severe

F15.20 Amphetamine-type substance

F14.20 Cocaine

F15.20 Other or unspecified stimulant

___.__ Severe, In early remission

F15.21 Amphetamine-type substance

F14.21 Cocaine

F15.21 Other or unspecified stimulant

___.__

Severe. In sustained remission

F15.21 Amphetamine-type substance

F14.21 Cocaine

F15.21 Other or unspecified stimulant

____.__ Stimulant Intoxication (640)

Specify the particular intoxicant

Without perceptual disturbances

li

____.__ Amphetamine-type substance or other stimulant intoxication

F15.120 With mild use disorder

F15.220 With moderate or severe use disorder

F15.920 Without use disorder

____.__ Cocaine intoxication

F14.120 With mild use disorder

F14.220 With moderate or severe use disorder

F14.920 Without use disorder

With perceptual disturbances

____.__ Amphetamine-type substance or other stimulant intoxication

F15.122 With mild use disorder

F15.222 With moderate or severe use disorder

F15.922 Without use disorder

____.__ Cocaine intoxication

F14.122 With mild use disorder

F14.222 With moderate or severe use disorder

F14.922 Without use disorder

Stimulant Withdrawal (643)

____.__

Specify the particular substance that causes the withdrawal syndrome

____.__ Amphetamine-type substance or other stimulant withdrawal

F15.13 With mild use disorder

F15.23 With moderate or severe use disorder

F15.93 Without use disorder

____.__ Cocaine withdrawal

F14.13 With mild use disorder

F14.23 With moderate or severe use disorder

F14.93 Without use disorder

____.__ Stimulant-Induced Mental Disorders (644)

Note: Disorders are listed in their order of appearance in the manual.

[a]*Specify* With onset during intoxication, With onset during withdrawal, With onset after medication use. **Note:** When prescribed as medication, amphetamine-type substances and other stimulants can also induce the relevant substance-induced mental disorder.

[b]*Specify* if: Acute, Persistent

[c]*Specify* if: Hyperactive, Hypoactive, Mixed level of activity

____.__ Amphetamine-Type Substance (or Other Stimulant)–Induced Psychotic Disorder[a] (126)

F15.159 With mild use disorder

F15.259 With moderate or severe use disorder

F15.959 Without use disorder

____.__ Cocaine-Induced Psychotic Disorder[a] (126)

F14.159 With mild use disorder

F14.259 With moderate or severe use disorder

F14.959 Without use disorder

Amphetamine-Type Substance (or Other Stimulant)–

____.__	Induced Bipolar and Related Disorder[a] (162)
F15.14	With mild use disorder
F15.24	With moderate or severe use disorder
F15.94	Without use disorder
____.__	Cocaine-Induced Bipolar and Related Disorder[a] (162)
F14.14	With mild use disorder
F14.24	With moderate or severe use disorder
F14.94	Without use disorder
____.__	Amphetamine-Type Substance (or Other Stimulant)–Induced Depressive Disorder[a] (201)
F15.14	With mild use disorder
F15.24	With moderate or severe use disorder
F15.94	Without use disorder
____.__	Cocaine-Induced Depressive Disorder[a] (201)
F14.14	With mild use disorder
F14.24	With moderate or severe use disorder
F14.94	Without use disorder
____.__	Amphetamine-Type Substance (or Other Stimulant)–Induced Anxiety Disorder[a] (255)
F15.180	With mild use disorder
F15.280	With moderate or severe use disorder
F15.980	Without use disorder
____.__	Cocaine-Induced Anxiety Disorder[a] (255)
F14.180	With mild use disorder
F14.280	With moderate or severe use disorder
F14.980	Without use disorder
____.__	Amphetamine-Type Substance (or Other Stimulant)–

Induced Obsessive-Compulsive and Related Disorder[a] (287)

F15.188 With mild use disorder

F15.288 With moderate or severe use disorder

F15.988 Without use disorder

___.__ Cocaine-Induced Obsessive-Compulsive and Related Disorder[a] (287)

F14.188 With mild use disorder

F14.288 With moderate or severe use disorder

F14.988 Without use disorder

___.__ Amphetamine-Type Substance (or Other Stimulant)–Induced Sleep Disorder[a] (468)

Specify whether Insomnia type, Daytime sleepiness type, Mixed type

F15.182 With mild use disorder

F15.282 With moderate or severe use disorder

F15.982 Without use disorder

___.__ Cocaine-Induced Sleep Disorder[a] (468)

Specify whether Insomnia type, Daytime sleepiness type, Mixed type

F14.182 With mild use disorder

F14.282 With moderate or severe use disorder

F14.982 Without use disorder

___.__ Amphetamine-Type Substance (or Other Stimulant)–Induced Sexual Dysfunction[a] (504)

Specify if: Mild, Moderate, Severe

F15.181 With mild use disorder

With moderate or severe use disorder

F15.281

F15.981 Without use disorder

____.__ Cocaine-Induced Sexual Dysfunction[a] (504)

Specify if: Mild, Moderate, Severe

F14.181 With mild use disorder

F14.281 With moderate or severe use disorder

F14.981 Without use disorder

____.__ Amphetamine-Type Substance (or Other Stimulant) Intoxication Delirium[b,c] (672)

F15.121 With mild use disorder

F15.221 With moderate or severe use disorder

F15.921 Without use disorder

____.__ Cocaine Intoxication Delirium[b,c] (672)

F14.121 With mild use disorder

F14.221 With moderate or severe use disorder

F14.921 Without use disorder

F15.921 Amphetamine-Type (or Other Stimulant) Medication–Induced Delirium[b,c] (674)

Note: When amphetamine-type or other stimulant medication taken as prescribed. The designation "taken as prescribed" is used to differentiate medication-induced delirium from substance intoxication delirium.

____.__ Amphetamine-Type Substance (or Other Stimulant)–Induced Mild Neurocognitive Disorder (712)

Specify if: Persistent

F15.188 With mild use disorder

F15.288 With moderate or severe use disorder

F15.988

F15.988 Without use disorder

____.__ Cocaine-Induced Mild Neurocognitive Disorder (712)

Specify if: Persistent

F14.188 With mild use disorder

F14.288 With moderate or severe use disorder

F14.988 Without use disorder

____.__ Unspecified Stimulant-Related Disorder (644)

F15.99 Amphetamine-type substance or other stimulant

F14.99 Cocaine

Tobacco-Related Disorders (645)

___.__ Tobacco Use Disorder (645)

Specify if: On maintenance therapy, In a controlled environment

Specify current severity/remission:

Z72.0 Mild

F17.200 Moderate

F17.201 In early remission

F17.201 In sustained remission

F17.200 Severe

F17.201 In early remission

F17.201 In sustained remission

F17.203 Tobacco Withdrawal (649)

Note: The ICD-10-CM code indicates the comorbid presence of a moderate or severe tobacco use disorder, which must be present in order to apply the code for tobacco withdrawal.

____.__ Tobacco-Induced Mental Disorders (651)

F17.208 Tobacco-Induced Sleep Disorder, With moderate or severe use disorder (468)

Specify whether Insomnia type, Daytime sleepiness type, Mixed type

Mixed type

Specify With onset during withdrawal, With onset after medication use

F17.209 Unspecified Tobacco-Related Disorder (651)

Other (or Unknown) Substance–Related Disorders (652)

___.__ Other (or Unknown) Substance Use Disorder (652)
Specify if: In a controlled environment
Specify current severity/remission:

F19.10 Mild

F19.11 In early remission

F19.11 In sustained remission

F19.20 Moderate

F19.21 In early remission

F19.21 In sustained remission

F19.20 Severe

F19.21 In early remission

F19.21 In sustained remission

___.__ Other (or Unknown) Substance Intoxication (656)

Without perceptual disturbances

F19.120 With mild use disorder

F19.220 With moderate or severe use disorder

F19.920 Without use disorder

With perceptual disturbances

F19.122 With mild use disorder

F19.222 With moderate or severe use disorder

F19.922 Without use disorder

____.___ Other (or Unknown) Substance Withdrawal (658)

Without perceptual disturbances

F19.130 With mild use disorder

F19.230 With moderate or severe use disorder

F19.930 Without use disorder

With perceptual disturbances

F19.132 With mild use disorder

F19.232 With moderate or severe use disorder

F19.932 Without use disorder

____.__ Other (or Unknown) Substance–Induced Mental Disorders (660)

Note: Disorders are listed in their order of appearance in the manual.

[a]*Specify* With onset during intoxication, With onset during withdrawal, With onset after medication use. **Note:** When prescribed as medication or taken over the counter, substances in this class can also induce the relevant substance-induced mental disorder.

[b]*Specify* if: Acute, Persistent

[c]*Specify* if: Hyperactive, Hypoactive, Mixed level of activity

____.__ Other (or Unknown) Substance–Induced Psychotic Disorder[a] (126)

F19.159 With mild use disorder

F19.259 With moderate or severe use disorder

F19.959 Without use disorder

____.__ Other (or Unknown) Substance–Induced Bipolar and Related Disorder[a] (162)

F19.14 With mild use disorder

F19.24 With moderate or severe use disorder

F19.94 Without use disorder

____.__ Other (or Unknown) Substance–Induced Depressive Disorder[a] (201)

F19.14 With mild use disorder

F19.24

With moderate or severe use disorder

F19.94 Without use disorder

___.__ Other (or Unknown) Substance–Induced Anxiety Disorder[a] (255)

F19.180 With mild use disorder

F19.280 With moderate or severe use disorder

F19.980 Without use disorder

___.__ Other (or Unknown) Substance–Induced Obsessive-Compulsive and Related Disorder[a] (287)

F19.188 With mild use disorder

F19.288 With moderate or severe use disorder

F19.988 Without use disorder

___.__ Other (or Unknown) Substance–Induced Sleep Disorder[a] (468)

Specify whether Insomnia type, Daytime sleepiness type, Parasomnia type, Mixed type

F19.182 With mild use disorder

F19.282 With moderate or severe use disorder

F19.982 Without use disorder

___.__ Other (or Unknown) Substance–Induced Sexual Dysfunction[a] (504)

Specify if: Mild, Moderate, Severe

F19.181 With mild use disorder

F19.281 With moderate or severe use disorder

F19.981 Without use disorder

___.__ Other (or Unknown) Substance Intoxication Delirium[b,c] (672)

F19.121 With mild use disorder

F19.221 With moderate or severe use disorder

F19.921 Without use disorder

____.__ Other (or Unknown) Substance Withdrawal Delirium[b,c] (673)

F19.131 With mild use disorder

F19.231 With moderate or severe use disorder

F19.931 Without use disorder

____.__ Other (or Unknown) Medication–Induced Delirium[b,c] (674)

Note: The designation "taken as prescribed" is used to differentiate medication-induced delirium from substance intoxication delirium and substance withdrawal delirium.

F19.921 When other (or unknown) medication taken as prescribed (674)

F19.931 During withdrawal from other (or unknown) medication taken as prescribed (674)

____.__ Other (or Unknown) Substance–Induced Major Neurocognitive Disorder (712)

Specify if: Persistent

F19.17 With mild use disorder

F19.27 With moderate or severe use disorder

F19.97 Without use disorder

____.__ Other (or Unknown) Substance–Induced Mild Neurocognitive Disorder (712)

Specify if: Persistent

F19.188 With mild use disorder

F19.288 With moderate or severe use disorder

F19.988

F19.__ Without use disorder

F19.99 Unspecified Other (or Unknown) Substance–Related Disorder (660)

Non-Substance-Related Disorders (661)

F63.0 Gambling Disorder (661)

Specify if: Episodic, Persistent

Specify if: In early remission, In sustained remission

Specify current severity: Mild, Moderate, Severe

Neurocognitive Disorders (667)

___.__ Delirium (672)

Specify if: Acute, Persistent

Specify if: Hyperactive, Hypoactive, Mixed level of activity

[a]**Note:** For applicable ICD-10-CM codes, refer to the substance classes under Substance-Related and Addictive Disorders for the specific substance/medication-induced delirium. See also the criteria set and corresponding recording procedures in the manual for more information.

Specify whether:

____.__ Substance intoxication delirium[a]

____.__ Substance withdrawal delirium[a]

____.__ Medication-induced delirium[a]

F05 Delirium due to another medical condition

F05 Delirium due to multiple etiologies

R41.0 Other Specified Delirium (678)

R41.0 Unspecified Delirium (678)

Major and Mild Neurocognitive Disorders (679)

Specify whether due to *[any of the following medical etiologies]*: Alzheimer's disease, Frontotemporal degeneration, Lewy body disease, Vascular disease, Traumatic brain injury, Substance/medication use, HIV infection, Prion disease, Parkinson's disease, Huntington's disease, Another medical condition, Multiple etiologies, Unspecified etiology

[a]*Specify* current severity: Mild, Moderate, Severe. *This specifier applies only to major neurocognitive disorders (including probable and possible).*

[b]*Specify:* Without behavioral disturbance, With behavioral disturbance. *For all mild neurocognitive disorders, substance/medication-induced major neurocognitive disorder, and unspecified neurocognitive disorder, behavioral disturbance cannot be coded but should still be recorded.*

Note: As indicated for each subtype, an additional medical code is needed for most major neurocognitive disorders, including those due to probable and possible medical etiologies. The medical etiology should be coded first, before the code for the major neurocognitive disorder. An additional medical code should *not* be used for any mild neurocognitive disorder and is not used for major or mild vascular neurocognitive disorder, substance/medication-induced major or mild neurocognitive disorder, and unspecified neurocognitive disorder.

Coding note: *For major and mild neurocognitive disorders:* Use additional code(s) to indicate clinically significant psychiatric symptoms due to the same medical condition causing the major NCD (e.g., F06.2 psychotic disorder due to Alzheimer's disease with delusions; F06.32 depressive disorder due to Parkinson's disease, with major depressive-like episode.) **Note:** The additional codes for mental disorders due to another medical condition are included with disorders with which they share phenomenology (e.g., for depressive disorders due to another medical condition, see "Depressive Disorders").

Major or Mild Neurocognitive Disorder Due to Alzheimer's Disease (690)

____.___ Major Neurocognitive Disorder Due to Probable Alzheimer's Disease[a]
Note: Code first **G30.9** Alzheimer's disease.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

____.___ Major Neurocognitive Disorder Due to Possible Alzheimer's Disease[a]
Note: Code first **G30.9** Alzheimer's disease.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder Due to Alzheimer's Disease[b]

Major or Mild Frontotemporal Neurocognitive Disorder (695)

____.___ Major Neurocognitive Disorder Due to Probable Frontotemporal Degeneration[a]
Note: Code first **G31.09** frontotemporal degeneration.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

Major Neurocognitive Disorder Due to Possible Frontotemporal Degeneration[a]

~~Major Neurocognitive Disorder Due to Possible Frontotemporal Degeneration~~

____.___

Note: Code first **G31.09** frontotemporal degeneration.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder Due to Frontotemporal Degeneration[b]

Major or Mild Neurocognitive Disorder With Lewy Bodies (699)

____.___ Major Neurocognitive Disorder With Probable Lewy Bodies[a]

Note: Code first **G31.83** Lewy body disease.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

____.___ Major Neurocognitive Disorder With Possible Lewy Bodies[a]

Note: Code first **G31.83** Lewy body disease.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder With Lewy Bodies[b]

Major or Mild Vascular Neurocognitive Disorder (702)

____.___ Major Neurocognitive Disorder Probably Due to Vascular Disease[a]

Note: No additional medical code for vascular disease.

F01.51 With behavioral disturbance

F01.50 Without behavioral disturbance

____.___ Major Neurocognitive Disorder Possibly Due to Vascular Disease[a]

Note: No additional medical code for vascular disease.

F01.51 With behavioral disturbance

F01.50 Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder Due to Vascular Disease[b]

Major or Mild Neurocognitive Disorder Due to Traumatic Brain Injury (706)

____.___ Major Neurocognitive Disorder Due to Traumatic Brain Injury[a]

Note: For ICD-10-CM, code first **S06.2X9S** diffuse traumatic brain injury with loss of consciousness of unspecified duration, sequela.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder Due to Traumatic Brain Injury[b]

Substance/Medication-Induced Major or Mild Neurocognitive Disorder (712)

Note: No additional medical code. For applicable ICD-10-CM codes, refer to the substance classes under Substance-Related and Addictive Disorders for the specific substance/medication-induced major or mild neurocognitive disorder. See also the criteria set and corresponding recording procedures in the manual for more information.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given.

Specify if: Persistent

____.___ Substance/Medication-Induced Major Neurocognitive Disorder[a,b]

Note: If a substance use disorder is present, record mild substance use disorder (ICD-10-CM code not available if mild substance use disorder does not cause a major neurocognitive disorder) or moderate or severe substance use disorder; if no substance use disorder is present, record only [*specific substance*]-induced major neurocognitive disorder.

____.____ Substance/Medication-Induced Mild Neurocognitive Disorder[b]

Note: If a substance use disorder is present, record mild substance use disorder or moderate or severe substance use disorder; if no substance use disorder is present, record only [*specific substance*]-induced mild neurocognitive disorder.

Major or Mild Neurocognitive Disorder Due to HIV Infection (717)

____.___ Major Neurocognitive Disorder Due to HIV Infection[a]

Note: Code first **B20** HIV infection.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder Due to HIV Infection[b]

Major or Mild Neurocognitive Disorder Due to Prion Disease (721)

____.___ Major Neurocognitive Disorder Due to Prion Disease[a]

Note: Code first **A81.9** prion disease.

F02.81 With behavioral disturbance

F02.80

Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder Due to Prion Disease[b]

Major or Mild Neurocognitive Disorder Due to Parkinson's Disease (723)

____.__ Major Neurocognitive Disorder Probably Due to Parkinson's Disease[a]
Note: Code first **G20** Parkinson's disease.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

____.__ Major Neurocognitive Disorder Possibly Due to Parkinson's Disease[a]
Note: Code first **G20** Parkinson's disease.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder Due to Parkinson's Disease[b]

Major or Mild Neurocognitive Disorder Due to Huntington's Disease (726)

____.__ Major Neurocognitive Disorder Due to Huntington's Disease[a]
Note: Code first **G10** Huntington's disease.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder Due to Huntington's Disease[b]

Major or Mild Neurocognitive Disorder Due to Another Medical Condition (729)

____.__ Major Neurocognitive Disorder Due to Another Medical Condition[a]
Note: Code first the other medical condition.

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

G31.84 Mild Neurocognitive Disorder Due to Another Medical Condition[b]

Major or Mild Neurocognitive Disorder Due to Multiple Etiologies

(731)

____.__ Major Neurocognitive Disorder Due to Multiple Etiologies[a]

Note: Code first all the etiological medical conditions (with the exception of vascular disease, which is not coded).

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

Note: If vascular disease is among the multiple etiological medical conditions, code either **F01.51** for major vascular neurocognitive disorder, with behavioral disturbance, or **F01.50** for major vascular neurocognitive disorder, without behavioral disturbance.

G31.84 Mild Neurocognitive Disorder Due to Multiple Etiologies[b]

Unspecified Neurocognitive Disorder[b] (732)

Note: No additional medical code.

R41.9 Unspecified Neurocognitive Disorder[b]

Personality Disorders (733)

Cluster A Personality Disorders

F60.0 Paranoid Personality Disorder (737)

F60.1 Schizoid Personality Disorder (741)

F21 Schizotypal Personality Disorder (744)

Cluster B Personality Disorders

F60.2 Antisocial Personality Disorder (748)

F60.3 Borderline Personality Disorder (752)

F60.4 Histrionic Personality Disorder (757)

F60.81 Narcissistic Personality Disorder (760)

Cluster C Personality Disorders

F60.6 Avoidant Personality Disorder (764)

F60.7 Dependent Personality Disorder (768)

F60.5 Obsessive-Compulsive Personality Disorder (771)

Other Personality Disorders

F07.0 Personality Change Due to Another Medical Condition (775)

Specify whether: Labile type, Disinhibited type, Aggressive type, Apathetic type, Paranoid type, Other type, Combined type, Unspecified type

F60.89 Other Specified Personality Disorder (778)

F60.9 Unspecified Personality Disorder (778)

Paraphilic Disorders (779)

The following specifier applies to Paraphilic Disorders where indicated:

[a]*Specify* if: In a controlled environment, In full remission

F65.3 Voyeuristic Disorder[a] (780)

F65.2 Exhibitionistic Disorder[a] (783)

Specify whether: Sexually aroused by exposing genitals to prepubertal children, Sexually aroused by exposing genitals to physically mature individuals, Sexually aroused by exposing genitals to prepubertal children and to physically mature individuals

F65.81 Frotteuristic Disorder[a] (785)

F65.51 Sexual Masochism Disorder[a] (788)

Specify if: With asphyxiophilia

F65.52 Sexual Sadism Disorder[a] (790)

F65.4 Pedophilic Disorder (792)

Specify whether: Exclusive type, Nonexclusive type

Specify if: Sexually attracted to males, Sexually attracted to females, Sexually attracted to both

Specify if: Limited to incest

F65.0 Fetishistic Disorder[a] (796)

Specify: Body part(s), Nonliving object(s), Other

F65.1 Transvestic Disorder[a] (798)

Specify if: With fetishism, With autogynephilia

F65.89 Other Specified Paraphilic Disorder (801)

F65.9 Unspecified Paraphilic Disorder (801)

Other Mental Disorders and Additional Codes (803)

F06.8 Other Specified Mental Disorder Due to Another Medical Condition (803)

F09 Unspecified Mental Disorder Due to Another Medical Condition (804)

F99 Other Specified Mental Disorder (804)

F99 Unspecified Mental Disorder (805)

Z03.89 No Diagnosis or Condition (805)

Medication-Induced Movement Disorders and Other Adverse Effects of Medication (807)

____.__ Medication-Induced Parkinsonism (807)

G21.11 Antipsychotic Medication– and Other Dopamine Receptor Blocking Agent–Induced Parkinsonism (807)

G21.19 Other Medication-Induced Parkinsonism (807)

G21.0 Neuroleptic Malignant Syndrome (810)

G24.02 Medication-Induced Acute Dystonia (812)

G25.71 Medication-Induced Acute Akathisia (813)

G24.01 Tardive Dyskinesia (814)

G24.09 Tardive Dystonia (816)

G25.71 Tardive Akathisia (816)

G25.1 Medication-Induced Postural Tremor (817)

G25.79 Other Medication-Induced Movement Disorder (818)

____.__ Antidepressant Discontinuation Syndrome (818)

T43.205A Initial encounter

T43.205D Subsequent encounter

T43.205S Sequelae

____.__ Other Adverse Effect of Medication (819)

T50.905A Initial encounter

T50.905D Subsequent encounter

T50.905S Sequelae

Other Conditions That May Be a Focus of Clinical Attention (821)

Suicidal Behavior and Nonsuicidal Self-Injury (822)

Suicidal Behavior (822)

____.__ Current Suicidal Behavior (822)

T14.91A Initial encounter

T14.91D Subsequent encounter

Z91.51 History of Suicidal Behavior (822)

Nonsuicidal Self-Injury (822)

R45.88 Current Nonsuicidal Self-Injury (822)

Z91.52 History of Nonsuicidal Self-Injury (822)

Abuse and Neglect (822)

Child Maltreatment and Neglect Problems (823)

Child Physical Abuse (823)

____.__ Child Physical Abuse, Confirmed (823)

T74.12XA Initial encounter

T74.12XD Subsequent encounter

____.___ Child Physical Abuse, Suspected (823)

T76.12XA Initial encounter

T76.12XD Subsequent encounter

____.___ Other Circumstances Related to Child Physical Abuse (823)

Z69.010 Encounter for mental health services for victim of child physical abuse by parent

Z69.020 Encounter for mental health services for victim of nonparental child physical abuse

Z62.810 Personal history (past history) of physical abuse in childhood

Z69.011 Encounter for mental health services for perpetrator of parental child physical abuse

Z69.021 Encounter for mental health services for perpetrator of nonparental child physical abuse

Child Sexual Abuse (823)

____.___ Child Sexual Abuse, Confirmed (824)

T74.22XA Initial encounter

T74.22XD Subsequent encounter

____.___ Child Sexual Abuse, Suspected (824)

T76.22XA Initial encounter

T76.22XD Subsequent encounter

____.___ Other Circumstances Related to Child Sexual Abuse (824)

Z69.010 Encounter for mental health services for victim of child sexual abuse by parent

Z69.020 Encounter for mental health services for victim of nonparental child sexual abuse

Z62.810 Personal history (past history) of sexual abuse in

childhood

Z69.011 Encounter for mental health services for perpetrator of parental child sexual abuse

Z69.021 Encounter for mental health services for perpetrator of nonparental child sexual abuse

Child Neglect (824)

____.__ Child Neglect, Confirmed (824)

T74.02XA Initial encounter

T74.02XD Subsequent encounter

____.__ Child Neglect, Suspected (824)

T76.02XA Initial encounter

T76.02XD Subsequent encounter

____.__ Other Circumstances Related to Child Neglect (824)

Z69.010 Encounter for mental health services for victim of child neglect by parent

Z69.020 Encounter for mental health services for victim of nonparental child neglect

Z62.812 Personal history (past history) of neglect in childhood

Z69.011 Encounter for mental health services for perpetrator of parental child neglect

Z69.021 Encounter for mental health services for perpetrator of nonparental child neglect

Child Psychological Abuse (825)

____.__ Child Psychological Abuse, Confirmed (825)

T74.32XA Initial encounter

T74.32XD Subsequent encounter

____.__ Child Psychological Abuse, Suspected (825)

T76.32XA Initial encounter

T76.32XD Subsequent encounter

____.__ Other Circumstances Related to Child Psychological Abuse (825)

Z69.010 Encounter for mental health services for victim of child psychological abuse by parent

Z69.020 Encounter for mental health services for victim of nonparental child psychological abuse

Z62.811 Personal history (past history) of psychological abuse in childhood

Z69.011 Encounter for mental health services for perpetrator of parental child psychological abuse

Z69.021 Encounter for mental health services for perpetrator of nonparental child psychological abuse

Adult Maltreatment and Neglect Problems (825)

Spouse or Partner Violence, Physical (825)

____.__ Spouse or Partner Violence, Physical, Confirmed (825)

T74.11XA Initial encounter

T74.11XD Subsequent encounter

____.__ Spouse or Partner Violence, Physical, Suspected (826)

T76.11XA Initial encounter

T76.11XD Subsequent encounter

____.__ Other Circumstances Related to Spouse or Partner Violence, Physical (826)

Z69.11 Encounter for mental health services for victim of spouse or partner violence, physical

Z91.410 Personal history (past history) of spouse or partner violence, physical

Z69.12 Encounter for mental health services for perpetrator of

spouse or partner violence, physical

Spouse or Partner Violence, Sexual (826)

____.___ Spouse or Partner Violence, Sexual, Confirmed (826)

T74.21XA Initial encounter

T74.21XD Subsequent encounter

____.___ Spouse or Partner Violence, Sexual, Suspected (826)

T76.21XA Initial encounter

T76.21XD Subsequent encounter

____.___ Other Circumstances Related to Spouse or Partner Violence, Sexual (826)

Z69.81 Encounter for mental health services for victim of spouse or partner violence, sexual

Z91.410 Personal history (past history) of spouse or partner violence, sexual

Z69.12 Encounter for mental health services for perpetrator of spouse or partner violence, sexual

Spouse or Partner Neglect (826)

____.___ Spouse or Partner Neglect, Confirmed (826)

T74.01XA Initial encounter

T74.01XD Subsequent encounter

____.___ Spouse or Partner Neglect, Suspected (827)

T76.01XA Initial encounter

T76.01XD Subsequent encounter

____.___ Other Circumstances Related to Spouse or Partner Neglect (827)

Z69.11 Encounter for mental health services for victim of spouse or partner neglect

Z91.412 Personal history (past history) of spouse or partner neglect

Z69.12 Encounter for mental health services for perpetrator of spouse or partner neglect

Spouse or Partner Abuse, Psychological (827)

____.___ Spouse or Partner Abuse, Psychological, Confirmed (827)

T74.31XA Initial encounter

T74.31XD Subsequent encounter

____.___ Spouse or Partner Abuse, Psychological, Suspected (827)

T76.31XA Initial encounter

T76.31XD Subsequent encounter

____.___ Other Circumstances Related to Spouse or Partner Abuse, Psychological (827)

Z69.11 Encounter for mental health services for victim of spouse or partner psychological abuse

Z91.411 Personal history (past history) of spouse or partner psychological abuse

Z69.12 Encounter for mental health services for perpetrator of spouse or partner psychological abuse

Adult Abuse by Nonspouse or Nonpartner (827)

____.___ Adult Physical Abuse by Nonspouse or Nonpartner, Confirmed (828)

T74.11XA Initial encounter

T74.11XD Subsequent encounter

____.___ Adult Physical Abuse by Nonspouse or Nonpartner, Suspected (828)

T76.11XA Initial encounter

T76.11XD Subsequent encounter

____.___ Adult Sexual Abuse by Nonspouse or Nonpartner, Confirmed (828)

T74.21XA Initial encounter

T74.21XD Subsequent encounter

____.___ Adult Sexual Abuse by Nonspouse or Nonpartner, Suspected (828)

T76.21XA Initial encounter

T76.21XD Subsequent encounter

____.___ Adult Psychological Abuse by Nonspouse or Nonpartner, Confirmed (828)

T74.31XA Initial encounter

T74.31XD Subsequent encounter

____.___ Adult Psychological Abuse by Nonspouse or Nonpartner, Suspected (828)

T76.31XA Initial encounter

T76.31XD Subsequent encounter

____.___ Other Circumstances Related to Adult Abuse by Nonspouse or Nonpartner (828)

Z69.81 Encounter for mental health services for victim of nonspousal or nonpartner adult abuse

Z69.82 Encounter for mental health services for perpetrator of nonspousal or nonpartner adult abuse

Relational Problems (828)

____.___ Parent-Child Relational Problem (829)

Z62.820 Parent–Biological Child (829)

Z62.821 Parent–Adopted Child (829)

Z62.822 Parent–Foster Child (829)

Z62.898 Other Caregiver–Child (829)

Z62.891 Sibling Relational Problem (829)

Z63.0 Relationship Distress With Spouse or Intimate Partner (829)

Problems Related to the Family Environment (829)

Z62.29 Upbringing Away From Parents (829)

Z62.898 Child Affected by Parental Relationship Distress (830)

Z63.5 Disruption of Family by Separation or Divorce (830)

Z63.8 High Expressed Emotion Level Within Family (830)

Educational Problems (830)

Z55.0 Illiteracy and Low-Level Literacy (830)

Z55.1 Schooling Unavailable and Unattainable (830)

Z55.2 Failed School Examinations (830)

Z55.3 Underachievement in School (830)

Z55.4 Educational Maladjustment and Discord With Teachers and Classmates (830)

Z55.8 Problems Related to Inadequate Teaching (830)

Z55.9 Other Problems Related to Education and Literacy (830)

Occupational Problems (830)

Z56.82 Problem Related to Current Military Deployment Status (830)

Z56.0 Unemployment (831)

Z56.1 Change of Job (831)

Z56.2 Threat of Job Loss (831)

Z56.3 Stressful Work Schedule (831)

Z56.4 Discord With Boss and Workmates (831)

Z56.5 Uncongenial Work Environment (831)

Z56.6 Other Physical and Mental Strain Related to Work (831)

Z56.81 Sexual Harassment on the Job (831)

Z56.9 Other Problem Related to Employment (831)

Housing Problems (831)

Z59.01 Sheltered Homelessness (831)

Z59.02 Unsheltered Homelessness (831)

Z59.1 Inadequate Housing (831)

Z59.2 Discord With Neighbor, Lodger, or Landlord (831)

Z59.3 Problem Related to Living in a Residential Institution (831)

Z59.9 Other Housing Problem (831)

Economic Problems (831)

Z59.41 Food Insecurity (832)

Z58.6 Lack of Safe Drinking Water (832)

Z59.5 Extreme Poverty (832)

Z59.6 Low Income (832)

Z59.7 Insufficient Social or Health Insurance or Welfare Support (832)

Z59.9 Other Economic Problem (832)

Problems Related to the Social Environment (832)

Z60.2 Problem Related to Living Alone (832)

Z60.3 Acculturation Difficulty (832)

Z60.4 Social Exclusion or Rejection (832)

Z60.5 Target of (Perceived) Adverse Discrimination or Persecution (832)

Z60.9 Other Problem Related to Social Environment (832)

Problems Related to Interaction With the Legal System (832)

Z65.0 Conviction in Criminal Proceedings Without Imprisonment (833)

Z65.1 Imprisonment or Other Incarceration (833)

Z65.2 Problems Related to Release From Prison (833)

Z65.3 Problems Related to Other Legal Circumstances (833)

Problems Related to Other Psychosocial, Personal, and Environmental Circumstances (833)

Z72.9 Problem Related to Lifestyle (833)

Z64.0 Problems Related to Unwanted Pregnancy (833)

Z64.1 Problems Related to Multiparity (833)

Z64.4 Discord With Social Service Provider, Including Probation Officer, Case Manager, or Social Services Worker (833)

Z65.4 Victim of Crime (833)

Victim of Terrorism or Torture (833)

Z65.4

Z65.5 Exposure to Disaster, War, or Other Hostilities (833)

Problems Related to Access to Medical and Other Health Care (833)

Z75.3 Unavailability or Inaccessibility of Health Care Facilities (833)

Z75.4 Unavailability or Inaccessibility of Other Helping Agencies (833)

Circumstances of Personal History (833)

Z91.49 Personal History of Psychological Trauma (833)

Z91.82 Personal History of Military Deployment (833)

Other Health Service Encounters for Counseling and Medical Advice (833)

Z31.5 Genetic Counseling (833)

Z70.9 Sex Counseling (834)

Z71.3 Dietary Counseling (834)

Z71.9 Other Counseling or Consultation (834)

Additional Conditions or Problems That May Be a Focus of Clinical Attention (834)

Z91.83 Wandering Associated With a Mental Disorder (834)

Z63.4 Uncomplicated Bereavement (834)

Z60.0 Phase of Life Problem (834)

Z65.8 Religious of Spiritual Problem (834)

Z72.811 Adult Antisocial Behavior (835)

Z72.810 Child or Adolescent Antisocial Behavior (835)

Z91.19 Nonadherence to Medical Treatment (835)

E66.9 Overweight or Obesity (835)

Z76.5 Malingering (835)

R41.81 Age-Related Cognitive Decline (835)

R41.83 Borderline Intellectual Functioning (836)

SECTION I

DSM-5 Basics

Introduction

Use of the Manual

Cautionary Statement for Forensic Use of DSM-5

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This section provides a basic orientation to the purpose, structure, content, and use of DSM-5. The Introduction begins with a description of the DSM-5 and DSM-5-TR revision processes, followed by an overview of the DSM-5 organizational structure (e.g., regrouping of disorders, harmonization with ICD-11) and key conceptual issues, such as the definition of a mental disorder, categorical and dimensional approaches to diagnosis, cultural and social structural issues, and sex and gender differences. Use of the Manual features information to facilitate the use of DSM-5, such as a brief overview of the diagnostic process, use of subtypes and specifiers, other specified and unspecified mental disorder categories, use of clinical judgment, coding and recording procedures, notes about terminology, descriptions of the types of information in the DSM-5-TR text, and online enhancements. The section concludes with a Cautionary Statement for Forensic Use of DSM-5.

Introduction

The creation of the fifth edition of *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) was a massive undertaking that involved hundreds of people working toward a common goal over a 12-year period. Much thought and deliberation were involved in evaluating the diagnostic criteria, considering the organization of every aspect of the manual, and creating new features believed to be most useful to clinicians, such as dimensional measures that can help to identify emerging symptoms or determine and monitor changes in severity. All of these efforts have been directed toward the goal of enhancing the usefulness of DSM-5 as a clinical guide in the diagnosis of mental disorders, as well as its value for research.

DSM-5 strives to fulfill the need of clinicians, patients, families, and researchers for a clear and concise description of each mental disorder, which has been operationalized using diagnostic criteria that are supplemented by dimensional measures of severity and is accompanied by a digest of information about the diagnosis, including risk factors and culture- and sex- and gender-related issues.

Clinical training and experience are needed to use DSM for determining a clinical diagnosis. The diagnostic criteria identify symptoms and signs comprising affects, behaviors, cognitive functions, and personality traits along with physical signs, symptom combinations (syndromes), and durations that require clinical expertise to differentiate from normal variation and transient responses to stress. The diagnostic process can be facilitated by a thorough examination of the range of symptoms that might be present, such as by conducting a review of mental systems using the DSM-5 Level 1 Cross-Cutting Symptom Measure as recommended by The American Psychiatric Association (APA) *Practice Guidelines for the Psychiatric Evaluation of Adults* (see “Cross-Cutting Symptom Measures”).

The use of DSM criteria has the clear virtue of creating a common language for communication between clinicians about the diagnosis of

disorders. Officially recognized disorders are located in Section II of the manual. However, it should be noted that these diagnostic criteria and their placement within the classification are based on current research and may need to be modified as research advances.

Development of DSM-5-TR

Brief History of Prior DSM Editions

The first edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* appeared in 1952. This was the first official manual of mental disorders to contain a glossary of descriptions of the diagnostic categories. The use of the term "reaction" throughout the classification reflected the influence of Adolf Meyer's psychobiological view that mental disorders represented reactions of the personality to psychological, social, and biological factors. In the development of the second edition (DSM-II), a decision was made to base the classification on the mental disorders section of

the eighth revision of the *International Classification of Diseases* (ICD-8), for which representatives of the APA had provided consultation. Both DSM-II and ICD-8 went into effect in 1968.

As had been the case for DSM-I and DSM-II, the development of DSM-III was coordinated with the development of the *International Classification of Diseases*, specifically ICD-9, which was published in 1975 and implemented in 1978. Work began on DSM-III in 1974, with publication in 1980. DSM-III, under the direction of Robert L. Spitzer, M.D., introduced a number of important methodological innovations, including explicit diagnostic criteria and a descriptive approach that attempted to be neutral with respect to theories of etiology of mental disorders. Experience with DSM-III revealed a number of inconsistencies in the system. Therefore, the APA appointed a Workgroup to Revise DSM-III,

which developed the revisions and corrections that led to DSM-III-R in 1987.

DSM-IV was published in 1994. It was the culmination of a 6-year effort that involved more than 1,000 individuals and numerous professional organizations. Much of the effort involved conducting a comprehensive review of the literature to establish a firm empirical basis for making modifications. Developers of DSM-IV and the tenth revision of the ICD worked closely to coordinate their efforts, resulting in increased congruence between the two systems. ICD-10 was published in 1992.

A thorough history of all DSM editions is located on the APA website: https://www.psychiatry.org/psychiatrists/practice/dsm/history-of-the-dsm.

DSM-5 Revision Process

In 1999, the APA launched an evaluation of the strengths and weaknesses of DSM. This effort was coordinated with the World Health Organization's (WHO) Division of Mental Health, the World Psychiatric Association, and the National Institute of Mental Health (NIMH) in the form of several conferences, the proceedings of which were published in the 2002 monograph *A Research Agenda for DSM-V*. Thereafter, from 2003 to 2008, a cooperative agreement between the APA and WHO, supported by the NIMH, the National Institute on Drug Abuse (NIDA), and the National Institute on Alcohol Abuse and Alcoholism (NIAAA), led to the convening of 13 international DSM-5 research planning conferences, involving 400 participants from 39 countries, to review the world literature in specific diagnostic areas to prepare for revisions in developing both DSM-5 and the *International Classification of Diseases*, 11th Revision (ICD-11). Reports from these conferences formed the basis for future DSM-5 Task Force reviews and set the stage for the new edition of DSM.

In 2006, the APA named David J. Kupfer, M.D., as Chair and Darrel A. Regier, M.D., M.P.H., as Vice-Chair of the DSM-5 Task Force. They were charged with recommending chairs for the 13 diagnostic work groups and additional task force members with a multidisciplinary range of expertise who would oversee the development of DSM-5. An additional vetting process was initiated by the APA Board of Trustees to disclose sources of income and thus avoid conflicts of interest by task force and work group

members. The full disclosure of all income and research grants from commercial sources, including the pharmaceutical industry, in the previous 3 years, the imposition of an income cap from all commercial sources, and the publication of disclosures on a website set a new standard for the field. Thereafter, the task force of 28 members was approved in 2007, and appointments of more than 130 work group members were approved in 2008. More than 400 additional work group advisors with no voting authority were also approved to participate in the process. A clear concept of the next evolutionary stage for the classification of mental disorders was central to the efforts of the task force and the work groups. This vision emerged as the task force and work groups recounted the history of DSM-IV's classification, its current strengths and limitations, and strategic directions for its revision.

An intensive 6-year process involved conducting literature reviews and secondary analyses, publishing research reports in scientific journals, developing draft diagnostic criteria, posting preliminary drafts on the DSM-5 website for public comment, presenting preliminary findings at professional meetings, performing field trials, and revising criteria and text. Overall, many health professional and educational groups were involved in this development and testing of DSM-5, including physicians, psychologists, social workers, nurses, counselors, epidemiologists, statisticians, neuroscientists, and neuropsychologists. Also, individuals with mental disorders and families of individuals with mental disorders, lawyers, consumer organizations, and advocacy groups participated in revising DSM-5 by providing feedback on the mental disorders described in this volume.

Proposals for Revisions

Proposals for the revision of DSM-IV diagnostic criteria were developed by members of the work groups on the basis of rationale, scope of change, expected impact on clinical management and public health, strength of the supporting research evidence, overall clarity, and clinical utility. Proposals

encompassed changes to diagnostic criteria; the addition of new disorders, subtypes, and specifiers; and the deletion of existing disorders.

In the proposals for revisions, strengths and weaknesses in the current criteria and nosology were first identified. Novel scientific findings over the previous two decades were considered, leading to the creation of a research plan to assess potential changes through literature reviews and secondary data analyses. Four principles guided the draft revisions: 1) DSM-5 is primarily intended to be a manual to be used by clinicians, and revisions must be feasible for routine clinical practice; 2) recommendations for revisions should be guided by research evidence; 3) where possible, continuity should be maintained with previous editions of DSM; and 4) no a priori constraints should be placed on the degree of change between DSM-IV and DSM-5.

Building on the initial literature reviews, work groups identified key issues within their diagnostic areas. Work groups also examined broader methodological concerns, such as the presence of contradictory findings within the literature; development of a refined definition of mental disorder; and a consideration of cross-cutting issues relevant to all disorders. Inclusion of a proposal for revision in Section II was informed by consideration of its advantages and disadvantages for public health and clinical utility, the strength of the evidence, and the magnitude of the change. New diagnoses and disorder subtypes and specifiers were subject to additional stipulations, such as demonstration of reliability (i.e., the degree to which two clinicians could independently arrive at the same diagnosis for a given patient). Disorders with low clinical utility and weak validity were considered for deletion. Placement of proposed conditions in “Conditions for Further Study” in Section III was contingent on the amount of empirical evidence generated on the proposed diagnosis, diagnostic reliability or validity, presence of clear clinical need, and potential benefit in advancing research.

DSM-5 Field Trials

The use of field trials to empirically demonstrate reliability was a noteworthy improvement introduced in DSM-III. The design and implementation strategy of the DSM-5 Field Trials represented several

changes from approaches used for DSM-III and DSM-IV, particularly in obtaining data on the precision of kappa reliability estimates (a statistical measure that assesses level of agreement between raters that corrects for chance agreement due to prevalence rates) in the context of real-world clinical settings with high levels of diagnostic comorbidity. For DSM-5, field trials were extended by using two distinctive designs: one in large, diverse medical-academic settings and the other in routine clinical practices. The former capitalized on the need for large sample sizes to test hypotheses on

reliability and clinical utility of a range of diagnoses in a variety of patient populations; the latter supplied valuable information about how proposed revisions performed in everyday clinical settings among a diverse sample of DSM users. It is anticipated that future clinical and basic research studies will focus on the validity of the revised categorical diagnostic criteria and the underlying dimensional features of these disorders (including those now being explored by the NIMH Research Domain Criteria initiative).

The medical-academic field trials were conducted at 11 North American medical-academic sites from December 2010 to October 2011 and assessed the reliability, feasibility, and clinical utility of select revisions, with priority given to those that represented the greatest degree of change from DSM-IV or those potentially having the greatest public health impact. The full clinical patient populations coming to each site were screened for DSM-IV diagnoses or qualifying symptoms likely to predict several specific DSM-5 disorders of interest. Stratified samples of four to seven specific disorders, plus a stratum containing a representative sample of all other diagnoses, were identified for each site. Patients consented to the study and were randomly assigned for a clinical interview by a clinician blind to the clinical diagnosis, followed by a second interview that occurred within 2 weeks with a clinician blind to the diagnoses made by the first interviewer. Patients first filled out a computer-assisted inventory of cross-cutting symptoms in more than a dozen psychological domains. These inventories were scored by a computer, and results were provided to clinicians before they conducted a typical clinical interview (with no structured protocol).

Clinicians were required to score the presence of qualifying criteria on a computer-assisted DSM-5 diagnostic checklist, determine diagnoses, score the severity of the diagnosis, and upload the data to a central repository. This study design allowed the calculation of the degree to which two independent clinicians could agree on a diagnosis (using the intraclass kappa statistic) and on clinician-administered ratings of cross-cutting and diagnosis-specific symptom severity (using intraclass correlation coefficients), as well as the level of agreement on measures of self-reported cross-cutting symptoms, personality traits, disability, and diagnostic severity administered to the same patient on two occasions up to 2 weeks apart (using intraclass correlation coefficients), along with information on the precision of these estimates of reliability. It was also possible to assess the prevalence rates of both DSM-IV and DSM-5 conditions in the respective clinical populations.

The routine clinical practice field trials involved recruitment of individual psychiatrists and other mental health clinicians and were conducted from October 2011 to March 2012. A volunteer sample was recruited that included generalist and specialty psychiatrists, psychologists, licensed clinical social workers, counselors, marriage and family therapists, and advanced practice psychiatric mental health nurses. The field trials provided exposure of the proposed DSM-5 diagnoses and dimensional measures to a wide range of clinicians to assess their feasibility and clinical utility.

Public and Professional Review

In 2010, the APA launched a website devoted to the DSM-5 revision to facilitate public and professional input into DSM-5. All draft diagnostic criteria and proposed changes in organization were posted on www.dsm5.org for a 2-month comment period. Feedback totaled more than 8,000 submissions, which were systematically reviewed by each of the 13 work groups, whose members, where appropriate, integrated questions and comments into discussions of draft revisions and plans for field trial testing. After revisions to the initial draft criteria and proposed chapter organization, a second posting occurred in 2011. Work groups considered feedback from both web postings and the results of the DSM-5 Field Trials

when drafting proposed final criteria, which were posted on the website for a third and final time in 2012. These three iterations of external review produced more than 13,000 individually signed comments on the website that were received and reviewed by the work

groups, plus thousands of organized petition signers for and against some proposed revisions, all of which allowed the task force to actively address concerns of DSM users, as well as patients and advocacy groups, and ensure that clinical utility remained a high priority.

Expert Review and Final Approval

The members of the 13 work groups, representing expertise in their respective areas, collaborated with advisors and reviewers under the overall direction of the DSM-5 Task Force to draft the diagnostic criteria and accompanying text. This effort was supported by a team of APA Division of Research staff and developed through a network of text coordinators from each work group. The preparation of the text was coordinated by the text editor, working in close collaboration with the work groups and under the direction of the task force chairs. The Scientific Review Committee (SRC) was established to provide a scientific peer review process that was external to that of the work groups. The SRC chair, vice-chair, and six committee members were charged with reviewing the degree to which the proposed changes from DSM-IV could be supported with scientific evidence. Each proposal for diagnostic revision required a memorandum of evidence for change prepared by the work group and accompanied by a summary of supportive data organized around validators for the proposed diagnostic criteria (i.e., antecedent validators such as familial aggregation, concurrent validators such as biological markers, and prospective validators such as response to treatment or course of illness). The submissions were reviewed by the SRC and scored according to the strength of the supportive scientific data. Other justifications for change, such as those arising from clinical experience or need or from a conceptual reframing of diagnostic categories, were generally seen as outside the purview of the SRC. The reviewers’

scores, which varied substantially across the different proposals, and an accompanying brief commentary were then returned to the APA Board of Trustees and the work groups for consideration and response.

The Clinical and Public Health Committee (CPHC), composed of a chair, vice-chair, and six members, was appointed to consider additional clinical utility, public health, and logical clarification issues for criteria that had not yet accumulated the type or level of evidence deemed sufficient for change by the SRC. This review process was particularly important for DSM-IV disorders with known deficiencies for which proposed remedies had neither been previously considered in the DSM revision process nor been subjected to replicated research studies. These selected disorders were evaluated by four to five external reviewers, and the blinded results were reviewed by CPHC members, who in turn made recommendations to the APA Board of Trustees and the work groups.

Forensic reviews of diagnostic criteria and text were conducted by the members of the APA Council on Psychiatry and Law for disorders frequently appearing in forensic environments and ones with high potential for influencing civil and criminal judgments in courtroom settings. Work groups also added forensic experts as advisors in pertinent areas to complement expertise provided by the Council on Psychiatry and Law.

A final recommendation from the task force was then provided to the APA Assembly's Committee on DSM-5 to consider some of the clinical utility and feasibility features of the proposed revisions. The Assembly is a deliberative body of the APA representing the district branches and wider membership that is composed of psychiatrists from throughout the United States who provide geographic, practice size, and interest-based diversity. The Committee on DSM-5 was composed of a diverse group of Assembly leaders.

Following the preceding review steps, executive "summit committee" sessions were held to consolidate input from review, including Assembly committee chairs, task force chairs, a forensic advisor, and a statistical advisor, for a preliminary review of each disorder by the Assembly and APA Board of Trustees executive committees. This preceded a preliminary review by the full APA Board of Trustees. The Assembly voted, in November 2012, to recommend that the Board approve the publication of DSM-5, and the APA Board

of Trustees approved its publication in December 2012. The many experts, reviewers, and advisors who contributed to this process are listed in the Appendix.

Revisions to DSM-5

DSM-5 Iterative Revision Process

Advances in digital publishing that allow timely dissemination of changes have paved the way for the American Psychiatric Association to adopt an iterative improvement model for DSM, in which revisions are pegged to specific scientific advances. The DSM Steering Committee (analogous to the DSM-5 Task Force) was appointed in Spring 2014, with Paul S. Appelbaum, M.D., as Chair and Ellen Leibenluft, M.D., and Kenneth Kendler, M.D., as Vice Chairs, to oversee the iterative revision process, along with the establishment of a web portal (www.dsm5.org) to field proposals on a continuous basis. Proposed changes can include the addition of new disorders and deletion or modification of diagnostic criteria sets in Sections II and III of DSM-5, as well as changes to the text. Submissions must be accompanied by supportive information in a structured format, including the reasons for the change, the magnitude of change, data documenting improvements in validity across a range of validators, evidence of reliability and clinical utility, and consideration of current or potential deleterious consequences associated with the proposed change.

Approaches to validating diagnostic criteria for categorical mental disorders have included the following types of evidence: antecedent validators (similar genetic markers, family traits, temperament, and environmental exposure), concurrent validators (similar neural substrates, biomarkers, emotional and cognitive processing, and symptom similarity), and predictive validators (similar clinical course and treatment response). New criteria for current disorders are adopted if they produce improvement in some of these classes of validators. Furthermore, new disorders are added to DSM if they are shown to be valid by a substantial subset of these

validators as well as meet the criteria for a mental disorder and demonstrate clinical utility.

Proposals submitted to the DSM web portal undergo an initial review by the Steering Committee to determine whether the evidence for the proposal, on its face, appears substantially likely to meet the criteria for approval. If so, the proposal is referred to one of the five standing Review Committees (functionally analogous to the DSM Work Groups), which cover broad domains of psychiatric diagnosis. On receipt of a proposal from the Steering Committee, the assigned Review Committee considers the evidence in support of the proposed change, requests additional information if necessary, and returns the proposal to the Steering Committee with recommendations for disposition and, in some cases, suggested modifications. If the Steering Committee concurs that sufficient evidence appears to exist in support of the proposal, the proposed revision is posted on the DSM-5 website for public comment. The final stage involves making necessary adjustments based on the comments and then forwarding the final version to the APA Assembly and Board of Trustees for their approval. Once approved, the online version of the manual (see https://psychiatryonline.org) is updated to reflect the changes. All changes that have been approved since the publication of DSM-5 in 2013 are included in DSM-5-TR.

DSM-5 Text Revision Process

In Spring 2019, APA started work on DSM-5-TR, with Michael B. First, M.D., and Philip Wang, M.D., Dr.P.H., as Revision Subcommittee Co-Chairs and Wilson M. Compton, M.D., and Daniel S. Pine, M.D., as Revision Subcommittee Vice Chairs. The DSM-5-TR development effort involved more than 200 experts (the majority of whom were involved in the development of DSM-5), who were given the task of conducting literature reviews covering the past 10 years and reviewing the text to identify out-of-date material. A review of

conflicts of interest for all proposed changes to the text was conducted to eliminate any possible compromise of the objectivity of the content.

Mirroring the structure of the DSM-5 process, experts were divided into 20 Disorder review groups, each headed by a section editor. Four cross-cutting review groups (Culture, Sex and Gender, Suicide, and Forensic) reviewed all the chapters, focusing on material involving their specific expertise. The text was also reviewed by a work group on Ethnoracial Equity and Inclusion to ensure appropriate attention to risk factors such as racism and discrimination and the use of nonstigmatizing language. Although the scope of the text revision did not include conceptual changes to the criteria sets, some necessary clarifications to certain diagnostic criteria became apparent during the review of the text. Proposals for changes in diagnostic criteria or specifier definitions that were a result of the text revision process were reviewed and approved by the DSM Steering Committee, as well as the APA Assembly and Board of Trustees, as part of the DSM-5 iterative revision process described in the prior section.

Changes in DSM-5 Organizational Structure

DSM is a medical classification of disorders and as such serves as a historically determined cognitive schema imposed on clinical and scientific information to increase its comprehensibility and utility. The classification of disorders (the way in which disorders are grouped) provides a high-level organization for the manual.

Regrouping of Disorders in DSM-5

Members of the DSM-5 diagnostic spectra study group examined whether scientific validators could inform possible new groupings of related disorders within the existing categorical framework. Eleven such indicators were recommended by the study group for this purpose (i.e., their ability to meaningfully separate groups of psychiatric illness from each other): neural substrates, family traits, genetic risk factors, specific environmental risk factors, biomarkers, temperamental antecedents, abnormalities of emotional or cognitive processing, symptom similarity, course of illness, high

comorbidity, and shared treatment response. These indicators served the study group as empirical guidelines to inform decision-making by the work groups and the task force about how to cluster disorders to maximize their validity and clinical utility (i.e., the more likely that disorders shared these validators, the more likely they would be in the same diagnostic grouping).

A series of papers was developed and published in a prominent international journal (*Psychological Medicine*, Vol. 39, 2009) as part of both the DSM-5 and the ICD-11 developmental processes to document that such validators were most useful for suggesting large groupings of disorders, as well as for "validating" proposed changes to diagnostic criteria.

As the APA and WHO began to plan their respective revisions of DSM and the *International Classification of Diseases* (ICD), both considered the possibility of improving clinical utility (e.g., by helping to explain apparent comorbidity) and facilitating scientific investigation by rethinking the organizational structures of both publications. It was critical to both the DSM-5 Task Force and the WHO International Advisory Group on the revision of the ICD-10 Section on Mental and Behavioral Disorders that the revisions to organization enhance clinical utility and remain within the bounds of well-replicated

scientific information. In that spirit, revision of the organizational structure was approached as a conservative, evolutionary diagnostic reform that would be guided by emerging scientific evidence on the relationships between disorder groups. By reordering and regrouping the existing disorders, the revised structure is meant to stimulate new clinical perspectives and to encourage researchers to identify the psychological and physiological cross-cutting factors that are not bound by strict categorical designations.

Early in the course of the revisions, it became apparent that a shared organizational structure would help harmonize the classifications. To the surprise of participants in both revision processes, large sections of the content fell relatively easily into place, reflecting real strengths in some areas of the scientific literature, such as epidemiology, analyses of

comorbidity, twin studies, and certain other genetically informed designs. When disparities emerged, they almost always reflected the need to make a judgment about where to place a disorder in the face of incomplete—or, more often, conflicting—data. Thus, for example, on the basis of patterns of symptoms, comorbidity, and shared risk factors, attention-deficit/hyperactivity disorder (ADHD) was placed within the DSM-5 chapter "Neurodevelopmental Disorders," although the same data also supported strong arguments to place ADHD within the chapter "Disruptive, Impulse-Control, and Conduct Disorders." These issues were settled with the preponderance of evidence supporting placement in the "Neurodevelopmental Disorders" chapter in DSM-5.

The organization of chapters of DSM-5 after the neurodevelopmental disorders is based on groups of internalizing disorders (i.e., disorders with prominent anxiety, depressive, and somatic symptoms), externalizing disorders (i.e., disorders with prominent impulsive, disruptive conduct, and substance use symptoms), neurocognitive disorders, and other disorders. It is hoped that this organization will encourage further study of underlying pathophysiological processes that give rise to diagnostic comorbidity and symptom heterogeneity. Furthermore, by arranging disorder clusters to mirror clinical reality, DSM-5 should facilitate identification of potential diagnoses by non–mental health specialists, such as primary care physicians.

Despite the problem posed by categorical diagnoses, the DSM-5 Task Force recognized that it would have been premature scientifically to propose alternative dimensional definitions for most disorders. The organizational structure is meant to serve as a bridge to new diagnostic approaches without disrupting current clinical practice or research. It is anticipated that the more dimensional DSM-5 approach and organizational structure will facilitate research across current diagnostic categories by encouraging broad investigations within the proposed chapters and across adjacent chapters. Such research should also keep DSM-5 central to the development of dimensional approaches to diagnosis that will likely supplement or supersede current categorical approaches in coming years.

Combining Developmental and Life Span Considerations

To improve clinical utility, DSM-5 is organized along developmental and life span trajectories. It begins with diagnoses thought to reflect developmental processes that manifest early in life (e.g., neurodevelopmental disorders and schizophrenia spectrum and other psychotic disorders), followed by diagnoses that more commonly manifest in adolescence and young adulthood (e.g., bipolar and related disorders, depressive disorders, and anxiety disorders), and ends with diagnoses relevant to adulthood and later life (e.g., neurocognitive disorders). A similar approach has been taken, where possible, within each chapter. This organizational structure facilitates the comprehensive use of life span information as a way to assist in diagnostic decision-making.

Harmonization With ICD-11

The groups tasked with revising the DSM and ICD systems shared the overarching goal of harmonizing the two classifications as much as possible, for the following reasons:

- The existence of two major classifications of mental disorders hinders the collection and use of national health statistics, the design of clinical trials aimed at developing new treatments, and the consideration of global applicability of the results by international regulatory agencies.
- More broadly, the existence of two classifications complicates attempts to replicate scientific results across national boundaries.
- Even when the intention was to identify identical patient populations, DSM-IV and ICD-10 diagnoses did not always agree.

As discussed earlier in this introduction, the effort to harmonize with ICD-11 was confined to the largely successful harmonization of the organizational structure. Because of differences in timing, complete harmonization of the DSM-5 diagnostic criteria with the ICD-11 disorder definitions was not possible because the DSM-5 developmental effort was

several years ahead of the ICD-11 revision process. Consequently, the DSM-5 diagnostic criteria were finalized just as the ICD-11 working groups were beginning to develop the ICD-11 clinical descriptions and diagnostic guidelines. Some improvement in harmonization at the disorder level was still achieved; many ICD-11 working group members had participated in the development of the DSM-5 diagnostic criteria, and the ICD-11 working groups were instructed to review the DSM-5 criteria sets and to strive to make ICD-11 diagnostic guidelines as similar to DSM-5 as possible unless there was a considered reason for them to differ. A review comparing DSM-5/ICD-11 differences with DSM-IV/ICD-10 differences found that ICD and DSM are now closer than at any time since DSM-II and ICD-8 and that current differences are based largely on differing priorities and uses of the two diagnostic systems and on differing interpretations of the evidence.

Although ICD-11 was officially endorsed for use by WHO member nations during the 72nd World Health Assembly in May 2019 and officially came into effect on January 1, 2022, each country chooses when to adopt ICD-11. There is currently no proposed timeline for implementation of ICD-11 in the United States. Consequently, for the foreseeable future the official coding system in the United States continues to be the *International Classification of Diseases*, Tenth Revision, Clinical Modification (ICD-10-CM).

Key Conceptual Frameworks and Approaches

Definition of a Mental Disorder

Each disorder identified in Section II of the manual (excluding those in the chapters “Medication-Induced Movement Disorders and Other Adverse Effects of Medication” and “Other Conditions That May Be a Focus of Clinical Attention”) must meet the definition of a mental disorder. Although no definition can capture all aspects of the range of disorders contained in DSM-5, the following elements are required:

> A *mental disorder* is a syndrome characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behavior that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning. Mental disorders are usually associated with significant distress or disability in social, occupational, or other important activities. An expectable or culturally approved response to a common stressor or loss, such as the death of a loved one, is not a mental disorder. Socially deviant behavior (e.g., political, religious, or sexual) and conflicts that are primarily between the individual and society are not mental disorders unless the deviance or conflict results from a dysfunction in the individual, as described above.

The diagnosis of a mental disorder should have clinical utility: it should help clinicians to determine prognosis, treatment plans, and potential treatment outcomes for their patients. However, the diagnosis of a mental disorder is not equivalent to a need for treatment. Need for treatment is a complex clinical decision that takes into consideration symptom severity, symptom salience (e.g., the presence of suicidal thoughts), the individual's distress (mental pain) associated with the symptom(s), disability related to the individual's symptoms, risks and benefits of available treatments, and other factors (e.g., psychiatric symptoms complicating other illness). Clinicians may thus encounter individuals whose symptoms do not meet full criteria for a mental disorder but who demonstrate a clear need for treatment or care. The fact that some individuals do not show all symptoms indicative of a diagnosis should not be used to justify limiting their access to appropriate care.

It should be noted that the definition of mental disorder was developed for clinical, public health, and research purposes. Additional information is usually required beyond that contained in the DSM-5 diagnostic criteria in order to make legal judgments on such issues as criminal responsibility,

eligibility for disability compensation, and competency (see “Cautionary Statement for Forensic Use of DSM-5” at the conclusion of Section I).

Categorical and Dimensional Approaches to Diagnosis

Structural problems rooted in the categorical design of DSM have emerged in both clinical practice and research. Relevant evidence of such problems includes high rates of comorbidity among disorders, symptom heterogeneity within disorders, and the substantial need for other specified and unspecified diagnoses to classify the substantial number of clinical presentations that do not meet criteria for any of the specific DSM disorders. Studies of both genetic and environmental risk factors, whether based on twin designs, familial transmission, or molecular analyses, have also raised questions about whether a categorical approach is the optimal way to structure of the DSM system.

There is broad recognition that a too-rigid categorical system does not capture clinical experience or important scientific observations. The results of numerous studies of comorbidity and disease transmission in families, including twin studies and molecular genetic studies, make strong arguments for what many astute clinicians have long observed: the boundaries between many disorder “categories” are more fluid over the life course than has been recognized, and many symptoms that make up the essential features of a particular disorder may occur, at varying levels of severity, in many other disorders.

A dimensional approach classifies clinical presentations on the basis of quantification of attributes rather than the assignment to categories and works best in describing phenomena that are distributed continuously and that do not have clear boundaries. Although dimensional systems increase reliability and communicate more clinical information

(because they report clinical attributes that might be subthreshold in a categorical system), they also have serious limitations, and thus far, they have been less useful than categorical systems in clinical practice.

Numerical dimensional descriptions are much less familiar and vivid than are the category names of mental disorders. Moreover, as yet there is no agreement on the choice of the optimal dimensions to be used for classification purposes. Nonetheless, with the increasing research on, and familiarity with, dimensional systems and the establishment of clinically meaningful cut points to guide treatment decisions, greater acceptance of dimensional approaches both as a method of conveying clinical information and as a research tool is eventually likely.

For reasons of both clinical utility and compatibility with the categorical ICD classification required for coding, DSM-5 continues to be a primarily categorical classification with dimensional elements that divides mental disorders into types based on criteria sets with defining features. Despite the categorical framework, it is important to recognize that in DSM-5 there is no assumption that each category of mental disorder is a completely discrete entity with absolute boundaries dividing it from other mental disorders or from no mental disorder. There is also no assumption that all individuals described as having the same mental disorder are alike in all important ways. The clinician using DSM-5 should therefore consider that individuals sharing a diagnosis are likely to be heterogeneous even in regard to the defining features of the diagnosis and that boundary cases will be difficult to diagnose in any but a probabilistic fashion. This outlook allows greater flexibility in the use of the system, encourages more specific attention to boundary cases, and emphasizes the need to capture additional clinical information that goes beyond diagnosis.

Cross-Cutting Symptom Measures

Given that psychiatric pathologies are not reliably discrete with sharp boundaries from one another, clinicians need to shift their approach to assessment and look beyond the prototypical presentations that neatly coincide with DSM categories. To assist with that transition, Section III, “Emerging Measures and Models,” provides the DSM-5 Level 1 Cross-Cutting Symptom Measure, developed to help clinicians assess all major areas of psychiatric functioning (e.g., mood, psychosis, cognition, personality, sleep) and more thoroughly uncover possible disorders, atypical presentations, subsyndromal conditions, and coexistent pathologies. Just as

the review of systems performed in general medicine acts as an inventory designed to call attention to symptoms or signs that otherwise could have been overlooked, the DSM-5 Level 1 Cross-Cutting Symptom Measure acts as a review of mental systems, intended to aid clinicians in better identifying latent disorders and symptoms in need of more detailed assessment (and potentially in need of treatment). The DSM-5 Level 1 Cross-Cutting Symptom Measure is recommended as an important component of the psychiatric evaluation of individuals presenting for psychiatric care, with *The American Psychiatric Association Practice Guidelines for the Psychiatric Evaluation of Adults* endorsing its use as a first step in identifying and addressing the heterogeneity of symptoms across diagnostic categories. Self-, parent/guardian-, and child (age 11–17)–rated versions of the DSM-5 Level 1 Cross-Cutting Symptom Measure are available online without charge for clinical use at www.psychiatry.org/dsm5.

Removal of the DSM-IV Multiaxial System

DSM-IV offered a multiaxial system of recording diagnoses that involved an assessment on several axes, each of which referred to a different domain of information. DSM-5 has moved to a nonaxial documentation of diagnosis. Disorders and conditions formerly listed on Axis I (clinical disorders), Axis II (personality disorders and intellectual developmental disorders), and Axis III (other medical conditions) are listed together without formal differentiation, typically in order of clinical importance. Psychosocial and

contextual factors (formerly listed on Axis IV) are listed along with the diagnoses and conditions using Z codes in the chapter “Other Conditions That May Be a Focus of Clinical Attention.” DSM-IV Axis V consisted of the Global Assessment of Functioning (GAF) scale, representing the clinician's judgment of the individual’s overall level of “functioning on a hypothetical continuum of mental health–illness.” This scale has been replaced by the WHO Disability Assessment Schedule (WHODAS), which

is included in Section III of DSM-5 (see the chapter “Assessment Measures”). The WHODAS is based on the International Classification of Functioning, Disability and Health (ICF) for use across all of medicine and health care.

Cultural and Social Structural Issues

Mental disorders are defined and recognized by clinicians and others in the context of local sociocultural and community norms and values. Cultural contexts shape the experience and expression of the symptoms, signs, behaviors, and thresholds of severity that constitute criteria for diagnosis. Sociocultural contexts also shape aspects of identity (such as ethnicity or race) that confer specific social positions and differentially expose individuals to social determinants of health, including mental health. These cultural elements are transmitted, revised, and recreated within families, communities, and other social systems and institutions and change over time. Diagnostic assessment should include how an individual’s experiences, symptoms, and behaviors differ from relevant sociocultural norms and lead to difficulties in adaptation in his or her current life context. Clinicians should also take into account how individuals’ clinical presentations are influenced by their position within social structures and hierarchies that shape exposure to adversity and access to resources. Key aspects of sociocultural context relevant to diagnostic classification and assessment have been carefully considered in the development of DSM-5-TR.

Impact of Cultural Norms and Practices

The boundaries between normality and pathology vary across cultural contexts for specific types of behaviors. Thresholds of tolerance for specific symptoms or behaviors differ across cultural contexts, social settings, and families. Hence, the level at which an experience becomes problematic or is perceived as pathological will differ. The judgment that a given behavior, experience, or concern requires clinical attention depends on cultural norms that are internalized by the individual and applied by others around him or her, including family members and clinicians. To accurately assess potential signs and symptoms of psychopathology, clinicians should routinely

consider the impact of cultural meanings, identities, and practices on the causes and course of illness, for example, through any of the following factors: levels of vulnerability and the mechanisms of specific disorders (e.g., by amplifying fears that maintain panic disorder or health anxiety); social stigma and support generated by family and community responses to mental illness; coping strategies that enhance resilience in response to illness or help-seeking pathways to access health care of various types, including alternative and complementary treatments; and acceptance or rejection of a diagnosis and adherence to treatments, affecting the course of illness and recovery. Cultural contexts also affect the conduct of the clinical encounter, including the diagnostic interview. Cultural differences between the clinician/members of the treatment team and the individual have implications for the accuracy and acceptance of diagnosis, as well as for treatment decisions, prognostic considerations, and clinical outcomes.

Cultural Concepts of Distress

Historically, the construct of the culture-bound syndrome was a focus of work on descriptive phenomenology in cultural psychiatry and psychology. Since DSM-5, this construct has been replaced by three concepts that offer greater clinical utility:

1. *Cultural idiom of distress* refers to a behavior or linguistic term, phrase, or way of talking about symptoms, problems, or suffering among individuals with similar cultural backgrounds to express or communicate essential features of distress (e.g., stating, “I feel so depressed” to express low mood or discouragement that does not meet the threshold for major depressive disorder). An idiom of distress need not be associated with specific symptoms, syndromes, or perceived causes. It may be used to convey a wide range of discomfort, including everyday concerns, subclinical conditions, or suffering due to social circumstances rather than mental disorders. For example, most cultural groups have common bodily idioms of distress used to express a wide range of suffering and concerns.

2. *Cultural explanation or perceived cause* refers to a label, attribution, or feature of an explanatory model that provides a culturally coherent concept of etiology or cause for symptoms, illness, or distress (e.g., the attribution of psychopathology to "stress," spirits, or failure to follow culturally prescribed practices). Causal explanations may be salient features of local classifications of disease used by laypersons or healers.
3. *Cultural syndrome* refers to a cluster or group of co-occurring, distinctive symptoms found in specific cultural groups, communities, or contexts (e.g., *ataque de nervios* [attack of nerves]). The syndrome may or may not be recognized as an illness in the local cultural context (i.e., it might be labeled in nonmedical ways), but such cultural patterns of distress and features of illness may nevertheless be recognizable by an outside observer.

These three concepts (for which discussion and examples are provided in the Section III chapter "Culture and Psychiatric Diagnosis") suggest cultural ways of understanding and describing experiences of distress or illness that can be elicited in the clinical encounter. They influence symptomatology, help-seeking, clinical presentations, expectations of treatment, illness adaptation, and treatment response. The same cultural term often serves more than one of these functions, and usage can change over time. For example, "depression" names a syndrome but has also become a common idiom of distress.

Impact of Racism and Discrimination on Psychiatric Diagnosis

Clinical work and research in psychiatry are deeply affected by social and cultural constructions of race and ethnicity. Race is a social, not a biological, construct. It is used to divide people into groups based on superficial physical traits such as skin color. Although there is no biological basis for the construct of race, discriminatory practices based on race have profound effects on physical and mental health.

The social process by which specific categories of identity are constructed on the basis of racial ideologies and practices is termed *racialization*. Racialized identities are important because they are strongly associated with systems of discrimination, marginalization, and social

exclusion. Other aspects of identity, including ethnicity, gender, language, religion, and sexual orientation, may also be the focus of bias or stereotyping that can affect the process of diagnostic assessment.

Racism exists at personal, interpersonal, systemic/institutional, and social structural levels. At the personal level, racism gives rise to internalized stereotypes and experiences of threat, devaluation, neglect, and injustice that affect individuals' health and well-being. At the interpersonal level, racism includes not only explicit behaviors but also *microaggressions*, which are everyday slights and offenses that communicate negative attitudes toward specific stigmatized groups, with stress-inducing and traumatizing consequences. Systemic/institutional racism refers to the ways that discrimination is embedded in everyday practices of institutions or organizations, including health care and psychiatry. Systemic racism may not be expressed in overt racial ideologies but may be maintained by

implicit and unintentional biases, habits, routines, and practices that result in misrecognition and inequity. As a result, individuals can participate in and inadvertently contribute to systemic racism without consciously endorsing racist ideas. The concept of social structural racism emphasizes the ways that racism and discrimination are manifested in the organization and norms of society and public policy with pervasive inequities in economic resources, power, and privilege that impact exposure to health risk and access to health care. The structural violence and oppression of racism have physical, psychological, and social consequences, including negative effects on mental health.

Racism is an important social determinant of health that contributes to a wide variety of adverse health outcomes, including hypertension, suicidal behavior, and posttraumatic stress disorder and can predispose individuals to substance use, mood disorders, and psychosis. Negative racial stereotypes and attitudes affect the psychological development and well-being of racialized groups. Other adverse consequences of discrimination include unequal access to care and clinician bias in diagnosis and treatment; for example, misdiagnosis of schizophrenia among African Americans

presenting with mood disorders and other conditions, more coercive pathways to care, less time in outpatient treatment, and more frequent use of physical restraints and suboptimal treatments. Clinicians should make active efforts to recognize and address all forms of racism, bias, and stereotyping in clinical assessment, diagnosis, and treatment.

Attention to Culture, Racism, and Discrimination in DSM-5-TR

During the DSM-5-TR review process, steps have been taken to address the impact of culture, racism, and discrimination on psychiatric diagnosis in the text of the disorder chapters. A Cross-Cutting Review Committee on Cultural Issues, composed of 19 U.S.-based and international experts in cultural psychiatry, psychology, and anthropology, reviewed the texts for cultural influences on disorder characteristics, incorporating relevant information in the sections on culture-related diagnostic issues. A separate Ethnoracial Equity and Inclusion Work Group, composed of 10 mental health practitioners from diverse ethnic and racialized backgrounds with expertise in disparity-reduction practices, reviewed references to race, ethnicity, and related concepts to avoid perpetuating stereotypes or including discriminatory clinical information.

DSM-5-TR is committed to the use of language that challenges the view that races are discrete and natural entities. The text uses terminology such as *racialized* instead of *racial* to highlight the socially constructed nature of race. When the term *ethnoracial* is used in the text, it denotes the U.S. Census categories, such as Hispanic, White, or African American, that combine ethnic and racialized identifiers. The emerging term *Latinx* (singular and plural) is used in place of *Latino/a* to promote gender-inclusive terminology. The term *Caucasian* is not used because it is based on obsolete and erroneous views about the geographic origin of a prototypical pan-European ethnicity. The terms *minority* and *non-White* are avoided because they describe social groups in relation to a racialized "majority," a practice that tends to perpetuate social hierarchies. When necessary for clarity in reporting epidemiological or other information based on specific studies, however, the text uses the group labels from the relevant studies. The term *culture* is used not to refer to a discrete social

group (e.g., “prevalence differs across cultures”) but rather to indicate the heterogeneity of cultural views and practices within societies; the terms *cultural contexts* or *cultural backgrounds* are preferred instead.

The sections on prevalence for each disorder were reviewed to ensure that findings are presented with clear reference to the geographic areas or social groups included in data collection (e.g., “in the U.S. general population”); this avoids overgeneralizing the findings to communities not yet studied. Prevalence data on specific ethnoracial groups were included when existing research documented reliable estimates based on representative

samples. The work group was concerned that data from nonrepresentative samples may be misleading. This explains the limited inclusion of data on certain ethnoracial groups, notably Native Americans. There is an urgent need for research on this and other important groups. Prevalence estimates also depend on the absence of assessment bias; the text indicates when more research is needed to ensure the accuracy of available data. Users are encouraged to read the sections on culture-related diagnostic issues to contextualize the sections on prevalence.

Sex and Gender Differences

Sex and gender differences as they relate to the causes and expression of medical conditions are established for a number of diseases, including a growing number of mental disorders. *Sex* refers to factors attributable to an individual’s reproductive organs and XX or XY chromosomal complement. *Gender* is a result of reproductive organs as well as an individual’s self-representation and includes the psychological, behavioral, and social consequences of the individual’s perceived gender. Much of the information on the expression of psychiatric illness in women and men is based on self-identified gender, and thus we commonly use *gender differences* or “women and men” or “boys and girls” in DSM-5-TR. However, if information is available and pertinent to “sex”—for example, sex differences in metabolism of substances, or life stages restricted to only one sex, such as

pregnancy or menopause—we use the term *sex differences* or "male and female."

Sex and gender can influence illness in a variety of ways. First, sex may exclusively determine whether an individual is at risk for a disorder (e.g., as in premenstrual dysphoric disorder). Second, sex or gender may moderate the overall risk for development of a disorder as shown by marked differences in the prevalence and incidence rates for selected mental disorders in men and women. Third, sex or gender may influence the likelihood that particular symptoms of a disorder are experienced by an individual. For example, ADHD may manifest differently in boys and girls. Sex or gender may also have other effects on the experience of a disorder that are indirectly relevant to psychiatric diagnosis. For example, certain symptoms may be more readily endorsed by men or women, and this endorsement contributes to differences in service provision (e.g., women may be more likely to recognize a depressive, bipolar, or anxiety disorder and endorse a more comprehensive list of symptoms than do men).

Reproductive life cycle events, including variations in ovarian hormones during the menstrual cycle, pregnancy, or menopause, may contribute to sex differences in risk and expression of illness. Thus, the specifier "with peripartum onset" that can apply to brief psychotic disorder or to a manic, hypomanic, or major depressive episode denotes a time frame wherein women may be at increased risk for the onset of an illness episode. In the case of sleep and energy, postpartum alterations are often normative and thus may have lower diagnostic reliability in postpartum women.

The manual is configured to include information on sex and gender at multiple levels. If there are gender-specific symptoms, they have been added to the diagnostic criteria. A sex-related specifier, such as "with peripartum onset" of a mood episode, provides additional information on sex and diagnosis. Prevalence estimates based on sex and gender are included in the "Prevalence" section of each disorder text. Finally, other issues that are pertinent to diagnosis and influenced by sex and/or gender considerations can be found in the section "Sex- and Gender-Related Diagnostic Issues" in the text for relevant disorders.

Association With Suicidal Thoughts or Behavior

DSM-5-TR contains a new text section for each diagnosis, “Association With Suicidal Thoughts or Behavior,” when such information is available in the literature. The information included is generally based on studies demonstrating associations of suicidal thoughts

or behavior with a given diagnosis. Within groups of individuals with the same diagnosis, a wide range of relevant psychopathology could have an impact on suicide risk, ranging from none to severe. Therefore, in the assessment of a specific individual’s suicide risk, clinicians should use clinical judgment informed by known risk factors and not rely solely on the presence of a diagnosis that has been associated with suicidal thoughts or behavior. The information in these sections should serve as an alert to clinicians that further inquiry may be indicated for an individual with a particular diagnosis. Clinical risk assessment requires an individualized assessment encompassing many factors and goes well beyond the formulation of a DSM-5 diagnosis and the scope of this manual.

Additional Resources and Future Directions

Conditions for Further Study

Described in Section III, “Conditions for Further Study,” are those proposed conditions for which sufficient scientific evidence is not yet available to support widespread clinical use. These proposed criteria and supporting text are included to highlight conditions that could benefit from further research.

Assessment and Monitoring Tools: Now and Looking to the Future

The various components of DSM-5 are provided to facilitate clinical assessment and to aid in developing a comprehensive case formulation (see "Use of the Manual") and identification of characteristics that can influence the prognosis of any diagnosed mental disorders. Whereas the diagnostic criteria in Section II are well established and have undergone extensive review, the assessment tools, Cultural Formulation Interview, and conditions for further study included in Section III are those for which additional scientific evidence may be needed to support widespread clinical use. These diagnostic aids and proposed criteria are included to highlight the evolution and direction of scientific advances in these areas and to stimulate further research.

Each of the measures in Section III, "Assessment Measures," is provided to aid in a comprehensive assessment of individuals that will contribute to a diagnosis and treatment plan tailored to the individual presentation and clinical context. Cross-cutting symptom and diagnosis-specific severity measures provide quantitative ratings of important clinical areas that are designed to be used at the initial evaluation to establish a baseline for comparison with ratings on subsequent encounters to monitor changes and inform treatment planning. Where cultural dynamics are particularly important for diagnostic assessment, the Cultural Formulation Interview (located in the Section III chapter "Culture and Psychiatric Diagnosis") should be considered as a useful aid to communicate with the individual. All of these measures are available online at: www.psychiatry.org/dsm5.

The organizational structure of DSM-5, its use of dimensional measures, and its compatibility with ICD codes will allow it to be readily adaptable to future scientific discoveries and refinements that enhance its clinical utility.

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Use of the Manual

This text is designed to provide a practical guide to using DSM-5, particularly in clinical practice.

Approach to Clinical Case Formulation

The primary purpose of DSM-5 is to assist trained clinicians in the diagnosis of mental disorders as part of a case formulation assessment that leads to an informed treatment plan for each individual. The case formulation for any given individual should involve a careful clinical history and concise summary of the social, psychological, and biological factors that may have contributed to developing a given mental disorder. It is not sufficient to simply check off the symptoms in the diagnostic criteria to make a mental disorder diagnosis. A thorough evaluation of these criteria may assure more reliable assessment (which may be aided by the use of dimensional symptom severity assessment tools); the relative severity and salience of an individual's signs and symptoms and their contribution to a diagnosis will ultimately require clinical judgment. Diagnosis requires clinical training to recognize when the combination of predisposing, precipitating, perpetuating, and protective factors has resulted in a psychopathological condition in which the signs and symptoms exceed normal ranges. The ultimate goal of a clinical case formulation is to use the available contextual and diagnostic information in developing a comprehensive treatment plan that is informed by the individual's cultural and social context. However, recommendations for the selection and use of the most appropriate evidence-based treatment options for each disorder are beyond the scope of this manual.

Elements of a Diagnosis

Diagnostic criteria are offered as guidelines for making diagnoses, and their use should be informed by clinical judgment. Text descriptions, including introductory sections of each diagnostic chapter, can help support diagnosis (e.g., describing the criteria more fully under “Diagnostic Features”; providing differential diagnoses).

Following the assessment of diagnostic criteria, clinicians should consider the application of disorder subtypes and/or specifiers as appropriate. Most specifiers are only applicable to the current presentation and may change over the course of the disorder (e.g., with good to fair insight; predominantly inattentive presentation; in a controlled environment) and can be given only if full criteria for the disorder are currently met. Other specifiers are indicative of the lifetime course (e.g., with seasonal pattern, bipolar type in schizoaffective disorder) and can be assigned regardless of current status.

When the symptom presentation does not meet full criteria for any disorder and the symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning, the “other specified” or “unspecified” category corresponding to the predominant symptoms should be considered.

Subtypes and Specifiers

Subtypes and specifiers are provided for increased diagnostic specificity. *Subtypes* define mutually exclusive and jointly exhaustive phenomenological subgroupings within a diagnosis and are indicated by the instruction “*Specify* whether” in the criteria set (e.g., in anorexia nervosa, *Specify* whether restricting type or binge-eating/purging type). In contrast, *specifiers* are not intended to be mutually exclusive or jointly exhaustive, and as a consequence, more than one specifier may be applied to a given diagnosis. Specifiers (as opposed to subtypes) are indicated by the instruction “*Specify*” or “*Specify* if” in the criteria set (e.g., in social anxiety disorder, “*Specify* if: performance only”). Specifiers and subtypes provide an opportunity to define a more homogeneous subgrouping of individuals

with the disorder who share certain features (e.g., major depressive disorder, with mixed features) and to convey information that is relevant to the management of the individual's disorder, such as the "with other medical comorbidity" specifier in sleep-wake disorders. Although the fifth character within an ICD-10-CM code is sometimes designated to indicate a particular subtype or specifier (e.g., "0" in the fifth character in the F02.80 diagnostic code for major neurocognitive disorder due to Alzheimer's disease, to indicate the absence of a behavioral disturbance versus a "1" in the fifth character of the F02.81 diagnostic code for major neurocognitive disorder due to Alzheimer's disease to indicate the presence of a behavioral disturbance), the majority of subtypes and specifiers included in DSM-5-TR are not reflected in the ICD-10-CM code and are indicated instead by recording the subtype or specifier after the name of the disorder (e.g., social anxiety disorder, performance type).

Use of Other Specified and Unspecified Mental Disorders

Although decades of scientific effort have gone into developing the diagnostic criteria sets for the disorders included in Section II, it is well recognized that this set of categorical diagnoses does not fully describe the full range of mental disorders that individuals experience and present to clinicians on a daily basis throughout the world. Hence, it is also necessary to include "other specified" or "unspecified" disorder options for presentations that do not fit exactly into the diagnostic boundaries of disorders in each chapter. Moreover, there are settings (e.g., emergency department) where it may only be possible to identify the most prominent symptom expressions associated with a particular chapter (e.g., delusions, hallucinations, mania, depression, anxiety, substance intoxication, neurocognitive symptoms). In such cases, it may be most appropriate to assign the corresponding "unspecified" disorder as a placeholder until a more complete differential diagnosis is possible.

DSM-5 provides two diagnostic options for presentations that do not meet the diagnostic criteria for any of the specific DSM-5 disorders: *other specified disorder* and *unspecified disorder*. The other specified category is provided to allow the clinician to communicate the specific reason that the presentation does not meet the criteria for any specific category within a

diagnostic class. This is done by recording the name of the category, followed by the specific reason. For example, with an individual with persistent hallucinations occurring in the absence of any other psychotic symptoms (a presentation that does not meet criteria for any of the specific disorders in the chapter “Schizophrenia Spectrum and Other Psychotic Disorders”), the clinician would record “other specified schizophrenia spectrum and other psychotic disorder, with persistent auditory hallucinations.” If the clinician chooses not to specify the reason that the criteria are not met for a specific disorder, then “unspecified schizophrenia spectrum and other psychotic disorder” would be diagnosed. Note that the differentiation between other specified and unspecified disorders is based on the clinician’s choice to indicate or not the reasons why the presentation does not meet full criteria, providing maximum flexibility for diagnosis. When the clinician determines that there is enough available clinical information to specify the nature of the

presentation, the “other specified” diagnosis can be given. In those cases where the clinician is not able to further specify the clinical presentation (e.g., in emergency room settings), the “unspecified” diagnosis can be given. This is entirely a matter of clinical judgment.

It is a long-standing DSM convention for conditions included in the “Conditions for Further Study” chapter in Section III to be listed as examples of presentations that can be specified using the “other specified” designation. The inclusion of these conditions for further study as examples does not represent endorsement by the American Psychiatric Association that these are valid diagnostic categories.

Use of Clinical Judgment

DSM-5 is a classification of mental disorders that was developed for use in clinical, educational, and research settings. The diagnostic categories, criteria, and textual descriptions are meant to be employed by individuals with appropriate clinical training and experience in diagnosis. It is important that DSM-5 not be applied mechanically by individuals without

clinical training. The specific diagnostic criteria included in DSM-5 are meant to serve as guidelines to be informed by clinical judgment and are not meant to be used in a rigid cookbook fashion. For example, the exercise of clinical judgment may justify giving a certain diagnosis to an individual even though the clinical presentation falls just short of meeting the full criteria for the diagnosis as long as the symptoms that are present are persistent and severe. On the other hand, lack of familiarity with DSM-5 or excessively flexible and idiosyncratic application of DSM-5 criteria substantially reduces its utility as a common language for communication.

Clinical Significance Criterion

In the absence of clear biological markers or clinically useful measurements of severity for many mental disorders, it has not been possible to completely separate normal from pathological symptom expressions contained in diagnostic criteria. This gap in information is particularly problematic in clinical situations in which the individual's symptom presentation by itself (particularly in mild forms) is not inherently pathological and may be encountered in those for whom a diagnosis of "mental disorder" would be inappropriate. Therefore, a generic diagnostic criterion requiring distress or disability has been used to establish disorder thresholds, usually worded "the disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning." Assessing whether this criterion is met, especially in terms of role function, is an inherently difficult clinical judgment. The text following the definition of a mental disorder acknowledges that this criterion may be especially helpful in determining an individual's need for treatment. Use of information from the individual as well as from family members and other third parties via interview or self- or informant-reported assessments regarding the individual's performance is often necessary.

Coding and Recording Procedures

The official coding system in use in the United States since October 1, 2015, is the *International Classification of Diseases*, Tenth Revision, Clinical Modification (ICD-10-CM), a version of the World Health Organization's ICD-10 that has been modified for clinical use by the

Centers for Disease Control and Prevention's National Center for Health Statistics (NCHS) and provides the only permissible diagnostic codes for mental disorders for clinical use in the United States. Most DSM-5 disorders have an alphanumeric ICD-10-CM code that appears preceding the name of the disorder (or coded subtype or specifier) in the DSM-5-TR Classification and in the accompanying criteria set for each disorder. For some diagnoses (e.g., neurocognitive disorders, substance/medication-induced disorders), the appropriate code depends on further specification and is listed within the criteria set for the

disorder with a coding note, and in some cases is further clarified in the text section "Recording Procedures." The names of some disorders are followed by alternative terms enclosed in parentheses.

The use of diagnostic codes is fundamental to medical record keeping. Diagnostic coding facilitates data collection and retrieval and compilation of statistical information. Codes also are often required to report diagnostic data to interested third parties, including governmental agencies, private insurers, and the World Health Organization. For example, in the United States, the use of ICD-10-CM codes for disorders in DSM-5-TR has been mandated by the Health Care Financing Administration for purposes of reimbursement under the Medicare system.

Principal Diagnosis/Reason for Visit

The general convention in DSM-5 is to allow multiple diagnoses to be assigned for those presentations that meet criteria for more than one DSM-5 disorder. When more than one diagnosis is given in an inpatient setting, the principal diagnosis is the condition established after study to be chiefly responsible for occasioning the admission of the individual. When more than one diagnosis is given for an individual in an outpatient setting, the reason for visit is the condition that is chiefly responsible for the ambulatory medical services received during the visit. In most cases, the principal diagnosis or the reason for visit is also the main focus of attention or treatment. It is often difficult (and somewhat arbitrary) to determine which diagnosis is the principal diagnosis or the reason for visit. For

example, it may be unclear which diagnosis should be considered "principal" for an individual hospitalized with both schizophrenia and alcohol use disorder, because each condition may have contributed equally to the need for admission and treatment. The principal diagnosis is indicated by listing it first, and the remaining disorders are listed in order of focus of attention and treatment. When the principal diagnosis or reason for visit is a mental disorder due to another medical condition (e.g., major neurocognitive disorder due to Alzheimer's disease, psychotic disorder due to malignant lung neoplasm), ICD coding rules require that the etiological medical condition be listed first. In that case, the principal diagnosis or reason for visit would be the mental disorder due to the medical condition, the second listed diagnosis. For maximum clarity, the disorder listed as the principal diagnosis or the reason for visit can be followed by the qualifying phrase "(principal diagnosis)" or "(reason for visit)."

Provisional Diagnosis

The modifier "provisional" can be used when there is currently insufficient information to indicate that the diagnostic criteria are met, but there is a strong presumption that the information will become available to allow that determination. The clinician can indicate the diagnostic uncertainty by recording "(provisional)" following the diagnosis. For example, this modifier might be used when an individual who appears to have a presentation consistent with a diagnosis of current major depressive disorder is unable to give an adequate history, but it is expected that such information will become available after interviewing an informant or reviewing medical records. Once that information becomes available and confirms that the diagnostic criteria were met, the modifier "(provisional)" would be removed. Another use of "provisional" is for those situations in which differential diagnosis depends exclusively on whether the duration of illness does not exceed an upper limit as required by the diagnostic criteria. For example, a diagnosis of schizophreniform disorder requires a duration of at least 1 month but less than 6 months. If an individual currently has symptoms consistent with a diagnosis of schizophreniform disorder except that the ultimate duration is unknown because the symptoms are still ongoing, the modifier "(provisional)" would be applied and then removed if

the symptoms remit within a period of 6 months. If they do not remit, the diagnosis would be changed to schizophrenia.

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Notes About Terminology

Substance/Medication-Induced Mental Disorder

The term “substance/medication-induced mental disorder” refers to symptomatic presentations that are due to the physiological effects of an exogenous substance on the central nervous system, including symptoms that develop during withdrawal from an exogenous substance that is capable of causing physiological dependence. Such exogenous substances include typical intoxicants (e.g., alcohol, inhalants, hallucinogens, cocaine), psychotropic medications (e.g., stimulants; sedatives, hypnotics, anxiolytics), other medications (e.g., steroids), and environmental toxins (e.g., organophosphate insecticides). Editions of DSM from DSM-III to DSM-IV referred to these as “substance-induced mental disorders.” To emphasize that medications and not just substances of abuse can cause psychiatric symptoms, the term was changed to “substance/medication-induced” in DSM-5.

Independent Mental Disorders

Historically, mental disorders were divided into those that were termed “organic” (caused by physical factors) versus those that were “nonorganic” (purely of the mind; also referred to as “functional” or “psychogenic”), terms that were included in DSM up through DSM-III-R. Because these dichotomies misleadingly implied that the nonorganic disorders have no biological basis and that mental disorders have no physical basis, DSM-IV updated this terminology as follows: 1) the terms “organic” and “nonorganic” were eliminated from DSM-IV; 2) the disorders formerly called “organic” were divided into those due to the direct physiological effects of a substance (substance-induced) and those due to the direct physiological effects of a medical condition on the central nervous system; and 3) the term “nonorganic mental disorders” (i.e., those disorders not due

to either substances or medical conditions) was replaced by "primary mental disorder." In DSM-5, this terminology was further refined, replacing "primary" with "independent" (e.g., Criterion C in substance/medication-induced anxiety disorder starts with "the disturbance is not better accounted for by an anxiety disorder that is not substance-induced. Evidence of an *independent* anxiety disorder could include . . ." *[italics added for reference]*). This was done to reduce the potential for confusion given that the term "primary" has historically had other meanings (e.g., it is sometimes used to indicate which disorder among several comorbid disorders was the first to occur). The use of "independent mental disorder" should not be construed to mean that the disorder is independent of other potential causal factors such as psychosocial or other environmental stressors.

Other Medical Conditions

Another dichotomy adopted by prior editions of DSM that reflected mind-body dualism was the division of disorders into "mental disorders" and "physical disorders." In conjunction with the elimination of organic/nonorganic terminology, DSM-IV replaced the "mental disorder" versus "physical disorder" dichotomy with a "mental disorder" vs. "general medical condition" dichotomy, based on chapter location within the International Classification of Diseases (ICD). Medical conditions in ICD have been divided into 17 chapters based on a variety of factors, which include etiology (e.g., Neoplasms [Chapter 2]), anatomical location (e.g., Diseases of the ear and mastoid process [Chapter 8]), body system (e.g., Diseases of the circulatory system [Chapter 9]), and context (e.g., Pregnancy, childbirth and the puerperium [Chapter 15]). In the ICD framework, mental disorders are those located in Chapter 5, and general medical conditions are those located within the other 16 chapters. Because of concerns that the term "general medical condition" could be conflated with general practice, DSM-5 uses the term "another medical condition" to emphasize the fact that mental disorders are medical conditions and that mental disorders can be precipitated by other medical conditions. It is important to recognize that "mental

disorder" and "another medical condition" are merely terms of convenience and should not be taken to imply that there is any fundamental distinction between mental disorders and other medical conditions, that mental disorders are unrelated to physical or biological factors or processes, or that other medical conditions are unrelated to behavioral or psychosocial factors or processes.

Types of Information in the DSM-5-TR Text

The DSM-5-TR text provides contextual information to aid in diagnostic decision-making. The text appears immediately following the diagnostic criteria for each disorder and systematically describes the disorder under the following headings: Recording Procedures, Subtypes, Specifiers, Diagnostic Features, Associated Features, Prevalence, Development and Course, Risk and Prognostic Factors, Culture-Related Diagnostic Issues, Sex- and Gender-Related Diagnostic Issues, Diagnostic Markers, Association With Suicidal Thoughts or Behavior, Functional Consequences, Differential Diagnosis, and Comorbidity. In general, when limited information is available for a section, that section is not included.

Recording Procedures provides guidelines for reporting the name of the disorder and for selecting and recording the appropriate ICD-10-CM diagnostic code. It also includes instructions for applying any appropriate subtypes and/or specifiers.

Subtypes and/or **Specifiers** provide brief descriptions of applicable subtypes and/or specifiers.

Diagnostic Features provides descriptive text illustrating the use of the criteria and includes key points on their interpretation. For example, within the diagnostic features for schizophrenia, it is explained that some symptoms that may appear to be negative symptoms could instead be attributable to medication side effects.

Associated Features includes clinical features that are not represented in the criteria but occur significantly more often in individuals with the disorder than those without the disorder. For example, individuals with generalized anxiety disorder may also experience somatic symptoms that are not contained within the disorder criteria.

Prevalence describes rates of the disorder in the community, most often described as 12-month prevalence, although for some disorders point prevalence is noted. Prevalence estimates are also provided by age group and by ethnoracial/cultural group when possible. Sex ratio (prevalence in men vs. women) is also provided in this section. When international data are available, geographic variance in prevalence rates is described. For some disorders, especially those for which there are limited data on rates in the community, prevalence in relevant clinical samples is noted.

Development and Course describes the typical lifetime patterns of presentation and evolution of the disorder. It notes the typical age at onset and whether the presentation may have prodromal/insidious features or may manifest abruptly. Other descriptions may include an episodic versus persistent course as well as a single episode versus a recurrent episodic course. Descriptors in this section may address duration of symptoms or episodes as well as progression of severity and associated functional impact. The general trend of the disorder over time (e.g., stable, worsening, improving) is described here. Variations that may be noted include features related to developmental stage (e.g., infancy, childhood, adolescence, adulthood, late life).

Risk and Prognostic Factors includes a discussion of factors thought to contribute to the development of a disorder. It is divided into subsections addressing *temperamental factors* (e.g., personality features); *environmental factors* (e.g., head trauma, emotional trauma, exposure to toxic substances, substance use); and *genetic and physiological factors*

(e.g., *APOE4* for dementia, other known familial genetic risks); this subsection may address familial patterns (traditional) as well as genetic and epigenetic factors. An additional subsection for *course modifiers* includes factors that may incur a deleterious course, and conversely factors that may have ameliorative or protective effects.

Culture-Related Diagnostic Issues includes information on variations in symptom expression, attributions for disorder causes or precipitants, factors associated with differential prevalence across demographic groups, cultural norms that may affect level of perceived pathology, risk of misdiagnosis when evaluating individuals from socially oppressed ethnoracial groups, and other material relevant to culturally informed diagnosis. Prevalence rates in specific cultural/ethnic groups are located in the Prevalence section.

Sex- and Gender-Related Diagnostic Issues includes correlates of the diagnosis that are related to sex or gender, predominance of symptoms or the diagnosis by sex or gender, and any other sex- and gender-related diagnostic implications of the diagnosis, such as differences in the clinical course by sex or gender. Prevalence rates by gender are located in the Prevalence section.

Diagnostic Markers addresses objective measures that have established diagnostic value. These may include physical examination findings (e.g., signs of malnutrition in avoidant/restrictive food intake disorder), laboratory findings (e.g., low CSF hypocretin-1 levels in narcolepsy), or imaging findings (e.g., regionally hypometabolic FDG PET imaging for neurocognitive disorder due to Alzheimer's disease).

Association With Suicidal Thoughts or Behavior provides information about disorder-specific prevalence of suicidal thoughts or behavior, as well as risk factors for suicide that may be associated with the disorder.

Functional Consequences discusses notable functional consequences associated with a disorder that are likely to have an impact on the daily lives of affected individuals; these consequences may affect the ability to engage in tasks related to education, work, and maintaining independent living. These may vary according to age and across the life span.

Differential Diagnosis discusses how to differentiate the disorder from other disorders that have some similar presenting characteristics.

Comorbidity includes descriptions of mental disorders and other medical conditions (i.e., conditions classified outside of the Mental and

Behavioral disorders chapter in ICD-10-CM), likely to co-occur with the diagnosis.

Other Conditions and Disorders in Section II

In addition to providing diagnostic criteria and text for DSM-5 mental disorders, Section II also includes two chapters for other conditions that are not mental disorders but may be encountered by mental health clinicians. These conditions may be listed as a reason for a clinical visit in addition to, or in place of, the mental disorders in Section II. The chapter **"Medication-Induced Disorders and Other Adverse Effects of Medication"** includes medication-induced parkinsonism, neuroleptic malignant syndrome, medication-induced acute dystonia, medication-induced acute akathisia, tardive dyskinesia, tardive dystonia/tardive akathisia, medication-induced postural tremor, antidepressant discontinuation syndrome, and other adverse effect of medication. These conditions are included in Section II because of their frequent importance in 1) the management by medication of mental disorders or other medical conditions, and 2) the differential diagnosis with mental disorders (e.g., anxiety disorder vs. medication-induced acute akathisia).

The chapter **"Other Conditions That May Be a Focus of Clinical Attention"** includes conditions and psychosocial or environmental problems that are not considered to be mental disorders but otherwise affect the diagnosis, course, prognosis, or treatment of an individual's mental disorder. These conditions are presented with their corresponding codes from ICD-10-CM (usually Z codes). A condition or problem in this chapter may be coded with or without an accompanying mental disorder diagnosis 1) if it is a reason for the current visit; 2) if it helps to explain the need for a test, procedure, or treatment; 3) if it plays a role in the initiation or exacerbation of a mental disorder; or 4) if it constitutes a problem that should be considered in the overall management plan. These include suicidal behavior and nonsuicidal self-injury; abuse and neglect; relational problems (e.g., Relationship Distress With Spouse or Intimate Partner);

educational, occupational, housing, and economic problems; problems related to the social environment, interaction with the legal system, and other psychosocial, personal, and environmental circumstances (e.g., problems related to unwanted pregnancy, being a victim of crime or terrorism); problems related to access to medical and other health care; circumstances of personal history (e.g., Personal History of Psychological Trauma); other health service encounters for counseling and medical advice (e.g., sex counseling); and additional conditions or problems that may be a focus of clinical attention (e.g., wandering associated with a mental disorder, uncomplicated bereavement, phase of life problem).

Online Enhancements

DSM-5-TR is available in online subscriptions at PsychiatryOnline.org, as well as an e-book that reflects the print edition. The online version provides a complete set of supporting in-text citations and references not available in print or e-book; it is also updated periodically to reflect any changes resulting from the DSM-5 iterative revision process, described in the Introduction. DSM-5 will be retained online in an archived format at PsychiatryOnline.org, joining prior versions of DSM.

Clinical rating scales and measures in the print edition and e-book (see "Assessment Measures" in Section III) are included online along with additional assessment measures used in the field trials (www.psychiatry.org/dsm5), linked to the relevant disorders. From the Section III chapter "Culture and Psychiatric Diagnosis," the Cultural Formulation Interview, Cultural Formulation Interview—Informant Version (both included in print and e-book), and supplementary modules to the core Cultural Formulation Interview are all available online at www.psychiatry.org/dsm5.

Cautionary Statement for Forensic Use of DSM-5

Although the DSM-5 diagnostic criteria and text are primarily designed to assist clinicians in conducting clinical assessment, case formulation, and treatment planning, DSM-5 is also used as a reference for the courts and attorneys in assessing the legal consequences of mental disorders. As a result, it is important to note that the definition of mental disorder included in DSM-5 was developed to meet the needs of clinicians, public health professionals, and research investigators rather than the technical needs of the courts and legal professionals. It is also important to note that DSM-5 does not provide treatment guidelines for any given disorder.

When used appropriately, diagnoses and diagnostic information can assist legal decision makers in their determinations. For example, when the presence of a mental disorder is the predicate for a subsequent legal determination (e.g., involuntary civil commitment), the use of an established system of diagnosis enhances the value and reliability of the determination. By providing a compendium based on a review of the pertinent clinical and research literature, DSM-5 may facilitate legal decision-makers' understanding of the relevant characteristics of mental disorders. The literature related to diagnoses also serves as a check on ungrounded speculation about mental disorders and about the functioning of a particular individual. Finally, diagnostic information about longitudinal course may improve decision-making when the legal issue concerns an individual's mental functioning at a past or future point in time.

However, the use of DSM-5 in forensic settings should be informed by an awareness of the risks and limitations of its use. When DSM-5 categories, criteria, and textual descriptions are employed for forensic purposes, there is a risk that diagnostic information will be misused or

misunderstood. These dangers arise because of the imperfect fit between the questions of ultimate concern to the law and the information contained in a clinical diagnosis. In most situations, the clinical diagnosis of a DSM-5 mental disorder such as intellectual developmental disorder (intellectual disability), schizophrenia, major neurocognitive disorder, gambling disorder, or pedophilic disorder does not imply that an individual with such a condition meets legal criteria for the presence of a mental disorder or "mental illness" as defined in law, or a specified legal standard (e.g., for competence, criminal responsibility, or disability). For the latter, additional information is usually required beyond that contained in the DSM-5 diagnosis, which might include information about the individual's functional impairments and how these impairments affect the particular abilities in question. It is precisely because impairments, abilities, and disabilities vary widely within each diagnostic category that assignment of a particular diagnosis does not imply a specific level of risk, impairment, or disability.

Use of DSM-5 to assess the presence of a mental disorder by nonclinical, nonmedical, or otherwise insufficiently trained individuals is not advised. Nonclinical decision-makers should also be cautioned that a diagnosis does not carry any necessary implications regarding the etiology or causes of the individual's mental disorder or the individual's degree of control over behaviors that may be associated with the disorder. Even when diminished control over the individual's own behavior is a feature of the disorder, having the diagnosis in itself does not demonstrate that a particular individual is (or was) unable to control his or her behavior at a particular time.

SECTION II

Diagnostic Criteria and Codes

Neurodevelopmental Disorders

Schizophrenia Spectrum and Other Psychotic Disorders

Bipolar and Related Disorders

Depressive Disorders

Anxiety Disorders

Obsessive-Compulsive and Related Disorders

Trauma- and Stressor-Related Disorders

Dissociative Disorders

Somatic Symptom and Related Disorders

Feeding and Eating Disorders

Elimination Disorders

Sleep-Wake Disorders

Sexual Dysfunctions

Gender Dysphoria

Disruptive, Impulse-Control, and Conduct Disorders

Substance-Related and Addictive Disorders

Neurocognitive Disorders

Personality Disorders

Paraphilic Disorders

Other Mental Disorders and Additional Codes

Medication-Induced Movement Disorders and Other Adverse Effects of Medication

Other Conditions That May Be a Focus of Clinical Attention

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This section contains the diagnostic criteria approved for routine clinical use along with ICD-10-CM diagnostic codes. For each mental disorder, the diagnostic criteria are followed by descriptive text to assist in diagnostic decision-making. Where needed, notes to facilitate the selection of the appropriate ICD-10-CM code as well as recording procedures are also provided.

Section II also includes two chapters of other conditions that are not mental disorders but may be encountered by clinicians. “Medication-Induced Movement Disorders and Other Adverse Effects of Medication” features conditions of frequent importance in the management by medication of mental disorders or other medical conditions and the differential diagnosis with mental disorders (e.g., an anxiety disorder vs. medication-induced acute akathisia). “Other Conditions That May Be a Focus of Clinical Attention” includes conditions and psychosocial or environmental problems that are not considered to be mental disorders but otherwise affect the diagnosis, course, prognosis, or treatment of an individual’s mental disorder.

These three components—the criteria and their descriptive text, the medication-induced movement disorders and other adverse effects of medication, and the descriptions of other conditions that may be a focus of clinical attention—represent the key elements of the clinical diagnostic process and thus are presented together.

Neurodevelopmental Disorders

The neurodevelopmental disorders are a group of conditions with onset in the developmental period. The disorders typically manifest early in development, often before the child enters school, and are characterized by developmental deficits or differences in brain processes that produce impairments of personal, social, academic, or occupational functioning. The range of developmental deficits or differences varies from very specific limitations of learning or control of executive functions to global impairments of social skills or intellectual ability. Once thought to be categorically defined, more recent dimensional approaches to measurement of the symptoms demonstrate a range of severity, often without a very clear boundary with typical development. Diagnosis of a disorder thus requires the presence of both symptoms and impaired function.

The neurodevelopmental disorders frequently co-occur with one another; for example, individuals with autism spectrum disorder often have intellectual developmental disorder (intellectual disability), and many children with attention-deficit/hyperactivity disorder (ADHD) also have a specific learning disorder. The neurodevelopmental disorders also frequently co-occur with other mental and behavioral disorders with onset in childhood (e.g., communication disorders and autism spectrum disorder may be associated with anxiety disorders; ADHD with oppositional defiant disorder; tics with obsessive-compulsive disorder). For some neurodevelopmental disorders, the clinical presentation includes behaviors that are more frequent or intense when compared with those of normal children of the same developmental age and gender, as well as deficits and delays in achieving expected milestones. For example, autism spectrum disorder is diagnosed only when the characteristic deficits of social communication are accompanied by excessively repetitive behaviors, restricted interests, and insistence on sameness.

Intellectual developmental disorder is characterized by deficits in general mental abilities, such as reasoning, problem solving, planning, abstract thinking, judgment, academic learning, and learning from experience. The deficits result in impairments of adaptive functioning, such that the individual fails to meet standards of personal independence and social responsibility in one or more aspects of daily life, including communication, social participation, academic or occupational functioning, and personal independence at home or in community settings. Global developmental delay, as its name implies, is diagnosed when an individual fails to meet expected developmental milestones in several areas of intellectual functioning. The diagnosis is used for individuals younger than 5 years who are unable to undergo systematic assessments of intellectual functioning, and thus the clinical severity level cannot be reliably assessed. Intellectual developmental disorder may result from an acquired insult during the developmental period from, for example, a severe head injury, in which case a neurocognitive disorder also may be diagnosed.

The communication disorders include language disorder, speech sound disorder, social (pragmatic) communication disorder, and childhood-onset fluency disorder (stuttering). The first three disorders are characterized by deficits in the development and use of language, speech, and social communication, respectively. Social communication disorder is characterized by deficits in both verbal and nonverbal communication skills that result in

social impairment and are not better explained by low abilities in structural language, intellectual developmental disorder, or autism spectrum disorder. Childhood-onset fluency disorder is characterized by disturbances of the normal fluency and motor production of speech, including repetitive sounds or syllables, prolongation of consonants or vowel sounds, broken words, blocking, or words produced with an excess of physical tension. Like other neurodevelopmental disorders, communication disorders begin early in life and may produce lifelong functional impairments.

Autism spectrum disorder is characterized by persistent deficits in social communication and social interaction across multiple contexts, including

deficits in social reciprocity, nonverbal communicative behaviors used for social interaction, and skills in developing, maintaining, and understanding relationships. In addition to the social communication deficits, the diagnosis of autism spectrum disorder requires the presence of restricted, repetitive patterns of behavior, interests, or activities. Because symptoms change with development and may be masked by compensatory mechanisms, the diagnostic criteria may be met based on historical information, although the current presentation must cause significant impairment.

Within the diagnosis of autism spectrum disorder, individual clinical characteristics are noted through the use of specifiers (with or without accompanying intellectual impairment; with or without accompanying structural language impairment; associated with a known genetic or other medical condition or environmental factor; associated with a neurodevelopmental, mental, or behavioral problem), as well as specifiers that describe the severity of autistic symptoms. These specifiers provide clinicians with an opportunity to individualize the diagnosis and communicate a richer clinical description of the affected individuals. For example, many individuals previously diagnosed with Asperger's disorder would now receive a diagnosis of autism spectrum disorder without language or intellectual impairment.

ADHD is a neurodevelopmental disorder defined by impairing levels of inattention, disorganization, and/or hyperactivity-impulsivity. Inattention and disorganization entail inability to stay on task, seeming not to listen, and losing materials necessary for tasks, at levels that are inconsistent with age or developmental level. Hyperactivity-impulsivity entails overactivity, fidgeting, inability to stay seated, intruding into other people's activities, and inability to wait—symptoms that are excessive for age or developmental level. In childhood, ADHD frequently overlaps with disorders that are often considered to be "externalizing disorders," such as oppositional defiant disorder and conduct disorder. ADHD often persists into adulthood, with resultant impairments of social, academic, and occupational functioning.

Specific learning disorder, as the name implies, is diagnosed when there are specific deficits in an individual's ability to perceive or process information for learning academic skills efficiently and accurately. This neurodevelopmental disorder first manifests during the years of formal

schooling and is characterized by persistent and impairing difficulties with learning foundational academic skills in reading, writing, and/or math. The individual's performance of the affected academic skills is well below average for age, or acceptable performance levels are achieved only with extraordinary effort. Specific learning disorder may occur in individuals identified as intellectually gifted and manifest only when the learning demands or assessment procedures (e.g., timed tests) pose barriers that cannot be overcome by their innate intelligence and compensatory strategies. For all individuals, specific learning disorder can produce lifelong impairments in activities dependent on the skills, including occupational performance.

The neurodevelopmental motor disorders include developmental coordination disorder, stereotypic movement disorder, and tic disorders. Developmental coordination disorder is characterized by deficits in the acquisition and execution of coordinated motor skills and is manifested by clumsiness and slowness or inaccuracy of performance of

motor skills that cause interference with activities of daily living. Stereotypic movement disorder is diagnosed when an individual has repetitive, seemingly driven, and apparently purposeless motor behaviors, such as hand flapping, body rocking, head banging, self-biting, or hitting. The movements interfere with social, academic, or other activities. If the behaviors cause self-injury, this should be specified as part of the diagnostic description. Tic disorders are characterized by the presence of motor or vocal tics, which are sudden, rapid, recurrent, nonrhythmic, stereotyped motor movements or vocalizations. The duration, presumed etiology, and clinical presentation define the specific tic disorder that is diagnosed: Tourette's disorder, persistent (chronic) motor or vocal tic disorder, provisional tic disorder, other specified tic disorder, and unspecified tic disorder. Tourette's disorder is diagnosed when the individual has multiple motor and vocal tics that have been present for at least 1 year and that have a waxing-waning symptom course.

The use of specifiers for the neurodevelopmental disorder diagnoses enriches the clinical description of the individual's clinical course and

current symptomatology. These include the following: Severity specifiers are available for intellectual developmental disorder, autism spectrum disorder, ADHD, specific learning disorder, and stereotypic movement disorder. Specifiers indicative of current symptomatology are available for ADHD, specific learning disorder, and persistent motor or vocal tic disorder. Autism spectrum disorder and stereotypic movement disorder also include the specifier "associated with a known genetic or other medical condition or environmental factor." This specifier gives clinicians an opportunity to document factors that may have played a role in the etiology of the disorder, as well as those that might affect the clinical course.

Intellectual Developmental Disorders

Intellectual Developmental Disorder (Intellectual Disability)

Diagnostic Criteria

Intellectual developmental disorder (intellectual disability) is a disorder with onset during the developmental period that includes both intellectual and adaptive functioning deficits in conceptual, social, and practical domains. The following three criteria must be met:

A. Deficits in intellectual functions, such as reasoning, problem solving, planning, abstract thinking, judgment, academic learning, and learning from experience, confirmed by both clinical assessment and individualized, standardized intelligence testing.
B. Deficits in adaptive functioning that result in failure to meet developmental and sociocultural standards for personal independence and social responsibility. Without ongoing

support, the adaptive deficits limit functioning in one or more activities of daily life, such as communication, social participation, and independent living, across multiple environments, such as home, school, work, and community.

C. Onset of intellectual and adaptive deficits during the developmental period.

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Note: The term *intellectual developmental disorder* is used to clarify its relationship with the WHO ICD-11 classification system, which uses the term *Disorders of Intellectual Development*. The equivalent term *intellectual disability* is placed in parentheses for continued use. The medical and research literature use both terms, while intellectual disability is the term in common use by educational and other professions, advocacy groups, and the lay public. In the United States, Public Law 111-256 (Rosa's Law) changed all references to "mental retardation" in federal laws to "intellectual disability."

Specify current severity (see Table 1):

F70 Mild

F71 Moderate

F72 Severe

F73 Profound

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TABLE 1 Severity levels for intellectual developmental disorder (intellectual disability)

Severity level	Conceptual domain	Social domain	Practical domain

Severity level	Conceptual domain	Social domain	Practical domain
Mild	For preschool children, there may be no obvious conceptual differences. For school-age children and adults, there are difficulties in learning academic skills involving reading, writing, arithmetic, time, or money, with support needed in one or more areas to meet age-related expectations. In adults, abstract thinking, executive function (i.e., planning, strategizing, priority setting, and cognitive flexibility), and short-term memory, as well as functional use of academic skills (e.g., reading, money management), are impaired. There is a somewhat concrete approach to problems and solutions compared with age-mates.	Compared with typically developing age-mates, the individual is immature in social interactions. For example, there may be difficulty in accurately perceiving peers' social cues. Communication, conversation, and language are more concrete or immature than expected for age. There may be difficulties regulating emotion and behavior in age-appropriate fashion; these difficulties are noticed by peers in social situations. There is limited understanding of risk in social situations; social judgment is immature for age, and the person is at risk of being manipulated by others (gullibility).	The individual may function age-appropriately in personal care. Individuals need some support with complex daily living tasks in comparison to peers. In adulthood, supports typically involve grocery shopping, transportation, home and child-care organizing, nutritious food preparation, and banking and money management. Recreational skills resemble those of age-mates, although judgment related to well-being and organization around recreation requires support. In adulthood, competitive employment is often seen in jobs that do not emphasize conceptual skills. Individuals generally need support to make health care decisions and legal decisions, and to learn to perform a skilled vocation competently. Support is typically needed to raise a family.

Severity level	Conceptual domain	Social domain	Practical domain
40 Moderate	All through development, the individual's conceptual skills lag markedly behind those of peers. For preschoolers, language and preacademic skills develop slowly. For school-age children, progress in reading, writing, mathematics, and understanding of time and money occurs slowly across the school years and is markedly limited compared with that of peers. For adults, academic skill development is typically at an elementary level, and support is required for all use of academic skills in work and personal life. Ongoing assistance on a daily basis is needed to complete conceptual tasks of day-to-day life, and others may take over these responsibilities fully for the individual.	The individual shows marked differences from peers in social and communicative behavior across development. Spoken language is typically a primary tool for social communication but is much less complex than that of peers. Capacity for relationships is evident in ties to family and friends, and the individual may have successful friendships across life and sometimes romantic relations in adulthood. However, individuals may not perceive or interpret social cues accurately. Social judgment and decision-making abilities are limited, and caretakers must assist the person with life decisions. Friendships with typically developing peers are often affected by communication or social limitations. Significant social and communicative support is needed in work settings for success.	The individual can care for personal needs involving eating, dressing, elimination, and hygiene as an adult, although an extended period of teaching and time is needed for the individual to become independent in these areas, and reminders may be needed. Similarly, participation in all household tasks can be achieved by adulthood, although an extended period of teaching is needed, and ongoing supports will typically occur for adult-level performance. Independent employment in jobs that require limited conceptual and communication skills can be achieved, but considerable support from coworkers, supervisors, and others is needed to manage social expectations, job complexities, and ancillary responsibilities such as scheduling, transportation, health benefits, and money management. A variety of recreational skills can be developed. These typically require additional supports and learning opportunities over an extended period of time. Maladaptive behavior is present in a significant minority and causes social problems.

Severity level	Conceptual domain	Social domain	Practical domain
41 Severe	Attainment of conceptual skills is limited. The individual generally has little understanding of written language or of concepts involving numbers, quantity, time, and money. Caretakers provide extensive supports for problem solving throughout life.	Spoken language is quite limited in terms of vocabulary and grammar. Speech may be single words or phrases and may be supplemented through augmentative means. Speech and communication are focused on the here and now within everyday events. Language is used for social communication more than for explication. Individuals understand simple speech and gestural communication. Relationships with family members and familiar others are a source of pleasure and help.	The individual requires support for all activities of daily living, including meals, dressing, bathing, and elimination. The individual requires supervision at all times. The individual cannot make responsible decisions regarding well-being of self or others. In adulthood, participation in tasks at home, recreation, and work requires ongoing support and assistance. Skill acquisition in all domains involves long-term teaching and ongoing support. Maladaptive behavior, including self-injury, is present in a significant minority.

Severity level	Conceptual domain	Social domain	Practical domain
Profound	Conceptual skills generally involve the physical world rather than symbolic processes. The individual may use objects in goal-directed fashion for self-care, work, and recreation. Certain visuospatial skills, such as matching and sorting based on physical characteristics, may be acquired. However, co-occurring motor and sensory impairments may prevent functional use of objects.	The individual has very limited understanding of symbolic communication in speech or gesture. He or she may understand some simple instructions or gestures. The individual expresses his or her own desires and emotions largely through nonverbal, nonsymbolic communication. The individual enjoys relationships with well-known family members, caretakers, and familiar others, and initiates and responds to social interactions through gestural and emotional cues. Co-occurring sensory and physical impairments may prevent many social activities.	The individual is dependent on others for all aspects of daily physical care, health, and safety, although he or she may be able to participate in some of these activities as well. Individuals without severe physical impairments may assist with some daily work tasks at home, like carrying dishes to the table. Simple actions with objects may be the basis of participation in some vocational activities with high levels of ongoing support. Recreational activities may involve, for example, enjoyment in listening to music, watching movies, going out for walks, or participating in water activities, all with the support of others. Co-occurring physical and sensory impairments are frequent barriers to participation (beyond watching) in home, recreational, and vocational activities. Maladaptive behavior is present in a significant minority.

Specifiers

The various levels of severity are defined on the basis of adaptive functioning, and not IQ scores, because it is adaptive functioning that

determines the level of supports required. Moreover, IQ measures are less valid in the lower end of the IQ range.

Diagnostic Features

The essential features of intellectual developmental disorder (intellectual disability) are deficits in general mental abilities (Criterion A) and impairment in everyday adaptive functioning, in comparison to an individual's age-, gender-, and socioculturally matched peers (Criterion B). Onset is during the developmental period (Criterion C). The diagnosis of intellectual developmental disorder is based on both clinical assessment and standardized testing of intellectual functions, standardized neuropsychological tests, and standardized tests of adaptive functioning.

Criterion A refers to intellectual functions that involve reasoning, problem solving, planning, abstract thinking, judgment, learning from instruction and experience, and practical understanding. Critical components include verbal comprehension, working memory, perceptual reasoning, quantitative reasoning, abstract thought, and cognitive efficacy. Intellectual functioning is typically measured with individually administered and psychometrically valid, comprehensive, and culturally appropriate tests of intelligence. Individuals with intellectual developmental disorder have scores of approximately two standard deviations or more below the population mean, including a margin for measurement error (generally ± 5 points). On tests with a standard deviation of 15 and a mean of 100, this involves a score of 65–75 (70 ± 5). Clinical training and judgment are required to interpret test results and assess intellectual performance.

Factors that may affect test scores include practice effects (i.e., learning from repeated testing) and the "Flynn effect" (i.e., overly high scores due to out-of-date test norms). Invalid scores may result from the use of brief intelligence screening tests or group tests; highly discrepant individual subtest scores may make an overall IQ score invalid. Instruments must be normed for the individual's sociocultural background and native language. Co-occurring disorders that affect communication, language, and/or motor or sensory function may affect test scores. Individual cognitive profiles based on neuropsychological testing as well as cross-battery intellectual

assessment (using multiple IQ or other cognitive tests to create a profile) are more useful for understanding intellectual abilities than a single IQ score.

Such testing may identify areas of relative strengths and weaknesses, an assessment important for academic and vocational planning. IQ test scores are approximations of conceptual functioning but may be insufficient to assess reasoning in real-life situations and mastery of practical tasks. For example, a person with deficits in intellectual functioning whose IQ score is somewhat above 65–75 may nevertheless have such substantial adaptive behavior problems in social judgment or other areas of adaptive functioning that the person's actual functioning is clinically comparable to that of individuals with a lower IQ score. Thus, clinical judgment is important in interpreting the results of IQ tests, and using them as the sole criteria for the diagnosis of an intellectual developmental disorder is insufficient.

Deficits in adaptive functioning (Criterion B) refer to how well a person meets community standards of personal independence and social responsibility, in comparison to others of similar age and sociocultural background. Adaptive functioning involves adaptive reasoning in three domains: conceptual, social, and practical. The *conceptual (academic) domain* involves competence in memory, language, reading, writing, math reasoning, acquisition of practical knowledge, problem solving, and judgment in novel situations, among others. The *social domain* involves awareness of others' thoughts, feelings, and experiences; empathy; interpersonal communication skills; friendship abilities; and social judgment, among others. The *practical domain* involves learning and self-management across life settings, including personal care, job responsibilities, money management, recreation, self-management of behavior, and school and work task organization, among others. Intellectual capacity, education, motivation, socialization, personality features, vocational opportunity, cultural experience, and coexisting other medical conditions or mental disorders influence adaptive functioning.

Adaptive functioning is assessed using both clinical evaluation and individualized, culturally appropriate, psychometrically sound measures.

Standardized measures are used with knowledgeable informants (e.g., parent or other family member; teacher; counselor; care provider) and the individual to the extent possible. Additional sources of information include educational, developmental, medical, and mental health evaluations. Scores from standardized measures and interview sources must be interpreted using clinical judgment. When standardized testing is difficult or impossible, because of a variety of factors (e.g., sensory impairment, severe problem behavior), the individual may be diagnosed with unspecified intellectual developmental disorder. Adaptive functioning may be difficult to assess in a controlled setting (e.g., prisons, detention centers); if possible, corroborative information reflecting functioning outside those settings should be obtained.

Criterion B is met when at least one domain of adaptive functioning—conceptual, social, or practical—is sufficiently impaired that ongoing support is needed in order for the person to perform adequately across multiple environments, such as home, school, work, and community. Criterion C, onset during the developmental period, refers to recognition that intellectual and adaptive deficits are present during childhood or adolescence.

A comprehensive evaluation includes an assessment of intellectual capacity and adaptive functioning; identification of genetic and nongenetic etiologies; evaluation for associated medical conditions (e.g., cerebral palsy, seizure disorder); and evaluation for co-occurring mental, emotional, and behavioral disorders. Components of the evaluation may include basic pre- and perinatal medical history, three-generational family pedigree, physical examination, genetic evaluation (e.g., karyotype or chromosomal microarray analysis and testing for specific genetic syndromes), and metabolic screening and neuroimaging assessment.

Associated Features

Intellectual developmental disorder is a heterogeneous condition with multiple causes. There may be associated difficulties with social judgment; assessment of risk; self-management of behavior, emotions, or interpersonal relationships; or motivation in school or

work environments. Because of a lack of awareness of risk and danger, accidental injury rates may be increased. Lack of communication skills may predispose to disruptive and aggressive behaviors. Gullibility is often a feature, involving naiveté in social situations and a tendency for being easily led by others. Gullibility and lack of awareness of risk may result in exploitation by others and possible victimization, fraud, unintentional criminal involvement, false confessions, and risk for physical and sexual abuse. These associated features can be important in criminal cases, including Atkins-type hearings involving the death penalty.

Beyond deficits in adaptive functioning, individuals also can become distressed about their intellectual limitations. While such distress may not always be seen as having an impact on functioning, distress can represent an important feature of the clinical scenario.

Prevalence

Intellectual developmental disorder has an overall general population prevalence of approximately 10 per 1,000; however, the global prevalence varies by country and level of development, being approximately 16 per 1,000 in middle-income countries and 9 per 1,000 in high-income countries. The prevalence also varies by age, being higher in youth than in adults. In the United States, prevalence per 1,000 population does not vary significantly by ethnoracial groups.

Development and Course

Onset of intellectual developmental disorder is in the developmental period. The age and characteristic features at onset depend on the etiology and severity of brain dysfunction. Delayed motor, language, and social milestones may be identifiable within the first 2 years of life among those with more severe intellectual developmental disorder, while mild levels may not be identifiable until school age when difficulty with academic learning becomes apparent. All criteria (including Criterion C) must be fulfilled by history or current presentation. Some children younger than 5 years whose presentation will eventually meet criteria for intellectual developmental disorder have deficits that meet criteria for global developmental delay.

When intellectual developmental disorder is associated with a genetic syndrome, there may be a characteristic physical appearance (e.g., as in Down syndrome). Some syndromes have a *behavioral phenotype*, which refers to specific behaviors that are characteristic of particular genetic disorder (e.g., Lesch-Nyhan syndrome). In acquired forms, the onset may be abrupt following an illness such as meningitis or encephalitis or head trauma occurring during the developmental period. When intellectual developmental disorder results from a loss of previously acquired cognitive skills, as in severe traumatic brain injury, the diagnoses of intellectual developmental disorder and of a neurocognitive disorder may both be assigned.

Although intellectual developmental disorder is generally nonprogressive, in certain genetic disorders (e.g., Rett syndrome) there are periods of worsening, followed by stabilization, and in others (e.g., Sanfilippo syndrome, Down syndrome) progressive worsening of intellectual function in varying degrees. In some cases, the progressive worsening of intellectual functioning may represent the overlay of neurocognitive disorder that develops in adulthood (i.e., persons with Down syndrome being at high risk for developing neurocognitive disorder due to Alzheimer's disease in adulthood). In this situation, both diagnoses, intellectual developmental disorder and neurocognitive disorder, are given.

The disorder is generally lifelong, although severity levels may change over time. The course may be influenced by underlying medical or genetic conditions and co-occurring conditions (e.g., hearing or visual impairments, epilepsy). Early and ongoing interventions may improve adaptive functioning throughout childhood and adulthood. In some cases, these result in significant improvement of intellectual functioning, such that the

diagnosis of intellectual developmental disorder is no longer appropriate. Thus, it is common practice when assessing infants and young children to delay diagnosis of intellectual developmental disorder until after an appropriate course of intervention is provided. For older children and adults, the extent of support provided may allow for full participation in all activities of daily living and improved adaptive function. Diagnostic

assessments must determine whether improved adaptive skills are the result of a stable, generalized new skill acquisition (in which case the diagnosis of intellectual developmental disorder may no longer be appropriate) or whether the improvement is contingent on the presence of supports and ongoing interventions (in which case the diagnosis of intellectual developmental disorder may still be appropriate).

Risk and Prognostic Factors

Genetic and physiological. Prenatal etiologies include genetic syndromes (e.g., sequence variations or copy number variants involving one or more genes; chromosomal disorders), inborn errors of metabolism, brain malformations, maternal disease (including placental disease), and environmental influences (e.g., alcohol, other drugs, toxins, teratogens). Perinatal causes include a variety of labor and delivery–related events leading to neonatal encephalopathy. Postnatal causes include hypoxic ischemic injury, traumatic brain injury, infections, demyelinating disorders, seizure disorders (e.g., infantile spasms), severe and chronic social deprivation, and toxic metabolic syndromes and intoxications (e.g., lead, mercury).

Culture-Related Diagnostic Issues

Intellectual developmental disorder occurs across ethnoracial groups. Prevalence differences across social and cultural contexts may be due to variation in environmental risks (e.g., perinatal injury, chronic social deprivation) for the disorder that are associated with socioeconomic status and access to quality health care. For example, in Western Australia, the population prevalence of intellectual developmental disorder among Aboriginal children is 39 per 1,000 people, as opposed to 16 per 1,000 for the more affluent non-Aboriginal youth population. Cultural sensitivity and knowledge of sociostructural conditions are needed during assessment, and the individual's socioeconomic, ethnic, cultural, and linguistic background; available experiences; and adaptive functioning within his or her community and cultural setting must be considered. Cultural explanations for intellectual developmental disorder vary and may include cultural beliefs about supernatural influences and punishment for presumed or

actual wrongdoing by the mother or parents, which can be associated with shame and underreporting of the disorder.

Sex- and Gender-Related Diagnostic Issues

Overall, males are more likely than females to be diagnosed with both mild (average male:female ratio 1.6:1) and severe (average male:female ratio 1.2:1) forms of intellectual developmental disorder. However, sex ratios vary widely in reported studies. Sex-linked genetic factors, sex differences in other genetic factors such as specific copy number variants, and male vulnerability to brain insult may account for some of the sex differences.

Association With Suicidal Thoughts or Behavior

Individuals with intellectual developmental disorder can be at risk for suicide associated with comorbid mental disorder, higher intellectual and adaptive function, and immediate past stressors. Comorbid mental disorder may manifest atypically in intellectual developmental disorder; thus, recognizing comorbidity and screening for suicidal thoughts is important in the assessment process, with particular attention to change in behavior of the individual.

Differential Diagnosis

The diagnosis of intellectual developmental disorder should be made whenever Criteria A, B, and C are met. A diagnosis of intellectual developmental disorder should not be assumed because of a particular genetic or medical condition. A genetic syndrome linked to intellectual developmental disorder should be noted as a concurrent diagnosis with the intellectual developmental disorder.

Major and mild neurocognitive disorders. Intellectual developmental disorder is categorized as a neurodevelopmental disorder and is distinct from the neurocognitive disorders, which are characterized by a loss of cognitive functioning. Major neurocognitive disorder may co-occur with intellectual developmental disorder (e.g., an individual with Down syndrome who develops Alzheimer’s disease, or an individual with intellectual

developmental disorder who loses further cognitive capacity following a head injury). In such cases, the diagnoses of intellectual developmental disorder and neurocognitive disorder may both be given. Moreover, when there is stabilization of cognitive functioning following traumatic or nontraumatic brain injury with onset in the developmental period (childhood and adolescence), and there is no continuing cognitive decline, both neurocognitive disorder and intellectual developmental disorder diagnoses can be used if diagnostic criteria are met for intellectual developmental disorder.

Communication disorders and specific learning disorder. These neurodevelopmental disorders are specific to the communication and learning domains and do not show deficits in intellectual and adaptive behavior. They may co-occur with intellectual developmental disorder. Both diagnoses are made if full criteria are met for intellectual developmental disorder and a communication disorder or specific learning disorder.

Autism spectrum disorder. Intellectual developmental disorder is common among individuals with autism spectrum disorder. Assessment of intellectual ability may be complicated by social-communication and behavior deficits inherent to autism spectrum disorder, which may interfere with understanding and complying with test procedures. Appropriate assessment of intellectual functioning in autism spectrum disorder is essential, with reassessment across the developmental period, because IQ scores in autism spectrum disorder may be unstable, particularly in early childhood.

Comorbidity

Co-occurring neurodevelopmental and other mental and medical conditions are frequent in intellectual developmental disorder, with rates of some conditions (e.g., mental disorders, cerebral palsy, and epilepsy) three to four times higher than in the general population. The prognosis and outcome of co-occurring diagnoses may be influenced by the presence of intellectual developmental disorder. Assessment procedures may require modifications because of associated disorders, including communication disorders, autism spectrum disorder, and motor, sensory, or other disorders. Knowledgeable informants are essential for identifying symptoms such as irritability, mood

dysregulation, aggression, eating problems, and sleep problems, and for assessing adaptive functioning in various community settings.

The most common co-occurring neurodevelopmental and other mental disorders are attention-deficit/hyperactivity disorder; depressive and bipolar disorders; anxiety disorders; autism spectrum disorder; stereotypic movement disorder (with or without self-injurious behavior); impulse-control disorders; and major neurocognitive disorder. Major depressive disorder may occur throughout the range of severity of intellectual developmental disorder. Self-injurious behavior requires prompt diagnostic attention and may warrant a separate diagnosis of stereotypic movement disorder. Individuals with intellectual developmental disorder, particularly those with more severe intellectual developmental disorder, may also exhibit aggression and disruptive behaviors, including harm of others or property destruction.

Individuals with intellectual developmental disorder disproportionately have more health problems, including obesity, than the general population. Frequently they cannot verbalize physical symptoms they are experiencing. This may lead to health problems being undiagnosed and untreated.

Relationship to Other Classifications

ICD-11 uses the term *disorders of intellectual development* to indicate that these are disorders that involve impaired brain functioning early in life. These disorders are described in ICD-11 as a metasyndrome occurring in the developmental period analogous to dementia or major neurocognitive disorder in later life. There are four subtypes of disorders of intellectual development in ICD-11: mild, moderate, severe, and profound.

The American Association on Intellectual and Developmental Disabilities (AAIDD) uses the term *intellectual disability*. The AAIDD's classification is multidimensional rather than categorical and is based on the disability construct. Rather than listing severity specifiers as is done in DSM-5, the AAIDD emphasizes a profile of supports based on severity.

Global Developmental Delay

F88

This diagnosis is reserved for individuals *under* the age of 5 years when the clinical severity level cannot be reliably assessed during early childhood. This category is diagnosed when an individual fails to meet expected developmental milestones in several areas of intellectual functioning, and applies to individuals who are unable to undergo systematic assessments of intellectual functioning, including children who are too young to participate in standardized testing. This category requires reassessment after a period of time.

Unspecified Intellectual Developmental Disorder (Intellectual Disability)

F79

This category is reserved for individuals *over* the age of 5 years when assessment of the degree of intellectual developmental disorder (intellectual disability) by means of locally available procedures is rendered difficult or impossible because of associated sensory or physical impairments, as in blindness or prelingual deafness; locomotor disability; or presence of severe problem behaviors or co-occurring mental disorder. This category should only be used in exceptional circumstances and requires reassessment after a period of time.

Communication Disorders

Disorders of communication include deficits in language, speech, and communication. *Speech* is the expressive production of sounds and includes an individual’s articulation, fluency, voice, and resonance quality. *Language* includes the form, function, and use of a conventional system of symbols (i.e., spoken words, sign language, written words, pictures) in a

rule-governed manner for communication. *Communication* includes any verbal or nonverbal behavior (whether intentional or unintentional) that has the potential to influence the behavior, ideas, or attitudes of another individual. Assessments of speech, language, and communication abilities must take into account the individual's cultural and language context, particularly for individuals growing up in bilingual environments. The standardized measures of language development and of nonverbal intellectual capacity must be relevant for the cultural and linguistic group (i.e., tests developed and standardized for one group may not provide appropriate norms for a different group). The diagnostic category of communication disorders includes the following: language disorder, speech sound disorder, childhood-onset fluency disorder (stuttering), social (pragmatic) communication disorder, and unspecified communication disorders Sex differences in the development of early communication may give rise to higher prevalence rates of communication disorders in boys compared with girls. Given the associated features of communication disorders and the relationship of communication to other developmental domains, communication disorders have high rates of comorbidity with other neurodevelopmental disorders (e.g., autism spectrum disorder, attention-deficit/hyperactivity disorder (ADHD), specific learning disorder, intellectual developmental disorder [intellectual disability]), mental disorders (e.g., anxiety disorders), and some medical conditions (e.g., seizure disorders, specific chromosome abnormalities).

Language Disorder

Diagnostic Criteria F80.2

A. Persistent difficulties in the acquisition and use of language across modalities (i.e., spoken, written, sign language, or other) due to deficits in comprehension or production that include the following:
   1. Reduced vocabulary (word knowledge and use).
   2. Limited sentence structure (ability to put words and word endings together to form sentences based on the rules of grammar and morphology).
   3. Impairments in discourse (ability to use vocabulary and connect sentences to explain or describe a topic or series of events or have a conversation).

B. Language abilities are substantially and quantifiably below those expected for age, resulting in functional limitations in effective communication, social participation, academic achievement, or occupational performance, individually or in any combination.

C. Onset of symptoms is in the early developmental period.

D. The difficulties are not attributable to hearing or other sensory impairment, motor dysfunction, or another medical or neurological condition and are not better explained by intellectual developmental disorder (intellectual disability) or global developmental delay.

Diagnostic Features

The essential features of language disorder are difficulties in the acquisition and use of language due to deficits in the comprehension or production of vocabulary, grammar, sentence structure, and discourse. The language deficits are evident in spoken communication, written communication, or sign language. Language learning and use is dependent on both receptive and expressive skills. *Expressive ability* refers to the production of vocal,

gestural, or verbal signals, while *receptive ability* refers to the process of receiving and comprehending language messages. Language skills need to be assessed in both expressive and receptive modalities as these may differ in severity.

Language disorder usually affects vocabulary and grammar, and these effects then limit the capacity for discourse. The child's first words and phrases are likely to be delayed

in onset; vocabulary size is smaller and less varied than expected; and sentences are shorter and less complex with grammatical errors, especially in past tense. Deficits in comprehension of language are frequently underestimated, as children may be good at using context to infer meaning. There may be word-finding problems, impoverished verbal definitions, or poor understanding of synonyms, multiple meanings, or word play appropriate for age and culture. Problems with remembering new words and sentences are manifested by difficulties following instructions of increasing length, difficulties rehearsing strings of verbal information (e.g., remembering a phone number or a shopping list), and difficulties remembering novel sound sequences, a skill that may be important for learning new words. Difficulties with discourse are shown by a reduced ability to provide adequate information about the key events and to narrate a coherent story.

The language difficulty is manifest by abilities substantially and quantifiably below that expected for age and significantly interfering with academic achievement, occupational performance, effective communication, or socialization (Criterion B). A diagnosis of language disorder is made based on the synthesis of the individual's history, direct clinical observation in different contexts (i.e., home, school, or work), and scores from standardized tests of language ability that can be used to guide estimates of severity.

Associated Features

Individuals, even children, can be adept at accommodating to their limited language. They may appear to be shy or reticent to talk. Affected

individuals may prefer to communicate only with family members or other familiar individuals. Although these social indicators are not diagnostic of a language disorder, if they are notable and persistent, they warrant referral for a full language assessment.

Development and Course

Language acquisition is marked by changes from onset in toddlerhood to the adult level of competency that appears during adolescence. Changes appear across the dimensions of language (sounds, words, grammar, narratives/expository texts, and conversational skills) in age-graded increments and synchronies. Language disorder emerges during the early developmental period; however, there is considerable variation in early vocabulary acquisition and early word combinations. Individual differences in early childhood are not, as single indicators, highly predictive of later outcomes, although a late onset of language at age 24 months in a population-based sample was the best predictor of outcomes at age 7 years. By age 4 years, individual differences in language ability are more stable, with better measurement accuracy, and are highly predictive of later outcomes. Language disorder diagnosed in children age 4 years and older is likely to be stable over time and typically persists into adulthood, although the particular profile of language strengths and deficits is likely to change over the course of development.

Language disorders can have social consequences across the lifespan. Children with language disorders are at risk for peer victimization. For females with childhood language disorders, there could be almost three times the risk compared with unaffected children for sexual assault in adulthood.

Risk and Prognostic Factors

Children with receptive language impairments have a poorer prognosis than those with predominantly expressive impairments. Receptive language impairments are more resistant to treatment, and difficulties with reading comprehension are frequently seen.

Environmental. Bilingualism does not cause or worsen a language disorder, but children who are bilingual may demonstrate delays or differences in

language development. A

language disorder in bilingual children will affect both languages; therefore, assessment across both languages is important to consider.

Genetic and physiological. Language disorders are highly heritable, and family members are more likely to have a history of language impairment. Population-based twin studies consistently report substantial heritability for language disorder, and molecular studies suggest multiple genes interacting on causal pathways.

Differential Diagnosis

Normal variations in language. Language disorder needs to be distinguished from normal developmental variations, and this distinction may be difficult to make before age 4 years. Regional, social, or cultural/ethnic variations of language (e.g., dialects) must be considered when an individual is being assessed for language impairment.

Hearing or other sensory impairment. Hearing impairment needs to be excluded as the primary cause of language difficulties. Language deficits may be associated with a hearing impairment, other sensory deficit, or a speech-motor deficit. When language deficits are in excess of those usually associated with these problems, a diagnosis of language disorder may be made.

Intellectual developmental disorder (intellectual disability). Language impairment is often the presenting feature of intellectual developmental disorder. However, the definitive diagnosis of intellectual developmental disorder may not be made until the child is able to complete standardized assessments. Language disorder can occur with varying degrees of intellectual ability, and a discrepancy between verbal and nonverbal ability is not necessary for a diagnosis of language disorder.

Autism spectrum disorder. Autism spectrum disorder frequently manifests with delayed language development. However, autism spectrum disorder is often accompanied by behaviors not present in language disorder, such as lack of social interest or unusual social interactions (e.g., pulling individuals

by the hand without any attempt to look at them), odd play patterns (e.g., carrying toys around but never playing with them), unusual communication patterns (e.g., knowing the alphabet but not responding to own name), and rigid adherence to routines and repetitive behaviors (e.g., flapping, spinning, echolalia).

Neurological disorders. Language disorder can be acquired in association with neurological disorders, including epilepsy (e.g., acquired aphasia or Landau-Kleffner syndrome).

Language regression. Loss of speech and language in a child at any age warrants thorough assessment to determine if there is a specific neurological condition, such as Landau-Kleffner syndrome. Language loss may be a symptom of seizures, and a diagnostic assessment is necessary to exclude the presence of epilepsy (e.g., routine and sleep electroencephalogram). Declines in critical social and communication behaviors during the first 2 years of life are evident in most children presenting with autism spectrum disorder and should signal the need for autism spectrum disorder assessment.

Comorbidity

Language disorder may be associated with other neurodevelopmental disorders in terms of specific learning disorder (literacy and numeracy), intellectual developmental disorder, attention-deficit/hyperactivity disorder, autism spectrum disorder, and developmental coordination disorder. It is also associated with social (pragmatic) communication disorder. In clinical samples, language disorder may co-occur with speech sound disorder, although data from a large population-based sample of 6-year-old children in the United States suggest comorbidity might be rare (1.3%). A positive family history of speech or language disorders is often present.

Speech Sound Disorder

Diagnostic Criteria

F80.0

A. Persistent difficulty with speech sound production that interferes with speech intelligibility or prevents verbal communication of messages.
B. The disturbance causes limitations in effective communication that interfere with social participation, academic achievement, or occupational performance, individually or in any combination.
C. Onset of symptoms is in the early developmental period.
D. The difficulties are not attributable to congenital or acquired conditions, such as cerebral palsy, cleft palate, deafness or hearing loss, traumatic brain injury, or other medical or neurological conditions.

Diagnostic Features

Speech sound production describes the clear articulation of the phonemes (i.e., individual sounds) that in combination make up spoken words. Speech sound production requires both the phonological knowledge of speech sounds and the ability to coordinate the movements of the articulators (i.e., the jaw, tongue, and lips,) with breathing and vocalizing for speech. Children with speech production difficulties may experience difficulty with phonological knowledge of speech sounds or the ability to coordinate movements for speech in varying degrees. A speech sound disorder is diagnosed when speech sound production is not what would be expected based on the child's age and developmental stage and when the deficits are not the result of a physical, structural, neurological, or hearing impairment. Among typically developing children at age 3 years, overall speech should be intelligible, whereas at age 2 years, only 50% may be understandable. Boys are more likely (range of 1.5–1.8 to 1.0) to have a speech sound disorder than girls.

Associated Features

Language disorder may be found to co-occur with speech sound disorder, although co-occurrences are rare by age 6 years. A positive family history

of speech or language disorders is often present.

If the ability to rapidly coordinate the articulators is a particular aspect of difficulty, there may be a history of delay or incoordination in acquiring skills that also utilize the articulators and related facial musculature; among others, these skills include chewing, maintaining mouth closure, and blowing the nose. Other areas of motor coordination may be impaired as in developmental coordination disorder. The terms *childhood apraxia of speech* and *verbal dyspraxia* are used for speech production problems with motor components.

Development and Course

Learning to produce speech sounds clearly and accurately and learning to produce connected speech fluently are developmental skills. Articulation of speech sounds follows a developmental pattern, which is reflected in the age norms of standardized tests. It is not unusual for typically developing children to use developmental processes for shortening words and syllables as they are learning to talk, but their progression in mastering speech sound production should result in mostly intelligible speech by age 3 years. Children with speech sound disorder continue to use immature phonological simplification processes past the age when most children can produce words clearly.

Most speech sounds should be produced clearly and most words should be pronounced accurately according to age and community norms by age 5 years. The most

frequently misarticulated sounds in English also tend to be learned later, leading them to be called the “late eight” (*l*, *r*, *s*, *z*, *th*, *ch*, *dzh*, and *zh*). Misarticulation of any of these sounds by itself could be considered within normal limits up to age 8 years; however, when multiple sounds are involved, it is important to target some of those sounds as part of a plan to improve intelligibility, rather than waiting until the age at which almost all children can produce them accurately. Lisping (i.e., misarticulating sibilants) is particularly common and may involve frontal or lateral patterns

of airstream direction. It may be associated with a tongue-thrust swallowing pattern.

Most children with speech sound disorder respond well to treatment, and speech difficulties improve over time, and thus the disorder may not be lifelong. However, when a language disorder is also present, the speech disorder has a poorer prognosis and may be associated with specific learning disorder.

Differential Diagnosis

Normal variations in speech. Regional, social, or cultural/ethnic variations of speech should be considered before making the diagnosis. Bilingual children may demonstrate an overall lower intelligibility rating, make more overall consonant and vowel errors, and produce more uncommon error patterns than monolingual English-speaking children when assessed only in English.

Hearing or other sensory impairment. Those who are deaf or hard of hearing may have speech sound production errors. When speech deficits are in excess of those usually associated with these problems, a diagnosis of speech sound disorder may be made.

Structural deficits. Speech impairment may be due to structural deficits (e.g., cleft palate).

Dysarthria. Speech impairment may be attributable to a motor disorder, such as cerebral palsy. Neurological signs, as well as distinctive features of voice, differentiate dysarthria from speech sound disorder, although in young children (under 3 years) differentiation may be difficult, particularly when there is no or minimal general body motor involvement (as in, e.g., Worster-Drought syndrome).

Selective mutism. Limited use of speech may be a sign of selective mutism, an anxiety disorder that is characterized by a lack of speech in one or more contexts or settings. Selective mutism may develop in children with a speech disorder because of embarrassment about their impairments, but many children with selective mutism exhibit normal speech in “safe” settings, such as at home or with close friends.

Comorbidity

Speech may be differentially impaired in certain genetic conditions (e.g., Down syndrome, 22q deletion, *FoxP2* gene mutation). If present, these should also be coded.

Childhood-Onset Fluency Disorder (Stuttering)

Diagnostic Criteria F80.81

A. Disturbances in the normal fluency and time patterning of speech that are inappropriate for the individual's age and language skills, persist over time, and are characterized by frequent and marked occurrences of one (or more) of the following:

1. Sound and syllable repetitions.
2. Sound prolongations of consonants as well as vowels.

3. Broken words (e.g., pauses within a word).
4. Audible or silent blocking (filled or unfilled pauses in speech).
5. Circumlocutions (word substitutions to avoid problematic words).
6. Words produced with an excess of physical tension.
7. Monosyllabic whole-word repetitions (e.g., "I-I-I-I see him").

B. The disturbance causes anxiety about speaking or limitations in effective communication, social participation, or academic or occupational performance, individually or in any combination.

C. The onset of symptoms is in the early developmental period. (**Note:** Later-onset cases are diagnosed as F98.5 adult-onset fluency disorder.)

D. The disturbance is not attributable to a speech-motor or sensory deficit, dysfluency associated with neurological insult (e.g., stroke, tumor, trauma), or another medical condition and is not better explained by another mental disorder.

Diagnostic Features

The essential feature of childhood-onset fluency disorder (stuttering) is a disturbance in the normal fluency and time patterning of speech that is inappropriate for the individual's age. This disturbance is characterized by frequent repetitions or prolongations of sounds or syllables and by other types of speech dysfluencies, including broken words (e.g., pauses within a word), audible or silent blocking (i.e., filled or unfilled pauses in speech), circumlocutions (i.e., word substitutions to avoid problematic words), words produced with an excess of physical tension, and monosyllabic whole-word repetitions (e.g., "I-I-I-I see him"). The disturbance in fluency may interfere with academic or occupational achievement and with social communication. The extent of the disturbance varies from situation to situation and often is more severe when there is special pressure to communicate (e.g., giving a report at school, interviewing for a job). Dysfluency is often absent during oral reading, singing, or talking to inanimate objects or to pets.

Associated Features

Fearful anticipation of the problem may develop. The speaker may attempt to avoid dysfluencies by linguistic mechanisms (e.g., altering the rate of speech, avoiding certain words or sounds) or by avoiding certain speech situations, such as telephoning or public speaking. In addition to being features of the condition, stress and anxiety have been shown to exacerbate dysfluency.

Childhood-onset fluency disorder may also be accompanied by motor movements (e.g., eye blinks, tics, tremors of the lips or face, jerking of the

head, breathing movements, fist clenching). Children with fluency disorder show a range of language abilities, and the relationship between fluency disorder and language abilities is unclear.

Studies have shown both structural and functional neurological differences in children who stutter. Males are more likely to stutter than females, with estimates varying depending on the age and possible cause of stuttering. Causes of stuttering are multifactorial, including certain genetic and neurophysiological factors.

Development and Course

Childhood-onset fluency disorder, or developmental stuttering, occurs by age 6 for 80%–90% of affected individuals, with age at onset ranging from 2 to 7 years. The onset can be insidious or more sudden. Typically, dysfluencies start gradually, with repetition of initial consonants, first words of a phrase, or long words. The child may not be aware of dysfluencies. As the disorder progresses, the dysfluencies become more frequent and interfering, occurring on the most meaningful words or phrases in the utterance. As the child becomes aware of the speech difficulty, he or she may develop mechanisms for avoiding the

dysfluencies and emotional responses, including avoidance of public speaking and use of short and simple utterances. Longitudinal research shows that 65%–85% of children recover from the dysfluency, with severity of fluency disorder at age 8 years predicting recovery or persistence into adolescence and beyond.

Risk and Prognostic Factors

Genetic and physiological. The risk of stuttering among first-degree biological relatives of individuals with childhood-onset fluency disorder is more than three times the risk in the general population. To date, mutations of four genes that underlie some cases of stuttering have been identified.

Functional Consequences of Childhood-Onset Fluency Disorder (Stuttering)

In addition to being features of the condition, stress and anxiety can exacerbate dysfluency. Impairment of social functioning may result from this anxiety. Negative communication attitudes may be a functional consequence of stuttering starting in the preschool years and increasing with age.

Differential Diagnosis

Sensory deficits. Dysfluencies of speech may be associated with a hearing impairment or other sensory deficit or a speech-motor deficit. When the speech dysfluencies are in excess of those usually associated with these problems, a diagnosis of childhood-onset fluency disorder may be made.

Normal speech dysfluencies. The disorder must be distinguished from normal dysfluencies that occur frequently in young children, which include whole-word or phrase repetitions (e.g., “I want, I want ice cream”), incomplete phrases, interjections, unfilled pauses, and parenthetical remarks. If these difficulties increase in frequency or complexity as the child grows older, a diagnosis of childhood-onset fluency disorder may be appropriate.

Specific learning disorder, with impairment in reading. Children who have dysfluencies when they read aloud may be diagnosed mistakenly as having a reading disorder. Oral reading fluency typically is measured by timed assessments. Slower reading rates may not accurately reflect the actual reading ability of children who stutter.

Bilingualism. It is necessary to distinguish between dysfluencies resulting from attempts to learn a new language and dysfluencies that indicate a fluency disorder, which typically appear in both languages.

Medication side effects. Stuttering may occur as a side effect of medication and may be detected by a temporal relationship with exposure to the medication.

Adult-onset dysfluencies. If onset of dysfluencies is during or after adolescence, it is an “adult-onset dysfluency” rather than a neurodevelopmental disorder. Adult-onset dysfluencies are associated with specific neurological insults and a variety of medical conditions and mental disorders and may be specified with them, but they are not a DSM-5 diagnosis.

Tourette's disorder. Vocal tics and repetitive vocalizations of Tourette's disorder should be distinguishable from the repetitive sounds of childhood-onset fluency disorder by their nature and timing.

Comorbidity

Childhood-onset fluency disorder can co-occur with other disorders, such as attention-deficit/hyperactivity disorder, autism spectrum disorder, intellectual developmental

disorder (intellectual disability), language disorder or specific learning disorder, seizure disorders, social anxiety disorder, speech sound disorder, and other developmental disorders.

Social (Pragmatic) Communication Disorder

Diagnostic Criteria F80.82

A. Persistent difficulties in the social use of verbal and nonverbal communication as manifested by all of the following:
   1. Deficits in using communication for social purposes, such as greeting and sharing information, in a manner that is appropriate for the social context.
   2. Impairment of the ability to change communication to match context or the needs of the listener, such as speaking differently in a classroom than on a playground, talking differently to a child than to an adult, and avoiding use of overly formal language.
   3. Difficulties following rules for conversation and storytelling, such as taking turns in conversation, rephrasing when misunderstood, and knowing how to use verbal and nonverbal signals to regulate interaction.

4. Difficulties understanding what is not explicitly stated (e.g., making inferences) and nonliteral or ambiguous meanings of language (e.g., idioms, humor, metaphors, multiple meanings that depend on the context for interpretation).

B. The deficits result in functional limitations in effective communication, social participation, social relationships, academic achievement, or occupational performance, individually or in combination.

C. The onset of the symptoms is in the early developmental period (but deficits may not become fully manifest until social communication demands exceed limited capacities).

D. The symptoms are not attributable to another medical or neurological condition or to low abilities in the domains of word structure and grammar, and are not better explained by autism spectrum disorder, intellectual developmental disorder (intellectual disability), global developmental delay, or another mental disorder.

Diagnostic Features

Social (pragmatic) communication disorder is characterized by a primary difficulty with *pragmatics* (i.e., the social use of language and communication), as manifested by deficits in understanding and following social rules of both verbal and nonverbal communication in naturalistic contexts, changing language according to the needs of the listener or situation, and following rules for conversations and storytelling. The deficits in social communication result in functional limitations in effective communication, social participation, development of social relationships, academic achievement, or occupational performance. The deficits are not better explained by low abilities in the domains of structural language or cognitive ability or by autism spectrum disorder.

Associated Features

The most common associated feature of social (pragmatic) communication disorder is language impairment, which is characterized by a history of

delay in reaching language milestones, and historical, if not current, structural language problems (see “Language Disorder” earlier in this chapter). Individuals with social communication deficits may

avoid social interactions. Attention-deficit/hyperactivity disorder (ADHD), emotional and behavioral problems, and specific learning disorders are also more common among affected individuals.

Development and Course

Because social (pragmatic) communication depends on adequate developmental progress in speech and language, diagnosis of social (pragmatic) communication disorder is rare among children younger than 4 years. By age 4 or 5 years, most children should possess adequate speech and language abilities to permit identification of specific deficits in social communication. Milder forms of the disorder may not become apparent until early adolescence, when language and social interactions become more complex.

The outcome of social (pragmatic) communication disorder is variable, with some children improving substantially over time and others continuing to have difficulties persisting into adulthood. Even among those who have significant improvements, the early deficits in pragmatics may cause lasting impairments in social relationships and behavior and also low performance of other related skills, such as written expression, reading comprehension, and oral reading.

Risk and Prognostic Factors

Genetic and physiological. A family history of autism spectrum disorder, communication disorders, or specific learning disorder appears to increase the risk for social (pragmatic) communication disorder; this includes siblings of children with these disorders who may present with early symptoms of social (pragmatic) communication disorder.

Differential Diagnosis

Autism spectrum disorder.

Autism spectrum disorder is the primary diagnostic consideration for individuals presenting with social communication deficits. The two disorders can be differentiated by the presence in autism spectrum disorder of restricted/repetitive patterns of behavior, interests, or activities and their absence in social (pragmatic) communication disorder. Individuals with autism spectrum disorder may only display the restricted/repetitive patterns of behavior, interests, and activities during the early developmental period, so a comprehensive history should be obtained. Current absence of symptoms would not preclude a diagnosis of autism spectrum disorder, if the restricted interests and repetitive behaviors were present in the past. A diagnosis of social (pragmatic) communication disorder should be considered only if the current symptoms or developmental history fails to reveal evidence of symptoms that meet the diagnostic criteria for restricted/repetitive patterns of behavior, interests, or activities of autism spectrum disorder (i.e., Criterion B) causing current impairment. The social communication symptoms may be milder in social (pragmatic) communication disorder than in autism spectrum disorder, although qualitatively similar.

Attention-deficit/hyperactivity disorder. Primary deficits of ADHD may cause impairments in social communication and functional limitations of effective communication, social participation, or academic achievement.

Social anxiety disorder. The symptoms of social (pragmatic) communication disorder overlap with those of social anxiety disorder. The differentiating feature is the timing of the onset of symptoms. In social (pragmatic) communication disorder, the individual has never had effective social communication; in social anxiety disorder, the social communication skills developed appropriately but are not utilized because of anxiety, fear, or distress about social interactions.

Intellectual developmental disorder (intellectual disability) and global developmental delay.

Social communication skills may be deficient among individuals with global developmental delay or intellectual developmental disorder, but a

separate diagnosis is not given unless the social communication deficits are clearly in excess of the intellectual limitations.

Unspecified Communication Disorder

F80.9

This category applies to presentations in which symptoms characteristic of communication disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for communication disorder or for any of the disorders in the neurodevelopmental disorders diagnostic class. The unspecified communication disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for communication disorder or for a specific neurodevelopmental disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Autism Spectrum Disorder

Autism Spectrum Disorder

Diagnostic Criteria **F84.0**

A. Persistent deficits in social communication and social interaction across multiple contexts, as manifested by all of the following, currently or by history (examples are illustrative, not exhaustive; see text):

1. Deficits in social-emotional reciprocity, ranging, for example, from abnormal social approach and failure of normal back-and-forth conversation; to reduced sharing of interests, emotions, or affect; to failure to initiate or respond to social interactions.
2. Deficits in nonverbal communicative behaviors used for social interaction, ranging, for example, from poorly integrated verbal and nonverbal communication; to abnormalities in eye contact and body language or deficits in understanding and use of gestures; to a total lack of facial expressions and nonverbal communication.
3. Deficits in developing, maintaining, and understanding relationships, ranging, for example, from difficulties adjusting behavior to suit various social contexts; to difficulties in sharing imaginative play or in making friends; to absence of interest in peers.

B. Restricted, repetitive patterns of behavior, interests, or activities, as manifested by at least two of the following, currently or by history (examples are illustrative, not exhaustive; see text):

1. Stereotyped or repetitive motor movements, use of objects, or speech (e.g., simple motor stereotypies, lining up toys or flipping objects, echolalia, idiosyncratic phrases).

2. Insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior (e.g., extreme distress at small changes, difficulties with transitions, rigid thinking patterns, greeting rituals, need to take same route or eat same food every day).
3. Highly restricted, fixated interests that are abnormal in intensity or focus (e.g., strong attachment to or

preoccupation with unusual objects, excessively circumscribed or perseverative interests).

4. Hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of the environment (e.g., apparent indifference to pain/temperature, adverse response to specific sounds or textures, excessive smelling or touching of objects, visual fascination with lights or movement).

C. Symptoms must be present in the early developmental period (but may not become fully manifest until social demands exceed limited capacities, or may be masked by learned strategies in later life).

D. Symptoms cause clinically significant impairment in social, occupational, or other important areas of current functioning.

E. These disturbances are not better explained by intellectual developmental disorder (intellectual disability) or global developmental delay. Intellectual developmental disorder and autism spectrum disorder frequently co-occur; to make comorbid diagnoses of autism spectrum disorder and intellectual developmental disorder, social communication should be below that expected for general developmental level.

Note: Individuals with a well-established DSM-IV diagnosis of autistic disorder, Asperger's disorder, or pervasive developmental disorder not otherwise specified should be given the diagnosis of autism spectrum disorder. Individuals who have marked deficits in social communication, but whose symptoms do not otherwise meet criteria for autism spectrum disorder, should be evaluated for social (pragmatic) communication disorder.

Specify current severity based on social communication impairments and restricted, repetitive patterns of behavior (see Table 2):

Requiring very substantial support

Requiring substantial support

Requiring support

Specify if:

With or without accompanying intellectual impairment

With or without accompanying language impairment

Specify if:

Associated with a known genetic or other medical condition or environmental factor (**Coding note:** Use additional code to identify the associated genetic or other medical condition.)

Associated with a neurodevelopmental, mental, or behavioral problem

Specify if:

With catatonia (refer to the criteria for catatonia associated with another mental disorder, p. 135, for definition) (**Coding note:** Use additional code F06.1 catatonia associated with autism spectrum disorder to indicate the presence of the comorbid catatonia.)

TABLE 2 Severity levels for autism spectrum disorder (examples of level of support needs)

Severity level	Social communication	Restricted, repetitive behaviors
Level 3 "Requiring very substantial support"	Severe deficits in verbal and nonverbal social communication skills cause severe impairments in functioning, very limited initiation of social interactions, and minimal response to social overtures from others. For example, a person with few words of intelligible speech who rarely initiates interaction and, when he or she does, makes unusual approaches to meet needs only and responds to only very direct social approaches.	Inflexibility of behavior, extreme difficulty coping with change, or other restricted/repetitive behaviors markedly interfere with functioning in all spheres. Great distress/difficulty changing focus or action.

Severity level	Social communication	Restricted, repetitive behaviors
Level 2 "Requiring substantial support"	Marked deficits in verbal and nonverbal social communication skills; social impairments apparent even with supports in place; limited initiation of social interactions; and reduced or abnormal responses to social overtures from others. For example, a person who speaks simple sentences, whose interaction is limited to narrow special interests, and who has markedly odd nonverbal communication.	Inflexibility of behavior, difficulty coping with change, or other restricted/repetitive behaviors appear frequently enough to be obvious to the casual observer and interfere with functioning in a variety of contexts. Distress and/or difficulty changing focus or action.
Level 1 "Requiring support"	Without supports in place, deficits in social communication cause noticeable impairments. Difficulty initiating social interactions, and clear examples of atypical or unsuccessful responses to social overtures of others. May appear to have decreased interest in social interactions. For example, a person who is able to speak in full sentences and engages in communication but whose to-and-fro conversation with others fails, and whose attempts to make friends are odd and typically unsuccessful.	Inflexibility of behavior causes significant interference with functioning in one or more contexts. Difficulty switching between activities. Problems of organization and planning hamper independence.

Recording Procedures

It may be helpful to note level of support needed for each of the two core psychopathological domains in Table 2 (e.g., "requiring very substantial support for deficits in social communication and requiring substantial support for restricted, repetitive behaviors"). Specification of "with accompanying intellectual impairment" or "without accompanying

intellectual impairment" should be recorded next. Language impairment specification should be recorded thereafter. If there is accompanying language impairment, the current level of verbal functioning should be

recorded (e.g., “with accompanying language impairment—no intelligible speech” or “with accompanying language impairment—phrase speech”).

For autism spectrum disorder for which the specifiers “associated with a known genetic or other medical condition or environmental factor” or “associated with a neurodevelopmental, mental, or behavioral problem” are appropriate, record autism spectrum disorder associated with (name of condition, disorder, or factor) (e.g., autism spectrum disorder associated with tuberous sclerosis complex). These specifiers apply to presentations in which the listed condition or problem is potentially relevant to the clinical care of the individual and do not necessarily indicate that the condition or problem is causally related to the autism spectrum disorder. If the associated neurodevelopmental, mental, or behavioral problem meets criteria for a neurodevelopmental or other mental disorder, both autism spectrum disorder and the other disorder should be diagnosed.

If catatonia is present, record separately “catatonia associated with autism spectrum disorder.” For more information, see criteria for catatonia associated with another mental disorder in the chapter “Schizophrenia Spectrum and Other Psychotic Disorders.”

Specifiers

The severity specifiers (see Table 2) may be used to describe succinctly the current symptomatology (which might fall below level 1), with the recognition that severity may vary by context and fluctuate over time. Severity of social communication difficulties and restricted, repetitive behaviors should be separately rated. The descriptive severity categories should not be used to determine eligibility for and provision of services. Indeed, individuals with relatively better skills overall may experience different or even greater psychosocial challenges. Thus, service needs can only be developed at an individual level and through discussion of personal priorities and targets.

Regarding the specifier “with or without accompanying intellectual impairment,” understanding the (often uneven) intellectual profile of a child or adult with autism spectrum disorder is necessary for interpreting diagnostic features. Separate estimates of verbal and nonverbal skill are

necessary (e.g., using untimed nonverbal tests to assess potential strengths in individuals with limited language).

To use the specifier “with or without accompanying language impairment,” the current level of verbal functioning should be assessed and described. Examples of the specific descriptions for “with accompanying language impairment” might include no intelligible speech (nonverbal), single words only, or phrase speech. Language level in individuals “without accompanying language impairment” might be further described as speaks in full sentences or has fluent speech. Since receptive language may lag behind expressive language development in autism spectrum disorder, receptive and expressive language skills should be considered separately.

The specifier “associated with a known genetic or other medical condition or environmental factor” can be applied when an individual has a known genetic condition (e.g., Rett syndrome, fragile X syndrome, Down syndrome), a known medical condition (e.g., epilepsy), or a history of environmental exposure in utero to a known teratogen or infection (e.g., fetal valproate syndrome, fetal alcohol syndrome, fetal rubella). This specifier should not be viewed as synonymous with causation of autism spectrum disorder. A condition may be listed as being associated with autism spectrum disorder when it is thought to be potentially clinically relevant or inform care and not because the clinician is asserting a cause. Examples include autism spectrum disorder associated with a unique genomic copy number variant that could be clinically relevant even if the specific abnormality may not have directly caused nor have previously been linked to autism spectrum disorder, or Crohn’s disease, which could exacerbate behavioral symptoms.

The specifier “associated with a neurodevelopmental, mental, or behavioral problem” can be applied to indicate problems (e.g., irritability, sleep problems, self-injurious behavior, or developmental regression) that contribute to the functional formulation or are a focus of treatment. Additional neurodevelopmental, mental, or behavioral disorders should also be noted as separate diagnoses (e.g., attention-deficit/hyperactivity disorder; developmental coordination disorder; disruptive behavior, impulse-control,

and conduct disorders; anxiety, depressive, or bipolar disorders; tics or Tourette's disorder; feeding, elimination, or sleep disorders).

Catatonia can occur as a comorbid condition with autism spectrum disorder. In addition to classic symptoms of posturing, negativism (opposition or no response to instructions or external stimuli), mutism, and stupor, an increase or worsening of stereotypy and self-injurious behavior may form part of the symptom complex of catatonia in the setting of autism spectrum disorder.

Diagnostic Features

The essential features of autism spectrum disorder are persistent impairment in reciprocal social communication and social interaction (Criterion A), and restricted, repetitive patterns of behavior, interests, or activities (Criterion B). These symptoms are present from early childhood and limit or impair everyday functioning (Criteria C and D). The stage at which functional impairment becomes obvious will vary according to characteristics of the individual and his or her environment. Core diagnostic features are evident in the developmental period, but intervention, compensation, and current supports may mask difficulties in at least some contexts. Manifestations of the disorder also vary greatly depending on the severity of the autistic condition, developmental level, chronological age, and possibly gender; hence, the term *spectrum.* Individuals without cognitive or language impairment may have more subtle manifestation of deficits (e.g., Criterion A, Criterion B) than individuals with accompanying intellectual or language impairments and may be making great efforts to mask these deficits. Criterion A deficits in social communication will be more subtle if an individual has better overall communication skills (e.g., is verbally fluent, does not have intellectual impairments). Similarly, Criterion B deficits (i.e., restricted patterns of behavior and interests) may be less obvious if the interests are closer to age-typical norms (e.g., Ancient Egypt or trains as compared to wiggling a string). Autism spectrum disorder encompasses disorders previously referred to as early infantile autism, childhood autism, Kanner's autism, high-functioning autism, atypical autism, pervasive developmental disorder not otherwise specified, childhood disintegrative disorder, and Asperger's disorder.

The impairments in social communication and social interaction specified in Criterion A are pervasive and sustained. Diagnoses are most valid and reliable when based on multiple sources of information, including clinician's observations, caregiver history, and, when possible, self-report. Verbal and nonverbal deficits in social communication have varying manifestations, depending on the individual's age, intellectual level, and language ability, as well as other factors such as treatment history and current support. Many individuals have language deficits, ranging from complete lack of speech through language delays, poor comprehension of speech, echoed speech, or stilted and overly literal language. Even when formal language skills (e.g., vocabulary, grammar) are intact, the use of language for reciprocal social communication is impaired in autism spectrum disorder.

Deficits in social-emotional reciprocity (i.e., the ability to engage with others and share thoughts and feelings) may be shown, for example, in young children with little or no initiation of social interaction and no sharing of emotions, along with reduced or absent imitation of others' behavior. What language exists is often one-sided, lacking in social reciprocity, and used to request or label rather than to comment, share feelings, or converse. In older children and adults without intellectual impairments or language delays, deficits in

social-emotional reciprocity may be most apparent in difficulties processing and responding to complex social cues (e.g., when and how to join a conversation, what not to say). Individuals who have developed compensation strategies for some social challenges still struggle in novel or unsupported situations and suffer from the effort and anxiety of consciously calculating what is socially intuitive for most individuals. This behavior may contribute to lower ascertainment of autism spectrum disorder in these individuals, perhaps especially in adult women. Thus, longer assessments, observation in naturalistic settings, and inquiring about any tolls of social interaction may be needed. If asked about the costs of social interaction, for example, these individuals might respond that social interactions are exhausting for them, that they are unable to concentrate because of the

mental effort in monitoring social conventions, that their self-esteem is adversely affected by being unable to be themselves, and so forth.

Deficits in nonverbal communicative behaviors used for social interaction are manifested by absent, reduced, or atypical use of eye contact (relative to cultural norms), gestures, facial expressions, body orientation, or speech intonation. An early feature of autism spectrum disorder is impaired joint attention as manifested by a lack of pointing, showing, or bringing objects to share interest with others, or failure to follow someone's pointing or eye gaze. Individuals may learn a few functional gestures, but their repertoire is smaller than that of others, and they often fail to use expressive gestures spontaneously in communication. Among young people and adults with fluent language, the difficulty in coordinating nonverbal communication with speech may give the impression of odd, wooden, or exaggerated "body language" during interactions. Impairment may be relatively subtle within individual modes (e.g., someone may have relatively good eye contact when speaking) but noticeable in poor integration of eye contact, gesture, body posture, prosody, and facial expression for social communication, or in difficulty maintaining these for sustained periods or when under stress.

Deficits in developing, maintaining, and understanding relationships should be judged against norms for age, gender, and culture. There may be absent, reduced, or atypical social interest, manifested by rejection of others, passivity, or inappropriate approaches that seem aggressive or disruptive. These difficulties are particularly evident in young children, in whom there is often a lack of shared social play and imagination (e.g., age-appropriate flexible pretend play) and, later, insistence on playing by very fixed rules. Older individuals may struggle to understand what behavior is considered appropriate in one situation but not another (e.g., casual behavior during a job interview), or the different ways that language may be used to communicate (e.g., irony, white lies). There may be an apparent preference for solitary activities or for interacting with much younger or older people. Frequently, there is a desire to establish friendships without a complete or realistic idea of what friendship entails (e.g., one-sided friendships or friendships based solely on shared special interests). Relationships with siblings, coworkers, and caregivers are also important to consider (in terms of reciprocity).

Autism spectrum disorder is also defined by restricted, repetitive patterns of behavior, interests, or activities (as specified in Criterion B), which show a range of manifestations according to age and ability, intervention, and current supports. Stereotyped or repetitive behaviors include simple motor stereotypies (e.g., hand flapping, finger flicking), repetitive use of objects (e.g., spinning coins, lining up toys), and repetitive speech (e.g., echolalia, the delayed or immediate parroting of heard words; use of “you” when referring to self; stereotyped use of words, phrases, or prosodic patterns). Excessive adherence to routines and restricted patterns of behavior may be manifest in resistance to change (e.g., distress at apparently small changes, such as taking an alternative route to school or work; insistence on adherence to rules; rigidity of thinking) or ritualized patterns of verbal or nonverbal behavior (e.g., repetitive questioning, pacing a perimeter). Highly restricted, fixated interests in autism spectrum disorder tend to be abnormal in intensity or focus (e.g., a toddler strongly attached to a pan or piece of string; a child preoccupied with vacuum

cleaners; an adult spending hours writing out timetables). Some fascinations and routines may relate to apparent hyper- or hyporeactivity to sensory input, manifested through extreme responses to specific sounds or textures, excessive smelling or touching of objects, fascination with lights or spinning objects, and sometimes apparent indifference to pain, heat, or cold. Extreme reaction to or rituals involving taste, smell, texture, or appearance of food or excessive food restrictions are common and may be a presenting feature of autism spectrum disorder.

Many individuals with autism spectrum disorder without intellectual or language impairments learn to suppress repetitive behavior in public. In these individuals, repetitive behaviors like rocking or finger flicking may serve an anxiolytic or self-soothing function.

Special interests may be a source of pleasure and motivation and provide avenues for education and employment later in life. Diagnostic criteria may be met when restricted, repetitive patterns of behavior, interests, or activities were clearly present during childhood or at some time in the past, even if symptoms are no longer present.

Criterion D requires that the features must cause clinically significant impairment in social, occupational, or other important areas of current functioning. Criterion E specifies that the social communication deficits, although sometimes accompanied by intellectual developmental disorder (intellectual disability), are not in line with the individual's developmental level; impairments exceed difficulties expected on the basis of developmental level.

Standardized behavioral diagnostic instruments with good psychometric properties, including caregiver interviews, questionnaires and clinician observation measures, are available and can improve reliability of diagnosis over time and across clinicians. However, the symptoms of autism spectrum disorder occur as dimensions without universally accepted cutoff scores for what would constitute a disorder. Thus, the diagnosis remains a clinical one, taking all available information into account, and is not solely dictated by the score on a particular questionnaire or observation measure.

Associated Features

Many individuals with autism spectrum disorder also have intellectual and/or language impairment (e.g., slow to talk, language comprehension behind production). Even those with average or high intelligence usually have an uneven profile of abilities. The gap between intellectual and adaptive functional skills is often large. It is common for individuals with autism to have theory-of-mind deficits (i.e., to have difficulty seeing the world from another person's perspective), but these are not necessarily present in all cases. Executive function deficits are also common but not specific, as are difficulties with central coherence (i.e., being able to understand context or to "see the big picture," and thus tending to overfocus on detail).

Motor deficits are often present, including odd gait, clumsiness, and other abnormal motor signs (e.g., walking on tiptoes). Self-injury (e.g., head banging, biting the wrist) may occur, and disruptive/challenging behaviors are more common in children and adolescents with autism spectrum disorder than other disorders, including intellectual developmental disorder. Some individuals develop catatonic-like motor behavior (slowing and "freezing" mid-action), but these are typically not of the magnitude of a

catatonic episode. However, it is possible for individuals with autism spectrum disorder to experience a marked deterioration in motor symptoms and display a full catatonic episode with symptoms such as mutism, posturing, grimacing, and waxy flexibility. The risk period for comorbid catatonia appears to be greatest in the adolescent years.

Prevalence

Frequencies for autism spectrum disorder across the United States have been reported to be between 1% and 2% of the population, with similar estimates in child and adult

samples. However, prevalence appears to be lower among U.S. African American (1.1%) and Latinx children (0.8%) compared with White children (1.3%), even after the effect of socioeconomic resources is taken into account. The reported prevalence of autism spectrum disorder may be affected by misdiagnosis, delayed diagnosis, or underdiagnosis of individuals from some ethnoracial backgrounds. Prevalence across non-U.S. countries has approached 1% of the population (0.62% median global prevalence), without substantial variation based on geographic region or ethnicity and across child and adult samples. Globally, the male:female ratio in well-ascertained epidemiological samples appears to be 3:1, with concerns about underrecognition of autism spectrum disorder in women and girls.

Development and Course

The age and pattern of onset also should be noted for autism spectrum disorder. The behavioral features of autism spectrum disorder first become evident in early childhood, with some cases presenting a lack of interest in social interaction in the first year of life. Symptoms are typically recognized during the second year of life (age 12–24 months) but may be seen earlier than 12 months if developmental delays are severe, or noted later than 24 months if symptoms are more subtle. The pattern of onset description might include information about early developmental delays or any losses of social or language skills. In cases where skills have been lost, parents or

caregivers may give a history of a gradual or relatively rapid deterioration in social behaviors or language skills. Typically, this would occur between ages 12 and 24 months.

Prospective studies demonstrate that in most cases the onset of autism spectrum disorder is associated with declines in critical social and communication behaviors in the first 2 years of life. Such declines in functioning are rare in other neurodevelopmental disorders and may be an especially useful indicator of the presence of autism spectrum disorder. In rare cases, there is developmental regression occurring after at least 2 years of normal development (previously described as childhood disintegrative disorder), which is much more unusual and warrants more extensive medical investigation (i.e., continuous spike and waves during slow-wave sleep syndrome and Landau-Kleffner syndrome). Often included in these encephalopathic conditions are losses of skills beyond social communication (e.g., loss of self-care, toileting, motor skills) (see also Rett syndrome in the section “Differential Diagnosis” for this disorder).

First symptoms of autism spectrum disorder frequently involve delayed language development, often accompanied by lack of social interest or unusual social interactions (e.g., pulling individuals by the hand without any attempt to look at them), odd play patterns (e.g., carrying toys around but never playing with them), and unusual communication patterns (e.g., knowing the alphabet but not responding to own name). Deafness may be suspected but is typically ruled out. During the second year, odd and repetitive behaviors and the absence of typical play become more apparent. Since many typically developing young children have strong preferences and enjoy repetition (e.g., eating the same foods, watching the same video multiple times), distinguishing restricted and repetitive behaviors that are diagnostic of autism spectrum disorder can be difficult in preschoolers. The clinical distinction is based on the type, frequency, and intensity of the behavior (e.g., a child who daily lines up objects for hours and is very distressed if any item is moved).

Autism spectrum disorder is not a degenerative disorder, and it is typical for learning and compensation to continue throughout life. Symptoms are often most marked in early childhood and early school years, with developmental gains typical in later childhood in at least some areas (e.g., increased interest in social interaction). A small proportion of individuals

deteriorate behaviorally during adolescence, whereas most others improve. While it was once the case that only a minority of individuals with autism spectrum disorder lived and worked independently in adulthood, as diagnosis of autism spectrum

disorder is made more frequently in those who have superior language and intellectual abilities, more individuals are able to find a niche that matches their special interests and skills and thus are productively employed. Access to vocational rehabilitation services significantly improves competitive employment outcomes for transition-age youth with autism spectrum disorder.

In general, individuals with lower levels of impairment may be better able to function independently. However, even these individuals may remain socially naive and vulnerable, have difficulties organizing practical demands without aid, and are prone to anxiety and depression. Many adults report using compensation strategies and coping mechanisms to mask their difficulties in public but suffer from the stress and effort of maintaining a socially acceptable facade. Relatively little is known about old age in autism spectrum disorder, but higher rates of co-occurring medical conditions have been documented in the literature.

Some individuals come for first diagnosis in adulthood, perhaps prompted by the diagnosis of autism in a child in the family or a breakdown of relations at work or home. Obtaining detailed developmental history in such cases may be difficult, and it is important to consider self-reported difficulties. Where clinical observation suggests criteria are currently met, autism spectrum disorder may be diagnosed, particularly if supported by a history of poor social and communication skills in childhood. A compelling report (by parents or another relative) that the individual had ordinary and sustained reciprocal friendships and good nonverbal communication skills throughout childhood would significantly lessen the likelihood of a diagnosis of autism spectrum disorder; however, ambiguous or absent developmental information in itself is not sufficient to rule out a diagnosis of autism spectrum disorder.

Manifestations of the social and communication impairments and restricted/repetitive behaviors that define autism spectrum disorder are clear in the developmental period. In later life, intervention or compensation, as well as current supports, may mask these difficulties in at least some contexts. Overall, symptoms remain sufficient to cause current impairment in social, occupational, or other important areas of functioning.

Risk and Prognostic Factors

The best established prognostic factors for individual outcome within autism spectrum disorder are presence or absence of associated intellectual developmental disorder and language impairment (e.g., functional language by age 5 years is a good prognostic sign) and additional mental health problems. Epilepsy, as a comorbid diagnosis, is associated with greater intellectual disability and lower verbal ability.

Environmental. A variety of risk factors for neurodevelopmental disorders, such as advanced parental age, extreme prematurity, or in utero exposures to certain drugs or teratogens like valproic acid, may broadly contribute to risk of autism spectrum disorder.

Genetic and physiological. Heritability estimates for autism spectrum disorder have ranged from 37% to higher than 90%, based on twin concordance rates, and a more recent five-country cohort estimated heritability at 80%. Currently, as many as 15% of cases of autism spectrum disorder appear to be associated with a known genetic mutation, with different de novo copy number variants or de novo mutations in specific genes associated with the disorder in different families. However, even when a known genetic mutation is associated with autism spectrum disorder, it does not appear to be fully penetrant (i.e., not all individuals with that same genetic abnormality will develop autism spectrum disorder). Risk for the majority of cases appears to be polygenic, with perhaps hundreds of genetic loci making relatively small contributions. Whether these findings apply to all racial/ethnic populations equally is unclear, given the limited inclusion of communities of color in genetic research.

Culture-Related Diagnostic Issues

Cultural differences exist in norms for social interaction, nonverbal communication, and relationships, but individuals with autism spectrum disorder are markedly impaired against the norms for their cultural context. Culture influences the perception of autistic behaviors, the perceived salience of some behaviors over others, and the expectations for child behavior and parenting practices. Considerable discrepancies are found in age at diagnosis of autism spectrum disorder in children from diverse ethnoracial backgrounds; most studies find delayed diagnosis among socially oppressed ethnic and racialized children. In addition to being diagnosed later, African American children are more often misdiagnosed with adjustment or conduct disorder than are White children.

Sex- and Gender-Related Diagnostic Issues

Autism spectrum disorder is diagnosed three to four times more often in males than in females, and on average, age at diagnosis is later in females. In clinic samples, females tend to be more likely to show accompanying intellectual developmental disorder as well as epilepsy, suggesting that girls without intellectual impairments or language delays may go unrecognized, perhaps because of subtler manifestation of social and communication difficulties. In comparison with males with autism spectrum disorder, females may have better reciprocal conversation, and be more likely to share interests, to integrate verbal and nonverbal behavior, and to modify their behavior by situation, despite having similar social understanding difficulties as males. Attempting to hide or mask autistic behavior (e.g., by copying the dress, voice, and manner of socially successful women) may also make diagnosis harder in some females. Repetitive behaviors may be somewhat less evident in females than in males, on average, and special interests may have a more social (e.g., a singer, an actor) or “normative” focus (e.g., horses), while remaining unusual in their intensity. Relative to the general population, rates of gender variance have been reported to be increased in autism spectrum disorder, with higher variance in females compared with males.

Association With Suicidal Thoughts or Behavior

Individuals with autism spectrum disorder are at greater risk for suicide death compared with those without autism spectrum disorder. Children with autism spectrum disorder who had impaired social communication had a higher risk of self-harm with suicidal intent, suicidal thoughts, and suicide plans by age 16 years as compared with those without impaired social communication. Adolescents and young adults with autism spectrum disorder have an increased risk of suicide attempts compared with age- and sex-matched control subjects, even after adjustments for demographic factors and psychiatric comorbidities.

Functional Consequences of Autism Spectrum Disorder

In young children with autism spectrum disorder, lack of social and communication abilities may hamper learning, especially learning through social interaction or in settings with peers. In the home, insistence on routines and aversion to change, as well as sensory sensitivities, may interfere with eating and sleeping and make routine care (e.g., haircuts, dental work) extremely difficult. Adaptive skills are typically below measured IQ. Extreme difficulties in planning, organization, and coping with change negatively impact academic achievement, even for students with above-average intelligence. During adulthood, these individuals may have difficulties establishing independence because of continued rigidity and difficulty with novelty.

Many individuals with autism spectrum disorder, even without intellectual developmental disorder, have poor adult psychosocial functioning as indexed by measures such

as independent living and gainful employment. Functional consequences in old age are unknown, but social isolation and communication problems (e.g., reduced help-seeking) are likely to have consequences for health in older adulthood.

Co-occurring intellectual developmental disorder, epilepsy, mental disorders, and chronic medical conditions may be associated with a higher risk of premature mortality for individuals with autism spectrum disorder. Deaths from injury and poisoning are higher than for the general

population, as are deaths from suicide. Drowning is the leading cause of accidental death in children with autism spectrum disorder.

Differential Diagnosis

Attention-deficit/hyperactivity disorder. Abnormalities of attention (overly focused or easily distracted) are common in individuals with autism spectrum disorder, as is hyperactivity. Moreover, some individuals with ADHD may exhibit social communication deficits such as interrupting others, speaking too loudly, and not respecting personal space. Although potentially difficult to discriminate ADHD from autism spectrum disorder, the developmental course and absence of restricted, repetitive behaviors and unusual interests in ADHD help in differentiating the two conditions. A concurrent diagnosis of ADHD should be considered when attentional difficulties or hyperactivity exceeds that typically seen in individuals of comparable mental age, and ADHD is one of the most common comorbidities in autism spectrum disorder.

Intellectual developmental disorder (intellectual disability) without autism spectrum disorder. Intellectual developmental disorder without autism spectrum disorder may be difficult to differentiate from autism spectrum disorder in very young children. Individuals with intellectual developmental disorder who have not developed language or symbolic skills also present a challenge for differential diagnosis, since repetitive behavior often occurs in such individuals as well. A diagnosis of autism spectrum disorder in an individual with intellectual developmental disorder is appropriate when social communication and interaction are significantly impaired relative to the developmental level of the individual's nonverbal skills (e.g., fine motor skills, nonverbal problem solving). In contrast, intellectual developmental disorder is the appropriate diagnosis when there is no apparent discrepancy between the level of social communicative skills and other intellectual skills.

Language disorders and social (pragmatic) communication disorder. In some forms of language disorder, there may be problems of communication and some secondary social difficulties. However, specific language disorder is not usually associated with abnormal nonverbal communication, nor with

the presence of restricted, repetitive patterns of behavior, interests, or activities.

When an individual shows impairment in social communication and social interactions but does not show restricted and repetitive behavior or interests, criteria for social (pragmatic) communication disorder, instead of autism spectrum disorder, may be met. The diagnosis of autism spectrum disorder supersedes that of social (pragmatic) communication disorder whenever the criteria for autism spectrum disorder are met, and care should be taken to enquire carefully regarding past or current restricted/repetitive behavior.

Selective mutism. In selective mutism, early development is not typically disturbed. The affected child usually exhibits appropriate communication skills in certain contexts and settings. Even in settings where the child is mute, social reciprocity is not impaired, nor are restricted or repetitive patterns of behavior present.

Stereotypic movement disorder. Motor stereotypies are among the diagnostic characteristics of autism spectrum disorder, so an additional diagnosis of stereotypic movement disorder is not given when such repetitive behaviors are better explained by the presence

of autism spectrum disorder. However, when stereotypies cause self-injury and become a focus of treatment, both diagnoses may be appropriate.

Rett syndrome. Disruption of social interaction may be observed during the regressive phase of Rett syndrome (typically between ages 1 and 4 years); thus, a substantial proportion of affected young girls may have a presentation that meets diagnostic criteria for autism spectrum disorder. However, after this period, most individuals with Rett syndrome improve their social communication skills, and autistic features are no longer a major area of concern. Consequently, autism spectrum disorder should be considered only when all diagnostic criteria are met.

Symptoms associated with anxiety disorders. The overlap of anxiety symptoms with the core symptoms of autism spectrum disorder can make the classification of anxiety symptoms in autism spectrum disorder challenging.

For example, social withdrawal and repetitive behaviors are core features of autism spectrum disorder but may also be expressions of anxiety. The most common anxiety disorders in autism spectrum disorder are specific phobia (in up to 30% of cases), and social anxiety and agoraphobia (in as many as 17% of cases).

Obsessive-compulsive disorder. Repetitive behavior is a defining feature of both obsessive-compulsive disorder and autism spectrum disorder. In both conditions, repetitive behaviors are considered to be inappropriate or odd. In obsessive-compulsive disorder, intrusive thoughts are often related to contamination, organization, or sexual or religious themes. Compulsions are performed in response to these intrusive thoughts in attempts to relieve anxiety. In autism spectrum disorder, repetitive behaviors classically include more stereotyped motor behaviors, such as hand flapping and finger shaking or more complex behaviors, such as insistence on routines or lining up objects. Contrary to obsessive-compulsive disorder, repetitive behaviors in autism spectrum disorder may be perceived as pleasurable and reinforcing.

Schizophrenia. Schizophrenia with childhood onset usually develops after a period of normal, or near normal, development. A prodromal state has been described in which social impairment and atypical interests and beliefs occur, which could be confused with the social deficits and restricted fixated interests seen in autism spectrum disorder. Hallucinations and delusions, which are defining features of schizophrenia, are not features of autism spectrum disorder. However, clinicians must take into account the potential for individuals with autism spectrum disorder to be concrete in their interpretation of questions regarding the key features of schizophrenia (e.g., “Do you hear voices when no one is there?” ”Yes [on the radio]”). Autism spectrum disorder and schizophrenia can co-occur, and both should be diagnosed when criteria are met.

Personality disorders. In adults without intellectual developmental disorder or significant language impairment, some behaviors associated with autism spectrum disorder may be perceived by others as symptoms of narcissistic, schizotypal, or schizoid personality disorder. Schizotypal personality disorder in particular may intersect with autism spectrum disorder in unusual preoccupations and perceptual experiences, odd thinking and

speech, constricted affect and social anxiety, lack of close friends, and odd or eccentric behavior. The early developmental course of autism spectrum disorder (lack of imaginative play, restricted/repetitive behavior, sensory sensitivities) is most helpful in differentiating it from personality disorders.

Comorbidity

Autism spectrum disorder is frequently associated with intellectual developmental disorder and language disorder (i.e., an inability to comprehend and construct sentences with proper grammar). Specific learning difficulties (literacy and numeracy) are common, as is developmental coordination disorder.

Psychiatric comorbidities also co-occur in autism spectrum disorder. About 70% of individuals with autism spectrum disorder may have one comorbid mental disorder, and 40% may have two or more comorbid mental disorders. Anxiety disorders, depression, and ADHD are particularly common. Avoidant/restrictive food intake disorder is a fairly frequent presenting feature of autism spectrum disorder, and extreme and narrow food preferences may persist.

Among individuals who are nonverbal or have language deficits, observable signs such as changes in sleep or eating and increases in challenging behavior should trigger an evaluation for anxiety or depression, as well as for potential pain or discomfort from undiagnosed medical or dental problems. Medical conditions commonly associated with autism spectrum disorder include epilepsy and constipation.

Attention-Deficit/Hyperactivity Disorder

Attention-Deficit/Hyperactivity Disorder

Diagnostic Criteria

A. A persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development, as characterized by (1) and/or (2):

1. **Inattention:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

   Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

   a. Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate).
   b. Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading).
   c. Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction).
   d. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).
   e. Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belongings in order; messy, disorganized

work; has poor time management; fails to meet deadlines).

f. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).

g. Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).

h. Is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).

i. Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

2. **Hyperactivity and impulsivity:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

   Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or a failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

   a. Often fidgets with or taps hands or feet or squirms in seat.

   b. Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the

classroom, in the office or other workplace, or in other situations that require remaining in place).

c. Often runs about or climbs in situations where it is inappropriate. (**Note:** In adolescents or adults, may be limited to feeling restless.)

d. Often unable to play or engage in leisure activities quietly.

e. Is often "on the go," acting as if "driven by a motor" (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with).

f. Often talks excessively.

g. Often blurts out an answer before a question has been completed (e.g., completes people's sentences; cannot wait for turn in conversation).

h. Often has difficulty waiting his or her turn (e.g., while waiting in line).

i. Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people's things without asking or receiving permission; for adolescents and adults, may intrude into or take over what others are doing).

B. Several inattentive or hyperactive-impulsive symptoms were present prior to age 12 years.

C. Several inattentive or hyperactive-impulsive symptoms are present in two or more settings (e.g., at home, school, or work; with friends or relatives; in other activities).

D. There is clear evidence that the symptoms interfere with, or reduce the quality of, social, academic, or occupational functioning.

E. The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better explained by another mental disorder (e.g., mood

disorder, anxiety disorder, dissociative disorder, personality disorder, substance intoxication or withdrawal).

Specify whether:

F90.2 Combined presentation: If both Criterion A1 (inattention) and Criterion A2 (hyperactivity-impulsivity) are met for the past 6 months.

F90.0 Predominantly inattentive presentation: If Criterion A1 (inattention) is met but Criterion A2 (hyperactivity-impulsivity) is not met for the past 6 months.

F90.1 Predominantly hyperactive/impulsive presentation: If Criterion A2 (hyperactivity-impulsivity) is met and Criterion A1 (inattention) is not met for the past 6 months.

Specify if:

In partial remission: When full criteria were previously met, fewer than the full criteria have been met for the past 6 months, and the symptoms still result in impairment in social, academic, or occupational functioning.

Specify current severity:

Mild: Few, if any, symptoms in excess of those required to make the diagnosis are present, and symptoms result in no more than minor impairments in social or occupational functioning.

Moderate: Symptoms or functional impairment between “mild” and “severe” are present.

Severe: Many symptoms in excess of those required to make the diagnosis, or several symptoms that are particularly severe, are present, or the symptoms result in marked impairment in social or occupational functioning.

Diagnostic Features

The essential feature of attention-deficit/hyperactivity disorder (ADHD) is a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development. *Inattention* manifests behaviorally in ADHD as wandering off task, failing to follow through on instructions or finishing work or chores, having difficulty sustaining focus, and being disorganized and is not attributable to defiance or lack of comprehension. *Hyperactivity* refers to excessive motor activity (such as a child running about) when it is not appropriate, or excessive fidgeting, tapping, or talkativeness. In adults, hyperactivity may manifest as extreme restlessness or wearing others out with their activity. *Impulsivity* refers to hasty actions that occur in the moment without forethought, which may have potential for harm to the individual (e.g., darting into the street without looking). Impulsivity may reflect a desire for immediate rewards or an inability to delay gratification. Impulsive behaviors may manifest as social intrusiveness (e.g., interrupting others excessively) and/or as making important decisions without consideration of long-term consequences (e.g., taking a job without adequate information).

ADHD begins in childhood. The requirement that several symptoms be present before age 12 years conveys the importance of a substantial clinical presentation during childhood. At the same time, an earlier age at onset is not specified because of difficulties in establishing precise childhood onset retrospectively. Adult recall of childhood symptoms tends to be unreliable, and it is beneficial to obtain ancillary information. ADHD cannot be diagnosed in the absence of any symptoms prior to age 12. When symptoms of what appears to be ADHD first occur after age 13, they are more likely to be explained by another mental disorder or to represent the cognitive effects of substance use.

Manifestations of the disorder must be present in more than one setting (e.g., home and school, or home and work). Confirmation of substantial symptoms across settings typically cannot be done accurately without consulting informants who have seen the individual in those settings. Typically, symptoms vary depending on context within a given setting. Signs of the disorder may be minimal or absent when the individual is receiving frequent rewards for appropriate behavior, is under close supervision, is in a novel setting, is engaged in especially interesting

activities, has consistent external stimulation (e.g., via electronic screens), or is interacting in one-on-one situations (e.g., the clinician's office).

Associated Features

Delays in language, motor, or social development are not specific to ADHD but often co-occur. Emotional dysregulation or emotional impulsivity commonly occurs in children and adults with ADHD. Individuals with ADHD self-report and are described by others as being quick to anger, easily frustrated, and overreactive emotionally.

Even in the absence of a specific learning disorder, academic or work performance is often impaired. Individuals with ADHD may exhibit neurocognitive deficits in a variety of areas, including working memory, set shifting, reaction time variability, response inhibition, vigilance, and planning/organization, although these tests are not sufficiently sensitive or specific to serve as diagnostic indices.

Although ADHD is not associated with specific physical features, rates of minor physical anomalies (e.g., hypertelorism, highly arched palate, low-set ears) may be elevated. Subtle motor delays and other neurological soft signs may occur. (Note that marked co-occurring clumsiness and motor delays should be coded separately [e.g., developmental coordination disorder].)

Children with neurodevelopmental disorders with a known cause (e.g., fragile X syndrome, 22q11 deletion syndrome) may often also have symptoms of inattention and impulsivity/hyperactivity; they should receive an ADHD diagnosis if their symptoms meet the full criteria for the disorder.

Prevalence

Population surveys suggest that ADHD occurs worldwide in about 7.2% of children; however, cross-national prevalence ranges widely, from 0.1% to 10.2% of children and adolescents. Prevalence is higher in special populations such as foster children or correctional settings. In a cross-national meta-analysis, ADHD occurred in 2.5% of adults.

Development and Course

Many parents first observe excessive motor activity when the child is a toddler, but symptoms are difficult to distinguish from highly variable normative behaviors before age 4 years. ADHD is most often identified during elementary school years when inattention becomes more prominent and impairing. The disorder is relatively stable through early adolescence, but some individuals have a worsened course with development of antisocial behaviors. In most individuals with ADHD, symptoms of motoric hyperactivity become less obvious in adolescence and adulthood, but difficulties with restlessness, inattention, poor planning, and impulsivity persist. A substantial proportion of children with ADHD remain relatively impaired into adulthood.

In preschool, the main manifestation is hyperactivity. Inattention becomes more prominent during elementary school. During adolescence, signs of hyperactivity (e.g., running and climbing) are less common and may be confined to fidgetiness or an inner feeling of jitteriness, restlessness, or impatience. In adulthood, along with inattention and restlessness, impulsivity may remain problematic even when hyperactivity has diminished.

Risk and Prognostic Factors

Temperamental. ADHD is associated with reduced behavioral inhibition, effortful control, or constraint; negative emotionality; and/or elevated novelty seeking. These traits may predispose some children to ADHD but are not specific to the disorder.

Environmental. Very low birth weight and degree of prematurity convey a greater risk for ADHD; the more extreme the low weight, the greater the risk. Prenatal exposure to smoking is associated with ADHD even after controlling for parental psychiatric history and socioeconomic status. A minority of cases may be related to reactions to aspects of diet. Neurotoxin exposure (e.g., lead), infections (e.g., encephalitis), and alcohol exposure in utero have been correlated with subsequent ADHD, but it is not known whether these associations are causal.

Genetic and physiological. The heritability of ADHD is approximately 74%. Large-scale genome-wide association studies (GWAS) have identified a

number of loci enriched in evolutionarily constrained genomic regions and loss-of-function genes as well as around brain-expressed regulatory regions. There is no single gene for ADHD.

Visual and hearing impairments, metabolic abnormalities, and nutritional deficiencies should be considered as possible influences on ADHD symptoms. ADHD is elevated in individuals with idiopathic epilepsy.

Course modifiers. Family interaction patterns in early childhood are unlikely to cause ADHD but may influence its course or contribute to secondary development of conduct problems.

Culture-Related Diagnostic Issues

Differences in ADHD prevalence across regions appear attributable mainly to different diagnostic procedures and methodological practices, including using different diagnostic interviews and differences in whether functional impairment was required and, if so, how it was defined. Prevalence is also affected by cultural variation in attitudes toward behavioral norms and expectations of children and youth in different social contexts, as well as cultural differences in interpretations of children's behaviors by parents and teachers, including differences by gender. Clinical identification rates in the United States for African American and Latinx populations tend to be lower than for non-Latinx White populations. Underdetection may result from mislabeling of ADHD symptoms as oppositional or disruptive in socially oppressed ethnic or racialized groups because of explicit or implicit clinician bias, leading to overdiagnosis of disruptive disorders. Higher prevalence in non-Latinx White youth may also be influenced by greater parental demand for diagnosis of behaviors seen as ADHD-related. Informant symptom ratings may be influenced by the cultural background of the child and the informant, suggesting that culturally competent diagnostic practices are relevant in assessing ADHD.

Sex- and Gender-Related Diagnostic Issues

ADHD is more frequent in males than in females in the general population, with a ratio of approximately 2:1 in children and 1.6:1 in adults. Females are more likely than males to present primarily with inattentive features. Sex differences in ADHD symptom severity may be due to differing genetic and cognitive liabilities between sexes.

Diagnostic Markers

No biological marker is diagnostic for ADHD. Although ADHD has been associated with elevated power of slow waves (4–7 Hz “theta”) as well as decreased power of fast waves (14–30 Hz “beta”), a later review found no differences in theta or beta power in either children or adults with ADHD relative to control subjects.

Although some neuroimaging studies have shown differences in children with ADHD compared with control subjects, meta-analysis of *all* neuroimaging studies do not show differences between individuals with ADHD and control subjects. This likely is due to differences in diagnostic criteria, sample size, task used, and technical aspects of the neuroimaging technique. Until these issues are resolved, no form of neuroimaging can be used for diagnosis of ADHD.

Association With Suicidal Thoughts or Behavior

ADHD is a risk factor for suicidal ideation and behavior in children. Similarly, in adulthood, ADHD is associated with an increased risk of suicide attempt, when comorbid with mood, conduct, or substance use disorders, even after controlling for comorbidity. Suicidal thoughts are also more common in ADHD populations than in non-ADHD control subjects. ADHD predicted persistence of suicidal thoughts in U.S. Army soldiers.

Functional Consequences of Attention-Deficit/Hyperactivity Disorder

ADHD is associated with reduced school performance and academic attainment. Academic deficits, school-related problems, and peer neglect tend to be most associated with elevated

symptoms of inattention, whereas peer rejection and, to a lesser extent, accidental injury are most salient with marked symptoms of hyperactivity or impulsivity. Inadequate or variable self-application to tasks that require sustained effort is often interpreted by others as laziness, irresponsibility, or failure to cooperate.

Young adults with ADHD have poor job stability. Adults with ADHD show poorer occupational performance, attainment, attendance, and higher probability of unemployment, as well as elevated interpersonal conflict. On average, individuals with ADHD obtain less schooling, have poorer vocational achievement, and have reduced intellectual scores than their peers, although there is great variability. In its severe form, the disorder is markedly impairing, affecting social, familial, and scholastic/occupational adjustment.

Family relationships may be characterized by discord and negative interactions. Individuals with ADHD have lower self-esteem relative to peers without ADHD. Peer relationships are often disrupted by peer rejection, neglect, or teasing of the individual with ADHD.

Children with ADHD are significantly more likely than their peers without ADHD to develop conduct disorder in adolescence and antisocial personality disorder in adulthood, consequently increasing the likelihood for substance use disorders and incarceration. The risk of subsequent substance use disorders is elevated, especially when conduct disorder or antisocial personality disorder develops.

Individuals with ADHD are more likely than peers to be injured. Children and adults with ADHD are at higher risk for suffering trauma and developing subsequent posttraumatic stress syndrome. Traffic accidents and violations are more frequent in drivers with ADHD. Individuals with ADHD have a higher overall mortality rate, largely because of accidents and injuries. There may also be an elevated likelihood of obesity and hypertension among individuals with ADHD.

Differential Diagnosis

Oppositional defiant disorder. Individuals with oppositional defiant disorder may resist work or school tasks that require self-application because they resist conforming to others' demands. Their behavior is characterized by

negativity, hostility, and defiance. These symptoms must be differentiated from aversion to school or mentally demanding tasks because of difficulty in sustaining mental effort, forgetting instructions, and impulsivity in individuals with ADHD. Complicating the differential diagnosis is the fact that some individuals with ADHD may develop secondary oppositional attitudes toward such tasks and devalue their importance.

Intermittent explosive disorder. ADHD and intermittent explosive disorder share high levels of impulsive behavior. However, individuals with intermittent explosive disorder show serious aggression toward others, which is not characteristic of ADHD, and they do not experience problems with sustaining attention as seen in ADHD. In addition, intermittent explosive disorder is rare in childhood. Intermittent explosive disorder may be diagnosed in the presence of ADHD.

Other neurodevelopmental disorders. The increased motoric activity that may occur in ADHD must be distinguished from the repetitive motor behavior that characterizes stereotypic movement disorder and some cases of autism spectrum disorder. In stereotypic movement disorder, the motoric behavior is generally fixed and repetitive (e.g., body rocking, self-biting), whereas the fidgetiness and restlessness in ADHD are typically generalized and not characterized by repetitive stereotypic movements. In Tourette's disorder, frequent multiple tics can be mistaken for the generalized fidgetiness of ADHD. Prolonged observation may be needed to differentiate fidgetiness from bouts of multiple tics.

Specific learning disorder. Children with specific learning disorder alone may appear inattentive because of frustration, lack of interest, or limited ability in neurocognitive

processes, including working memory and processing speed, whereas their inattention is much reduced when performing a skill that does not require the impaired cognitive process.

Intellectual developmental disorder (intellectual disability). Symptoms of ADHD are common in children with intellectual developmental disorder placed in academic settings that are inappropriate to their intellectual ability. In such

cases, the symptoms are not evident during nonacademic tasks. A diagnosis of ADHD in intellectual developmental disorder requires that inattention or hyperactivity be excessive for mental age.

Autism spectrum disorder. Individuals with ADHD and those with autism spectrum disorder exhibit inattention, social dysfunction, and difficult-to-manage behavior. The social dysfunction and peer rejection seen in individuals with ADHD must be distinguished from the social disengagement, isolation, and indifference to facial and tonal communication cues seen in individuals with autism spectrum disorder. Children with autism spectrum disorder may display tantrums because of an inability to tolerate a change from their expected course of events. In contrast, children with ADHD may misbehave or have a tantrum during a major transition because of impulsivity or poor self-control.

Reactive attachment disorder. Children with reactive attachment disorder may show social disinhibition, but not the full ADHD symptom cluster, and display other features such as a lack of enduring relationships that are not characteristic of ADHD.

Anxiety disorders. ADHD shares symptoms of inattention with anxiety disorders. Individuals with ADHD are inattentive because of their preferential engagement with novel and stimulating activities or preoccupation with enjoyable activities. This is distinguished from the inattention attributable to worry and rumination seen in anxiety disorders. Restlessness might be seen in anxiety disorders. However, in ADHD, the symptom is not associated with worry and rumination.

Posttraumatic stress disorder. Concentration difficulties associated with posttraumatic stress disorder (PTSD) may be misdiagnosed in children as ADHD. Children younger than 6 years often manifest PTSD in nonspecific symptoms such as restlessness, irritability, inattention, and poor concentration, which can mimic ADHD. Parents may also minimize their children’s trauma-related symptoms, and teachers and other caregivers are often unaware of the child’s exposure to traumatic events. A comprehensive assessment of past exposure to traumatic events can rule out PTSD.

Depressive disorders. Individuals with depressive disorders may present with inability to concentrate. However, poor concentration in mood

disorders becomes prominent only during a depressive episode.

Bipolar disorder. Individuals with bipolar disorder may have increased activity, poor concentration, and increased impulsivity, but these features are episodic, unlike ADHD, in which the symptoms are persistent. Moreover, in bipolar disorder, increased impulsivity or inattention is accompanied by elevated mood, grandiosity, and other specific bipolar features. Children with ADHD may show significant changes in mood within the same day; such lability is distinct from a manic or hypomanic episode, which must last 4 or more days to be a clinical indicator of bipolar disorder, even in children. Bipolar disorder is rare in preadolescents, even when severe irritability and anger are prominent, whereas ADHD is common among children and adolescents who display excessive anger and irritability.

Disruptive mood dysregulation disorder. Disruptive mood dysregulation disorder is characterized by pervasive irritability, and intolerance of frustration, but impulsiveness and disorganized attention are not essential features. However, most children and adolescents with the disorder also have symptoms that meet criteria for ADHD, which is diagnosed separately.

Substance use disorders. Differentiating ADHD from substance use disorders may be problematic if the first presentation of ADHD symptoms follows the onset of abuse or

frequent use. Clear evidence of ADHD before substance misuse from informants or previous records may be essential for differential diagnosis.

Personality disorders. In adolescents and adults, it may be difficult to distinguish ADHD from borderline, narcissistic, and other personality disorders. Some personality disorders tend to share the features of disorganization, social intrusiveness, emotional dysregulation, and cognitive dysregulation. However, ADHD is not characterized by fear of abandonment, self-injury, extreme ambivalence, or other features of personality disorder. It may take extended clinical observation, informant interview, or detailed history to distinguish impulsive, socially intrusive, or

inappropriate behavior from narcissistic, aggressive, or domineering behavior to make this differential diagnosis.

Psychotic disorders. ADHD is not diagnosed if the symptoms of inattention and hyperactivity occur exclusively during the course of a psychotic disorder.

Medication-induced symptoms of ADHD. Symptoms of inattention, hyperactivity, or impulsivity attributable to the use of medication (e.g., bronchodilators, isoniazid, neuroleptics [resulting in akathisia], thyroid replacement medication) are diagnosed as other specified or unspecified other (or unknown) substance–related disorders.

Neurocognitive disorders. While impairment in complex attention may be one of the affected cognitive domains in a neurocognitive disorder, it must represent a decline from a previous level of performance in order to justify a diagnosis of major or mild neurocognitive disorder. Moreover, major or mild neurocognitive disorder typically has its onset in adulthood. In contrast, the inattention in ADHD must have been present prior to age 12 and does not represent a decline from previous functioning.

Comorbidity

Although ADHD is more common in males, females with ADHD have higher rates of a number of comorbid disorders, particularly oppositional defiant disorder, autism spectrum disorder, and personality and substance use disorders. Oppositional defiant disorder co-occurs with ADHD in approximately half of children with the combined presentation and about a quarter with the predominantly inattentive presentation. Conduct disorder co-occurs in about a quarter of children or adolescents with the combined presentation, depending on age and setting. Most children and adolescents with disruptive mood dysregulation disorder have symptoms that also meet criteria for ADHD; a lesser percentage of children with ADHD have symptoms that meet criteria for disruptive mood dysregulation disorder. Anxiety disorders, major depressive disorder, obsessive-compulsive disorder, and intermittent explosive disorder occur in a minority of individuals with ADHD but more often than in the general population. Although substance use disorders are relatively more frequent among adults with ADHD in the general population, the disorders are present in only a

minority of adults with ADHD. In adults, antisocial and other personality disorders may co-occur with ADHD.

ADHD may co-occur in variable symptom profiles with other neurodevelopmental disorders, including specific learning disorder, autism spectrum disorder, intellectual developmental disorder, language disorders, developmental coordination disorder, and tic disorders.

Comorbid sleep disorders in ADHD are associated with daytime impairments in cognition (e.g., inattention). Many individuals with ADHD report daytime sleepiness that may meet criteria for hypersomnolence disorder. One quarter to one-half of individuals with ADHD report sleep difficulties; studies have shown an association of ADHD with insomnia, circadian rhythm sleep-wake disorder, sleep-disordered breathing, and restless legs syndrome.

Individuals with ADHD have been found to have elevated rates of a number of medical conditions, particularly allergy and autoimmune disorders, as well as epilepsy.

Other Specified Attention-Deficit/Hyperactivity Disorder

F90.8

This category applies to presentations in which symptoms characteristic of attention-deficit/hyperactivity disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for attention-deficit/hyperactivity disorder or any of the disorders in the neurodevelopmental disorders diagnostic class. The other specified attention-deficit/hyperactivity disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for attention-

deficit/hyperactivity disorder or any specific neurodevelopmental disorder. This is done by recording “other specified attention-deficit/hyperactivity disorder” followed by the specific reason (e.g., “with insufficient inattention symptoms”).

Unspecified Attention-Deficit/Hyperactivity Disorder

F90.9

This category applies to presentations in which symptoms characteristic of attention-deficit/hyperactivity disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for attention-deficit/hyperactivity disorder or any of the disorders in the neurodevelopmental disorders diagnostic class. The unspecified attention-deficit/hyperactivity disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for attention-deficit/hyperactivity disorder or for a specific neurodevelopmental disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Specific Learning Disorder

Specific Learning Disorder

Diagnostic Criteria

A. Difficulties learning and using academic skills, as indicated by the presence of at least one of the following symptoms that have persisted for at least 6 months, despite the provision of interventions that target those difficulties:

   1. Inaccurate or slow and effortful word reading (e.g., reads single words aloud incorrectly or slowly and hesitantly, frequently guesses words, has difficulty sounding out words).
   2. Difficulty understanding the meaning of what is read (e.g., may read text accurately but not understand the sequence, relationships, inferences, or deeper meanings of what is read).
   3. Difficulties with spelling (e.g., may add, omit, or substitute vowels or consonants).

   4. Difficulties with written expression (e.g., makes multiple grammatical or punctuation errors within sentences; employs poor paragraph organization; written expression of ideas lacks clarity).
   5. Difficulties mastering number sense, number facts, or calculation (e.g., has poor understanding of numbers, their magnitude, and relationships; counts on fingers to add single-digit numbers instead of recalling the math fact as peers do; gets lost in the midst of arithmetic computation and may switch procedures).
   6. Difficulties with mathematical reasoning (e.g., has severe difficulty applying mathematical concepts, facts, or procedures to solve quantitative problems).

B. The affected academic skills are substantially and quantifiably below those expected for the individual's chronological age, and cause significant interference with academic or occupational performance, or with activities of daily living, as

confirmed by individually administered standardized achievement measures and comprehensive clinical assessment. For individuals age 17 years and older, a documented history of impairing learning difficulties may be substituted for the standardized assessment.

C. The learning difficulties begin during school-age years but may not become fully manifest until the demands for those affected academic skills exceed the individual's limited capacities (e.g., as in timed tests, reading or writing lengthy complex reports for a tight deadline, excessively heavy academic loads).

D. The learning difficulties are not better accounted for by intellectual disabilities, uncorrected visual or auditory acuity, other mental or neurological disorders, psychosocial adversity, lack of proficiency in the language of academic instruction, or inadequate educational instruction.

Note: The four diagnostic criteria are to be met based on a clinical synthesis of the individual's history (developmental, medical, family, educational), school reports, and psychoeducational assessment.

Coding note: Specify all academic domains and subskills that are impaired. When more than one domain is impaired, each one should be coded individually according to the following specifiers.

Specify if:

F81.0 With impairment in reading:

Word reading accuracy

Reading rate or fluency

Reading comprehension

Note: *Dyslexia* is an alternative term used to refer to a pattern of learning difficulties characterized by problems with accurate or fluent word recognition, poor decoding,

and poor spelling abilities. If dyslexia is used to specify this particular pattern of difficulties, it is important also to specify any additional difficulties that are present, such as difficulties with reading comprehension or math reasoning.

F81.81 With impairment in written expression:

Spelling accuracy

Grammar and punctuation accuracy

Clarity or organization of written expression

F81.2 With impairment in mathematics:

Number sense

Memorization of arithmetic facts

Accurate or fluent calculation

Accurate math reasoning

Note: *Dyscalculia* is an alternative term used to refer to a pattern of difficulties characterized by problems processing numerical information, learning arithmetic facts, and performing accurate or fluent calculations. If dyscalculia is used to specify this particular pattern of mathematic difficulties, it is important also to specify any additional difficulties that are present, such as difficulties with math reasoning or word reasoning accuracy.

Specify current severity:

Mild: Some difficulties learning skills in one or two academic domains, but of mild enough severity that the individual may be able to compensate or function well when provided with appropriate accommodations or support services, especially during the school years.

Moderate: Marked difficulties learning skills in one or more academic domains, so that the individual is unlikely to become proficient without some intervals of intensive and specialized teaching during the school years. Some accommodations or supportive services at least part of the

day at school, in the workplace, or at home may be needed to complete activities accurately and efficiently.

Severe: Severe difficulties learning skills, affecting several academic domains, so that the individual is unlikely to learn those skills without ongoing intensive individualized and specialized teaching for most of the school years. Even with an array of appropriate accommodations or services at home, at school, or in the workplace, the individual may not be able to complete all activities efficiently.

Recording Procedures

Each impaired academic domain and subskill of specific learning disorder should be recorded. Because of ICD coding requirements, impairments in reading, impairments in written expression, and impairments in mathematics, with their corresponding impairments in subskills, must be coded and recorded separately. For example, impairments in reading and mathematics and impairments in the subskills of reading rate or fluency, reading comprehension, accurate or fluent calculation, and accurate math reasoning would be coded and recorded as F81.0 specific learning disorder with impairment in reading, with impairment in reading rate or fluency, and impairment in reading comprehension; F81.2 specific learning disorder with impairment in mathematics, with impairment in accurate or fluent calculation and impairment in accurate math reasoning.

Diagnostic Features

Specific learning disorder is a neurodevelopmental disorder with a biological origin that is the basis for abnormalities at a cognitive level that are associated with the behavioral signs of the disorder. The biological origin includes an interaction of genetic, epigenetic, and environmental factors, which affect the brain's ability to perceive or process verbal or nonverbal information efficiently and accurately.

One essential feature of specific learning disorder is persistent difficulties learning keystone academic skills (Criterion A), with onset during the years of formal schooling (i.e., the developmental period). Key

academic skills include reading of single words accurately and fluently, reading comprehension, written expression and spelling, arithmetic calculation, and mathematical reasoning (solving mathematical problems). In contrast to talking or walking, which are acquired developmental milestones that emerge with brain maturation, academic skills (e.g., reading, spelling, writing, mathematics) have to be taught and learned explicitly. Specific learning disorder disrupts the normal pattern of learning academic skills; it is not simply a consequence of lack of opportunity of learning or inadequate instruction. Difficulties mastering these key academic skills may also impede learning in other academic subjects (e.g., history, science, social studies), but those problems are attributable to difficulties learning the underlying academic skills.

Difficulties learning to map letters with the sounds of one's language—to read printed words (often called *dyslexia* [specific learning disorder with impairment in reading])—is one of the most common manifestations of specific learning disorder. The learning difficulties manifest as a range of observable, descriptive behaviors or symptoms (as listed in Criteria A1–A6). These clinical symptoms may be observed, probed by means of the clinical interview, or ascertained from school reports, rating scales, or descriptions in previous educational or psychological assessments. The learning difficulties are persistent, not transitory. In children and adolescents, *persistence* is defined as restricted progress in learning (i.e., no evidence that the individual is catching up with classmates) for at least 6 months despite the provision of extra help at home or school. For example, difficulties learning to read single words that do not fully or rapidly remit with the provision of instruction in phonological skills or word identification strategies may indicate a specific learning disorder. Evidence of persistent learning difficulties may be derived from cumulative school reports, portfolios of the child's evaluated work, curriculum-based measures, or clinical interview. In adults, persistent difficulty refers to ongoing difficulties in literacy or numeracy skills that manifest during childhood or adolescence, as indicated by cumulative evidence from school reports, evaluated portfolios of work, or previous assessments.

A second key feature is that the individual's performance of the affected academic skills is well below expected for age (Criterion B). One robust clinical indicator of difficulties learning academic skills is low academic achievement for age or average achievement that is sustainable only by extraordinarily high levels of effort or support. In children, the low academic skills cause significant interference in school performance (as indicated by school reports and teacher's grades or ratings). Another clinical indicator, particularly in adults, is avoidance of activities that require the academic skills. Also in adulthood, low academic skills interfere with occupational performance or everyday activities requiring those skills (as indicated by self-report or report by others). However, this criterion also requires psychometric evidence from an individually administered, psychometrically sound and culturally appropriate test of academic achievement that is norm-referenced or criterion-referenced. Academic skills are distributed along a continuum, so there is no natural cutpoint that can be used to differentiate individuals with and without specific learning disorder. Thus, any threshold used to specify what constitutes significantly low academic achievement (e.g., academic skills well below age expectation) is to a large extent arbitrary. Low achievement scores on one or more standardized tests or subtests within an academic domain (i.e., at least 1.5 standard deviations [SD] below the population mean for age, which translates to a standard score of 78 or less, which is below the 7th percentile) are needed for the greatest diagnostic certainty. However, precise scores will vary according to the particular standardized tests that are used. On the basis of clinical judgment, a more lenient threshold may be used (e.g., 1.0 SD below the population mean for age), when learning difficulties are supported by converging evidence from clinical assessment, academic history, school reports, or test scores. Moreover, since standardized tests are not available in all languages, the diagnosis may then be based in part on clinical judgment of scores on available test measures.

A third core feature is that the learning difficulties are readily apparent in the early school years in most individuals (Criterion C). However, in others, the learning difficulties may not manifest fully until later school years, by which time learning demands have increased and exceed the individual's limited capacities.

Another key diagnostic feature is that the learning difficulties are considered “specific” for four reasons. First, they are not better explained by intellectual developmental disorders (intellectual developmental disorder [intellectual disability]; global developmental delay); hearing or vision disorders; or neurological or motor disorders (Criterion D). Specific learning disorder affects learning in individuals who otherwise demonstrate normal levels of intellectual functioning (generally estimated by an IQ score of greater than about 70 [± 5 points allowing for measurement error]). The phrase “unexpected academic

underachievement” is often cited as the defining characteristic of specific learning disorder in that the specific learning disabilities are not part of a more general learning difficulty as manifested in intellectual developmental disorder or global developmental delay.

Second, the learning difficulty cannot be attributed to more general external factors, such as economic or environmental disadvantage, chronic absenteeism, or lack of education as typically provided in the individual’s community context. Third, the learning difficulty cannot be attributed to neurological (e.g., pediatric stroke) or motor disorders or to vision or hearing disorders, which are often associated with problems learning academic skills but are distinguishable by presence of neurological signs. Finally, the learning difficulty may be restricted to one academic skill or domain (e.g., reading single words, retrieving or calculating number facts).

Specific learning disorder may also occur in individuals identified as intellectually “gifted.” These individuals may be able to sustain apparently adequate academic functioning by using compensatory strategies, extraordinarily high effort, or support, until the learning demands or assessment procedures (e.g., timed tests) pose barriers to their demonstrating their learning or accomplishing required tasks. In these cases, the individual’s achievement scores will be low relative to ability level or achievement in other domains, rather than to the population mean for achievement.

Comprehensive assessment is required. Specific learning disorder can only be diagnosed after formal education starts but can be diagnosed at any

point afterward in children, adolescents, or adults, providing there is evidence of onset during the years of formal schooling (i.e., the developmental period). No single data source is sufficient for a diagnosis of specific learning disorder. Rather, specific learning disorder is a clinical diagnosis based on a synthesis of the individual's medical, developmental, educational, and family history; the history of the learning difficulty, including its previous and current manifestation; the impact of the difficulty on academic, occupational, or social functioning; previous or current school reports; portfolios of work requiring academic skills; curriculum-based assessments; and previous or current scores from individual standardized tests of academic achievement. If an intellectual, sensory, neurological, or motor disorder is suspected, then the clinical assessment for specific learning disorder should also include methods appropriate for these disorders. Thus, comprehensive assessment will involve professionals with expertise in specific learning disorder and psychological/cognitive assessment. Since specific learning disorder typically persists into adulthood, reassessment is rarely necessary, unless indicated by marked changes in the learning difficulties (amelioration or worsening) or requested for specific purposes.

Associated Features

The symptoms of specific learning disorder (difficulty with aspects of reading, writing, or mathematics) frequently co-occur. An uneven profile of abilities is common, such as a combination of above-average abilities in drawing, design, and other visuospatial abilities, and slow, effortful, and inaccurate reading and poor reading comprehension and written expression. Specific learning disorder is frequently but not invariably preceded, in preschool years, by delays in attention, language, or motor skills that may persist and co-occur with specific learning disorder.

Individuals with specific learning disorder typically (but not invariably) exhibit poor performance on psychological tests of cognitive processing. However, it remains unclear whether these cognitive abnormalities are the cause, correlate, or consequence of the learning difficulties. Cognitive deficits associated with difficulties learning to read words are well documented, and there is a burgeoning understanding of the cognitive

deficits associated with difficulty acquiring mathematics skills, but cognitive deficits associated with other manifestations of specific learning disorder (e.g., reading comprehension, written expression) are underspecified.

Although individual cognitive deficits particularly contribute to each specific learning disorder symptom, some cognitive deficits are shared across different specific learning disorder subtypes (e.g., processing speed) and may contribute to co-occurring symptoms of specific learning disorder. The co-occurring nature of the symptoms of specific learning disorder and the shared cognitive deficits across the specific learning disorder subtypes suggest shared underlying biological mechanisms.

Thus, individuals with similar behavioral symptoms or test scores are found to have a variety of cognitive deficits, and many of these processing deficits are also found in other neurodevelopmental disorders (e.g., attention-deficit/hyperactivity disorder [ADHD], autistic spectrum disorder, communication disorders, developmental coordination disorder).

As a group, individuals with the disorder show circumscribed alterations in cognitive processing and brain structure and function. Genetic differences are also evident at the group level. However, cognitive testing, neuroimaging, or genetic testing are not useful for diagnosis at this time, and assessment of cognitive processing deficits is not required for diagnostic assessment.

Prevalence

The prevalence of specific learning disorder across the academic domains of reading, writing, and mathematics is 5%–15% among school-age children in Brazil, Northern Ireland, and the United States. Prevalence in adults is unknown.

Development and Course

Onset, recognition, and diagnosis of specific learning disorder usually occur during the elementary school years when children are required to learn to read, spell, write, and learn mathematics. However, precursors such as

language delays or deficits, difficulties in rhyming or counting, or difficulties with fine motor skills required for writing commonly occur in early childhood before the start of formal schooling.

Manifestations may be behavioral (e.g., a reluctance to engage in learning; oppositional behavior). Specific learning disorder is lifelong, but the course and clinical expression are variable, in part depending on the interactions among the task demands of the environment, the range and severity of the individual's learning difficulties, the individual's learning abilities, comorbidity, and the available support systems and intervention. Nonetheless, problems with reading fluency and comprehension, spelling, written expression, and numeracy skills in everyday life typically persist into adulthood.

Changes in manifestation of symptoms occur with age, so that an individual may have a persistent or shifting array of learning difficulties across the lifespan. Adults with specific learning disorder appear to experience limitations and restrictions in activity and participation in domains of communication, interpersonal interactions and community, and social and civic life.

Examples of symptoms that may be observed among preschool-age children include a lack of interest in playing games with language sounds (e.g., repetition, rhyming), and they may have trouble learning nursery rhymes. Preschool children with specific learning disorder may frequently use baby talk, mispronounce words, and have trouble remembering names of letters, numbers, or days of the week. They may fail to recognize letters in their own names and have trouble learning to count. Kindergarten-age children with specific learning disorder may be unable to recognize and write letters, may be unable to write their own names, or may have persistent use of invented spelling beyond developmentally typical time frames.

They may have trouble breaking down spoken words into syllables (e.g., "cowboy" into "cow" and "boy") and trouble recognizing words that rhyme (e.g., cat, bat, hat). Kindergarten-age children also may have trouble connecting letters with their sounds (e.g., letter *b* makes the sound /b/) and may be unable to recognize phonemes (e.g., do not know which in a set of words [e.g., dog, man, car] starts with the same sound as "cat").

Specific learning disorder in elementary school–age children typically manifests as marked difficulty learning letter-sound correspondence (particularly in English-speaking children),

fluent word decoding, spelling, or math facts; reading aloud is slow, inaccurate, and effortful, and some children struggle to understand the magnitude that a spoken or written number represents. Children in primary grades (grades 1–3) may continue to have problems recognizing and manipulating phonemes, be unable to read common one-syllable words (such as mat or top), and be unable recognize common irregularly spelled words (e.g., said, two). They may commit reading errors that indicate problems in connecting sounds and letters (e.g., “big” for “got”) and have difficulty sequencing numbers and letters. Children in grades 1–3 also may have difficulty remembering number facts or arithmetic procedures for adding, subtracting, and so forth, and may complain that reading or arithmetic is hard and avoid doing it. Children with specific learning disorder in the middle grades (grades 4–6) may mispronounce or skip parts of long, multisyllable words (e.g., say “conible” for “convertible,” “aminal” for “animal”) and confuse words that sound alike (e.g., “tornado” for “volcano”). They may have trouble remembering dates, names, and telephone numbers and may have trouble completing homework or tests on time. Children in the middle grades also may have poor comprehension with or without slow, effortful, and inaccurate reading, and they may have trouble reading small function words (e.g., that, the, an, in). They may have very poor spelling and poor written work. They may get the first part of a word correctly, then guess wildly (e.g., read “clover” as “clock”), and may express fear of reading aloud or refuse to read aloud.

By contrast, adolescents may have mastered word decoding, but reading remains slow and effortful, and they are likely to show marked problems in reading comprehension and written expression (including poor spelling) and poor mastery of math facts or mathematical problem solving. During adolescence and into adulthood, individuals with specific learning disorder may continue to make numerous spelling mistakes and read single words and connected text slowly and with much effort, with trouble pronouncing

multisyllable words. They may frequently need to reread material to understand or get the main point and have trouble making inferences from written text. Adolescents and adults may avoid activities that demand reading or arithmetic (reading for pleasure, reading instructions). Adults with specific learning disorder have ongoing spelling problems, slow and effortful reading, or problems making important inferences from numerical information in work-related written documents. They may avoid both leisure and work-related activities that demand reading or writing or use alternative approaches to access print (e.g., text-to-speech/speech-to-text software, audiobooks, audiovisual media).

An alternative clinical expression is that of circumscribed learning difficulties that persist across the lifespan, such as an inability to master the basic sense of number (e.g., to know which of a pair of numbers or dots represents the larger magnitude), or lack of proficiency in word identification or spelling. Avoidance of or reluctance to engage in activities requiring academic skills is common in children, adolescents, and adults. Individuals with poor reading and math skills are more likely to report socioemotional distress (e.g., sadness, loneliness) as they advance across elementary grade levels.

Episodes of severe anxiety or anxiety disorders, including somatic complaints or panic attacks, are common across the lifespan and accompany both the circumscribed and the broader expression of learning difficulties.

Risk and Prognostic Factors

Environmental. Environmental factors, including socioeconomic conditions (e.g., low socioeconomic status) and exposure to neurotoxicants, increase the risk for specific learning disorder or difficulties in reading and mathematics. Risks for specific learning disorder or difficulties in reading and mathematics include prenatal or early-life exposure to any of the following: air pollution, nicotine, polybrominated diphenyl ethers or polychlorinated biphenyls (flame retardants), lead, or manganese.

Genetic and physiological. Specific learning disorder appears to aggregate in families, particularly when affecting reading, mathematics, and spelling. The relative risk of specific

learning disorder in reading or mathematics is substantially higher (e.g., 4–8 times and 5–10 times higher, respectively) in first-degree relatives of individuals with these learning difficulties compared with those without them. Notably, rates vary depending on method of ascertainment (objective testing or self-report) of parent diagnostic status. Family history of reading difficulties (dyslexia) and parental literacy skills predict literacy problems or specific learning disorder in offspring, indicating the combined role of genetic and environmental factors.

There is high heritability for both reading ability and reading disability in alphabetic and nonalphabetic languages, including high heritability for most manifestations of learning abilities and disabilities (e.g., heritability estimate values greater than 0.6). Covariation between various manifestations of learning difficulties is high, suggesting that genes related to one presentation are highly correlated with genes related to another manifestation.

Preterm delivery or very low birthweight is a risk for specific learning disorder. In individuals with neurofibromatosis type 1, risk of specific learning disorder is high, with up to 75% of individuals demonstrating a learning disorder.

Course modifiers. Marked problems with inattentive, internalizing, and externalizing behaviors in preschool years are predictive of later difficulties in reading and mathematics (but not necessarily specific learning disorder) and nonresponse to effective academic interventions. Language impairment in preschool years is strongly associated with later impairment in reading (e.g., word reading, reading comprehension). For example, delay or disorders in speech or language, or impaired cognitive processing (e.g., phonological awareness, working memory, rapid serial naming), may predict later specific learning disorder in reading and in written expression. Additionally, a diagnosis of ADHD in childhood is associated with underachievement in reading and math in adulthood. Comorbidity with ADHD is predictive of worse mental health outcome than that associated with specific learning disorder without ADHD. Systematic, intensive, individualized instruction, using evidence-based interventions, may

improve or ameliorate the learning difficulties in some individuals or promote the use of compensatory strategies in others, thereby mitigating the otherwise poor outcomes.

Culture-Related Diagnostic Issues

Specific learning disorder occurs across linguistic and ethnoracial backgrounds and across cultural and socioeconomic contexts but may vary in its manifestation according to the nature of the spoken and written symbol systems and cultural and educational practices. For example, the cognitive processing requirements of reading and of working with numbers vary greatly across orthographies. In the English language, the observable hallmark clinical symptom of difficulties learning to read is inaccurate and slow reading of single words; in other alphabetic languages that have more direct mapping between sounds and letters (e.g., Spanish, German) and in nonalphabetic languages (e.g., Chinese, Japanese), the hallmark feature is slow but accurate reading. In English-language learners, assessment should include consideration of whether the source of reading difficulties is a limited proficiency with English or a specific learning disorder. Risk factors for specific learning disorder in English-language learners include a family history of specific learning disorder or language delay in the native language, as well as learning difficulties and deficits in phonological memory in English and failure to catch up with peers. If there is suspicion of cultural or language differences (e.g., that an English-language learner is influenced by limited English proficiency), the assessment needs to take into account the individual’s language proficiency in his or her first or native language as well as in the second language (in this example, English). Importantly, children who speak a language at home that differs phonologically from the language of academic instruction are not more likely to have phonological deficits than their peers who speak the same language at home and at school. Comorbid reading difficulties may vary with different languages; for example, reading difficulties are less frequent among Chinese-reading children with developmental coordination disorder in Taiwan compared

with children in English-speaking countries, possibly because of the characteristics of the two written languages (logographic vs. alphabetic). Considerations in assessment may include the linguistic and cultural context in which the individual is living, as well as his or her educational and learning history in the original linguistic and cultural context. Risk factors for learning problems among refugee and migrant children include teacher stereotyping and low expectations, bullying, ethnic and racialized discrimination, parental misunderstandings about educational styles and expectations, trauma, and postmigration stressors.

Sex- and Gender-Related Diagnostic Issues

Specific learning disorder is more common in males than in females (ratios range from about 2:1 to 3:1) and cannot be attributed to factors such as ascertainment bias, definitional or measurement variation, language, ethnoracial background, or socioeconomic status. Sex differences in dyslexia (specific learning disorder with impairment in reading) may be partially mediated by processing speed.

Association With Suicidal Thoughts or Behavior

In U.S. adolescents age 15 years in public school, poor reading ability was associated with suicidal thoughts and behavior compared with adolescents with typical reading scores, even when controlling for sociodemographic and psychiatric variables. In a population-based study of adults in Canada, prevalence of lifetime suicide attempts among those with specific learning disorder was higher than that among those without a specific learning disorder, even after adjustment for childhood adversities, history of mental illness and substance use, and sociodemographic factors. Among those with specific learning disorder, a history of witnessing chronic parental domestic violence and ever having had a major depressive disorder were associated with increased risk for suicidal behavior.

Functional Consequences of Specific Learning Disorder

Specific learning disorder can have negative functional consequences across the lifespan, including lower academic attainment, higher rates of high school dropout, lower rates of postsecondary education, high levels of

psychological distress and poorer overall mental health, higher rates of unemployment and underemployment, and lower incomes. School dropout and co-occurring depressive symptoms increase the risk for poor mental health outcomes, including suicidal thoughts or behavior, whereas high levels of social or emotional support predict better mental health outcomes.

Differential Diagnosis

Normal variations in academic attainment. Specific learning disorder is distinguished from normal variations in academic attainment attributable to external factors (e.g., lack of educational opportunity, consistently poor instruction, learning in a second language), because the learning difficulties persist in the presence of adequate educational opportunity and exposure to the same instruction as the peer group, and competency in the language of instruction, even when it is different from one's primary spoken language.

Intellectual developmental disorder (intellectual disability). Specific learning disorder differs from general learning difficulties associated with intellectual developmental disorder, because the learning difficulties occur in the presence of normal levels of intellectual functioning (i.e., IQ score of at least 70 ± 5). If intellectual developmental disorder is present, specific learning disorder can be diagnosed only when the learning difficulties are in excess of those usually associated with the intellectual developmental disorder.

Learning difficulties due to neurological or sensory disorders. Specific learning disorder is distinguished from learning difficulties due to neurological or sensory disorders

(e.g., pediatric stroke, traumatic brain injury, hearing impairment, vision impairment), because in these cases there are abnormal findings on neurological examination.

Neurocognitive disorders. Specific learning disorder is distinguished from learning problems associated with neurodegenerative cognitive disorders. In specific learning disorder, the clinical expression of specific learning difficulties occurs during the developmental period, which sometimes only

becomes evident when learning demands have increased and exceed the individual's limited capacities (as may occur in adulthood), and the difficulties do not manifest as a marked decline from a former state.

Attention-deficit/hyperactivity disorder. Specific learning disorder is distinguished from the poor academic performance associated with ADHD, because in the latter condition the problems may not necessarily reflect specific difficulties in learning academic skills but rather may reflect difficulties in performing those skills. However, the co-occurrence of specific learning disorder and ADHD is more frequent than expected by chance. If criteria for both disorders are met, both diagnoses can be given.

Psychotic disorders. Specific learning disorder is distinguished from the cognitive-processing difficulties associated with schizophrenia or other psychotic disorders, because with these disorders there is a decline (often rapid) in these functional domains. However, deficits in reading ability are more severe in specific learning disorder than what would be predicted by the general cognitive impairments associated with schizophrenia. If criteria for both disorders are met, both diagnoses can be given.

Comorbidity

The different types of specific learning disorder commonly co-occur with one another (e.g., specific learning disorder with impairment in mathematics and with impairment in reading) and with other neurodevelopmental disorders (e.g., ADHD, communication disorders, developmental coordination disorder, autism spectrum disorder) or other mental disorders (e.g., anxiety and depressive disorders) or behavioral problems. Notably, estimates of the comorbidity of math and reading difficulties vary depending on the tests used to define the math difficulty, likely because the same symptom (e.g., arithmetic problems) can be associated with different cognitive deficits (e.g., a deficit in language skills or a deficit in number processing). These comorbidities do not necessarily exclude the diagnosis of specific learning disorder but may make testing and differential diagnosis more difficult, because each of the co-occurring disorders independently interferes with the execution of activities of daily living, including learning. Thus, clinical judgment is required to attribute such impairment to learning difficulties. If there is an indication that

another diagnosis could account for the difficulties learning keystone academic skills described in Criterion A, specific learning disorder should not be diagnosed.

Motor Disorders

Developmental Coordination Disorder

Diagnostic Criteria F82

A. The acquisition and execution of coordinated motor skills is substantially below that expected given the individual's chronological age and opportunity for skill learning and use. Difficulties are manifested as clumsiness (e.g., dropping or bumping into

objects) as well as slowness and inaccuracy of performance of motor skills (e.g., catching an object, using scissors or cutlery, handwriting, riding a bike, or participating in sports).

B. The motor skills deficit in Criterion A significantly and persistently interferes with activities of daily living appropriate to chronological age (e.g., self-care and self-maintenance) and impacts academic/school productivity, prevocational and vocational activities, leisure, and play.

C. Onset of symptoms is in the early developmental period.

D. The motor skills deficits are not better explained by intellectual developmental disorder (intellectual disability) or visual impairment and are not attributable to a neurological condition affecting movement (e.g., cerebral palsy, muscular dystrophy, degenerative disorder).

Diagnostic Features

The diagnosis of developmental coordination disorder is made by a clinical synthesis of the history (developmental and medical), physical examination, school or workplace report, and individual assessment using psychometrically sound and culturally appropriate standardized tests. The manifestation of impaired skills requiring motor coordination (Criterion A) varies with age. Young children may be delayed in achieving motor milestones (i.e., sitting, crawling, walking), although many achieve typical motor milestones. They also may be delayed in developing skills such as negotiating stairs, pedaling, buttoning shirts, completing puzzles, and using zippers. Even when the skill is achieved, movement execution may appear awkward, slow, or less precise than that of peers. Older children and adults may display slow speed or inaccuracy with motor aspects of activities such as assembling puzzles, building models, playing ball games (especially in teams), handwriting, typing, driving, or carrying out self-care skills.

Developmental coordination disorder is diagnosed only if the impairment in motor skills significantly interferes with the performance of, or participation in, daily activities in family, social, school, or community life (Criterion B). Examples of such activities include getting dressed, eating meals with age-appropriate utensils and without mess, engaging in physical games with others, using specific tools in class such as rulers and scissors, and participating in team exercise activities at school. Not only is ability to perform these actions impaired, but also marked slowness in execution is common. Handwriting competence is frequently affected, consequently affecting legibility and/or speed of written output and affecting academic achievement (the impact is distinguished from specific learning difficulty by the emphasis on the motoric component of written output skills). In adults, everyday skills in education and work, especially those in which speed and accuracy are required, are affected by coordination problems.

Criterion C states that the onset of symptoms of developmental coordination disorder must be in the early developmental period. However, developmental coordination disorder is typically not diagnosed before age 5 years because there is considerable variation in the age at acquisition of

many motor skills or a lack of stability of measurement in early childhood (e.g., some children catch up) or because other causes of motor delay may not have fully manifested.

Criterion D specifies that the diagnosis of developmental coordination disorder is made if the coordination difficulties are not better explained by visual impairment or attributable to a neurological condition. Thus, visual function examination and neurological examination must be included in the diagnostic evaluation. If intellectual developmental disorder (intellectual disability) is present, the motor difficulties are in excess of those expected for the mental age; however, no IQ cutoff or discrepancy criterion is specified.

Developmental coordination disorder does not have discrete subtypes; however, individuals may be impaired predominantly in gross motor skills or in fine motor skills, including handwriting skills.

Other terms used to describe developmental coordination disorder include *childhood dyspraxia, specific developmental disorder of motor function,* and *clumsy child syndrome*.

Associated Features

Some children with developmental coordination disorder show additional (usually suppressed) motor activity, such as choreiform movements of unsupported limbs or mirror movements. These “overflow” movements are referred to as *neurodevelopmental immaturities* or *neurological soft signs* rather than neurological abnormalities. In both current literature and clinical practice, their role in diagnosis is still unclear, requiring further evaluation.

Prevalence

The prevalence of developmental coordination disorder in children ages 5–11 years ranges from 5% to 8% cross-nationally (in the United Kingdom, 1.8% of children age 7 years are diagnosed with severe developmental coordination disorder and 3% with probable developmental coordination disorder); and 7%–8% in Canada, Sweden, and Taiwan. Males are more often affected than females, with a male:female ratio between 2:1 and 7:1.

Development and Course

The course of developmental coordination disorder is variable but stable at least to 1-year and 2-year follow-up. Although there may be improvement in the longer term, problems with coordinated movements continue through adolescence in an estimated 50%–70% of children. Onset is in early childhood. Delayed motor milestones may be the first signs, or the disorder is first recognized when the child attempts tasks such as holding a knife and fork, buttoning clothes, or playing ball games. In middle childhood, there are difficulties with motor aspects of assembling puzzles, building models, playing ball, and handwriting, as well as with organizing belongings, when motor sequencing and coordination are required. In early adulthood, there is continuing difficulty in learning new tasks involving complex/automatic motor skills, including driving and using tools. Inability to take notes and handwrite quickly may affect performance in the workplace. Co-occurrence with other disorders (see the section "Comorbidity" for this disorder) has an additional impact on presentation, course, and outcome.

Risk and Prognostic Factors

Environmental. Developmental coordination disorder is associated with prematurity and low birth weight and with prenatal exposure to alcohol.

Genetic and physiological. Impairments in underlying neurodevelopmental processes have been found in visual-motor skills, including both visual-motor perception and spatial mentalizing. Cerebellar dysfunction, which affects the ability to make rapid motoric adjustments as the complexity of the required movements increases, may also be involved. However, the precise neural basis of developmental coordination disorder remains unclear. Because of the co-occurrence of developmental coordination disorder with other neurodevelopmental disorders, including attention-deficit/hyperactivity disorder (ADHD), specific learning disabilities, and autism spectrum disorder, shared genetic effect has been proposed. However, consistent co-occurrence in twins appears only in severe cases.

Course modifiers. Individuals with ADHD and with developmental coordination disorder demonstrate more impairment than individuals with ADHD without developmental coordination disorder.

Culture-Related Diagnostic Issues

Developmental coordination disorder occurs across cultural, ethnoracial, and socioeconomic contexts. At the same time, cultural variations in motor development (both accelerated and delayed relative to U.S. norms) have been reported. These appear to be associated with caregiving practices related to expectations of independent mobility during development, inadequate opportunities for mobility among children in severe poverty, and differences in measurement methodology. By definition, “activities of daily living” implies cultural differences

necessitating consideration of the context in which the individual child is living as well as whether the child has had appropriate opportunities to learn and practice such activities. Higher prevalence of developmental coordination disorder in studies of children from some low- and middle-income countries may reflect the impact of socioeconomic disadvantage on motor development.

Functional Consequences of Developmental Coordination Disorder

Developmental coordination disorder leads to impaired functional performance in activities of daily living (Criterion B), and the impairment is increased with co-occurring conditions. Consequences of developmental coordination disorder include reduced participation in team play and sports; poor self-esteem and sense of self-worth; emotional or behavioral problems; impaired academic achievement; poor physical fitness; reduced physical activity and obesity; and poor health-related quality of life.

Differential Diagnosis

Motor impairments due to another medical condition. Problems in coordination may be associated with visual function impairment and specific neurological disorders (e.g., cerebral palsy, progressive lesions of the cerebellum, neuromuscular disorders). In such cases, there are additional findings on neurological examination.

Intellectual developmental disorder (intellectual disability). If intellectual developmental disorder is present, motor competences may be impaired in accordance with the intellectual disability. However, if the motor difficulties are in excess of what could be accounted for by the intellectual developmental disorder, and criteria for developmental coordination disorder are met, developmental coordination disorder can be diagnosed as well.

Attention-deficit/hyperactivity disorder. Individuals with ADHD may fall, bump into objects, or knock things over. Careful observation across different contexts is required to ascertain if lack of motor competence is attributable to distractibility and impulsiveness rather than to developmental coordination disorder. If criteria for both ADHD and developmental coordination disorder are met, both diagnoses can be given.

Autism spectrum disorder. Individuals with autism spectrum disorder may be uninterested in participating in tasks requiring complex coordination skills, such as ball sports, which will affect test performance and function but not reflect core motor competence. Co-occurrence of developmental coordination disorder and autism spectrum disorder is common. If criteria for both disorders are met, both diagnoses can be given.

Joint hypermobility syndrome. Individuals with syndromes causing hyperextensible joints (found on physical examination; often with a complaint of pain) may present with symptoms similar to those of developmental coordination disorder.

Comorbidity

Disorders that commonly co-occur with developmental coordination disorder include communication disorders; specific learning disorder (especially reading and writing); problems of inattention, including ADHD (the most frequent coexisting condition, with about 50% co-occurrence); autism spectrum disorder; disruptive and emotional behavior problems; and joint hypermobility syndrome. Different clusters of co-occurrence may be present (e.g., a cluster with severe reading disorders, fine motor problems, and handwriting problems; another cluster with impaired movement control and motor planning). Presence of other disorders does not exclude developmental coordination disorder but may make testing more difficult

and may independently interfere with the execution of activities of daily living, thus requiring examiner judgment in ascribing impairment to motor skills.

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Stereotypic Movement Disorder

Diagnostic Criteria F98.4

A. Repetitive, seemingly driven, and apparently purposeless motor behavior (e.g., hand shaking or waving, body rocking, head banging, self-biting, hitting own body).

B. The repetitive motor behavior interferes with social, academic, or other activities and may result in self-injury.

C. Onset is in the early developmental period.

D. The repetitive motor behavior is not attributable to the physiological effects of a substance or neurological condition and is not better explained by another neurodevelopmental or mental disorder (e.g., trichotillomania [hair-pulling disorder], obsessive-compulsive disorder).

Specify if:

With self-injurious behavior (or behavior that would result in an injury if preventive measures were not used)

Without self-injurious behavior

Specify if:

Associated with a known genetic or other medical condition, neurodevelopmental disorder, or environmental factor (e.g., Lesch-Nyhan syndrome, intellectual developmental disorder [intellectual disability], intrauterine alcohol exposure)

Coding note: Use additional code to identify the associated genetic or other medical condition, neurodevelopmental disorder, or environmental factor.

Specify current severity:

Mild: Symptoms are easily suppressed by sensory stimulus or distraction.

Moderate: Symptoms require explicit protective measures and behavioral modification.

Severe: Continuous monitoring and protective measures are required to prevent serious injury.

Recording Procedures

For stereotypic movement disorder that is associated with a known genetic or other medical condition, neurodevelopmental disorder, or environmental factor, record stereotypic movement disorder associated with (name of condition, disorder, or factor) (e.g., stereotypic movement disorder associated with Lesch-Nyhan syndrome).

Specifiers

The severity of non-self-injurious stereotypic movements ranges from mild presentations that are easily suppressed by a sensory stimulus or distraction to continuous movements that markedly interfere with all activities of daily living. Self-injurious behaviors range in severity along various dimensions, including the frequency, impact on adaptive functioning, and severity of bodily injury (from mild bruising or erythema from hitting hand against body, to lacerations or amputation of digits, to retinal detachment from head banging).

Diagnostic Features

The essential feature of stereotypic movement disorder is repetitive, seemingly driven, and apparently purposeless motor behavior (Criterion A). These behaviors are often rhythmical movements of the head, hands, or body without obvious adaptive function. The movements may or may not respond to efforts to stop them. Among typically developing children, the

repetitive movements can usually be stopped when attention is directed to them

or when the child is distracted from performing them. Among children with neurodevelopmental disorders, the behaviors are typically less responsive to such efforts. In other cases, the individual demonstrates self-restraining behaviors (e.g., sitting on hands, wrapping arms in clothing, finding a protective device).

The repertoire of behaviors is variable; each individual presents with his or her own individually patterned, “signature” behavior. Examples of non-self-injurious stereotypic movements include, but are not limited to, body rocking, bilateral flapping or rotating hand movements, flicking or fluttering fingers in front of the face, arm waving or flapping, and head nodding; mouth stretching is commonly seen in association with upper limb movements. Stereotyped self-injurious behaviors include, but are not limited to, repetitive head banging, face slapping, eye poking, and biting of hands, lips, or other body parts. Eye poking is particularly concerning; it occurs more frequently among children with visual impairment. Multiple movements may be combined (e.g., cocking the head, rocking the torso, waving a small string repetitively in front of the face).

Stereotypic movements may occur many times during a day, lasting a few seconds to several minutes or longer. Frequency can vary from many occurrences in a single day to several weeks elapsing between episodes. The behaviors vary in context, occurring when the individual is engrossed in other activities, when excited, stressed, fatigued, or bored. Criterion A requires that the movements be “apparently” purposeless. However, some functions may be served by the movements. For example, stereotypic movements might reduce anxiety in response to external stressors.

Criterion B requires that the stereotypic movements interfere with social, academic, or other activities and, in some children, may result in self-injury (or would if protective measures were not used). The presence or absence of self-injurious behavior should be indicated using the specifiers “with self-injurious behavior” or “without self-injurious behavior.” Onset of stereotypic movements is in the early developmental period (Criterion C).

Criterion D requires that the repetitive, stereotyped behavior in stereotypic movement disorder is not attributable to the physiological effects of a substance or neurological condition and is not better explained by another neurodevelopmental or mental disorder. The presence of stereotypic movements may indicate an undetected neurodevelopmental problem, especially in children ages 1–3 years.

Prevalence

Simple stereotypic movements (e.g., rocking) are common in young typically developing children (e.g., 5%–19% in the United Kingdom and United States). Complex stereotypic movements are much less common (occurring in approximately 3%–4%). Between 4% and 16% of individuals with intellectual developmental disorder (intellectual disability) in samples from high-income countries engage in stereotypy and self-injury. The risk is greater in individuals with severe intellectual developmental disorder. Among individuals with intellectual developmental disorder living in residential facilities, 10%–15% may have stereotypic movement disorder with self-injury. Repetitive and restricted behaviors and interests may be risk markers for the onset of self-injury, aggression, and destruction in children with severe intellectual developmental disorder.

Development and Course

Stereotypic movements typically begin within the first 3 years of life. Simple stereotypic movements are common in infancy and may be involved in acquisition of motor mastery. In children who develop complex motor stereotypies, approximately 80% exhibit symptoms before age 24 months, 12% between 24 and 35 months, and 8% at 36 months or older. In most typically developing children, the severity and frequency of stereotyped movements diminish over time. Onset of complex motor stereotypies may be in infancy or later in the developmental period. Among individuals with intellectual developmental

disorder, the stereotyped, self-injurious behaviors may persist for years, even though the typography or pattern of self-injury may change.

Risk and Prognostic Factors

Environmental. Social isolation is a risk factor for self-stimulation that may progress to stereotypic movements with repetitive self-injury. Environmental stress may also trigger stereotypic behavior. Fear may alter physiological state, resulting in increased frequency of stereotypic behaviors.

Genetic and physiological. Stereotypic movement disorder is believed to be somewhat heritable based on the high frequency of cases that have a positive family history of motor stereotypies. Significant reduction in the putamen volume in children with stereotypies suggests that distinct cortical-striatal pathways associated with habitual behaviors (i.e., premotor to posterior putamen circuits) may be the underlying anatomical site in complex motor stereotypies. Lower cognitive functioning is linked to greater risk for stereotypic behaviors and poorer response to interventions. Stereotypic movements are more frequent among individuals with moderate-to-severe/profound intellectual developmental disorder, who by virtue of a particular syndrome (e.g., Rett syndrome) or environmental factor (e.g., an environment with relatively insufficient stimulation) seem to be at higher risk for stereotypies. Repetitive self-injurious behavior may be a behavioral phenotype in neurogenetic syndromes. For example, in Lesch-Nyhan syndrome, there are both stereotypic dystonic movements and self-mutilation of fingers, lip biting, and other forms of self-injury unless the individual is restrained, and in Rett syndrome and Cornelia de Lange syndrome, self-injury may result from the hand-to-mouth stereotypies. Stereotypic behaviors may also result from a painful medical condition (e.g., middle ear infection, dental problems, gastroesophageal reflux).

Culture-Related Diagnostic Issues

Stereotypic repetitive behaviors, with or without self-injury, variedly manifest in many cultures. Cultural attitudes toward unusual behaviors may result in delayed diagnosis. Overall cultural tolerance and attitudes toward stereotypic movement vary and must be considered.

Differential Diagnosis

Normal development.

Simple stereotypic movements are common in infancy and early childhood. Rocking may occur in the transition from sleep to awake, a behavior that usually resolves with age. Complex stereotypies are less common in typically developing children and can usually be suppressed by distraction or sensory stimulation. The individual's daily routine is rarely affected, and the movements generally do not cause the child distress. The diagnosis would not be appropriate in these circumstances.

Autism spectrum disorder. Stereotypic movements may be a presenting symptom of autism spectrum disorder and should be considered when repetitive movements and behaviors are being evaluated. Deficits of social communication and reciprocity manifesting in autism spectrum disorder are generally absent in stereotypic movement disorder, and thus social interaction, social communication, and rigid repetitive behaviors and interests are distinguishing features. When autism spectrum disorder is present, stereotypic movement disorder is diagnosed only when there is self-injury or when the stereotypic behaviors are sufficiently severe to become a focus of treatment.

Tic disorders. Typically, stereotypies have an earlier age at onset (before 3 years) than do tics, which have a mean age at onset of 4–6 years. They also are consistent and fixed in their pattern or topography compared with tics, which are variable in their presentation,

typically changing in character over time. Stereotypies may involve arms, hands, or the entire body, while tics commonly involve eyes, face, head, and shoulders. Stereotypies are more fixed, rhythmic, and prolonged in duration than tics, which, generally, are brief, rapid, random, and fluctuating. Stereotypies are ego-syntonic (children enjoy them) as opposed to tics, which are usually ego-dystonic. Tics wax and wane in location and time and are uniquely associated with premonitory urge (a physical feeling that precedes many tic movements). Tics and stereotypic movements are both reduced by distraction.

Obsessive-compulsive and related disorders. Stereotypic movement disorder is distinguished from obsessive-compulsive disorder (OCD) by the absence of

obsessions, as well as by the nature of the repetitive behaviors. In OCD the individual feels driven to perform repetitive behaviors in response to an obsession or according to rules that must be applied rigidly, whereas in stereotypic movement disorder the behaviors are seemingly driven but apparently purposeless. Trichotillomania (hair-pulling disorder) and excoriation (skin-picking) disorder are characterized by body-focused repetitive behaviors (i.e., hair pulling and skin picking) that may be seemingly driven but that are not apparently purposeless, and that may not be patterned or rhythmical. Furthermore, onset in trichotillomania and excoriation disorder is not typically in the early developmental period, but rather around puberty or later.

Other neurological and medical conditions. The diagnosis of stereotypic movements requires the exclusion of habits, mannerisms, paroxysmal dyskinesias, and benign hereditary chorea. A neurological history and examination are required to assess features suggestive of other disorders, such as myoclonus, dystonia, tics, and chorea. Involuntary movements associated with a neurological condition may be distinguished by their signs and symptoms. For example, repetitive, stereotypic movements in tardive dyskinesia can be distinguished by a history of chronic neuroleptic use and characteristic oral or facial dyskinesia or irregular trunk or limb movements. These types of movements do not result in self-injury. Stereotypies are a common manifestation of a variety of neurogenetic disorders, such as Lesch-Nyhan syndrome, Rett syndrome, fragile X syndrome, Cornelia de Lange syndrome, and Smith-Magenis syndrome. For stereotypic movement disorder that is associated with a known genetic or other medical condition, neurodevelopmental disorder, or environmental factor, record stereotypic movement disorder associated with (name of condition, disorder, or factor) (e.g., stereotypic movement disorder associated with Lesch-Nyhan syndrome).

Substance-induced repetitive behaviors. A diagnosis of stereotypic movement disorder is not appropriate for repetitive skin picking or scratching associated with amphetamine intoxication or abuse. In such cases, the diagnosis substance/medication-induced obsessive-compulsive and related disorder would apply.

Functional (conversion) stereotypies.

Stereotyped movements must be distinguished from functional (conversion) movements. Sudden onset, distractibility, changing pattern with unexplained improvement or aggravation, and the coexistence of other symptoms of functional neurological symptom disorder (conversion disorder) are some of the typical features that help identify functional stereotypies.

Comorbidity

Common comorbidities in children with chronic motor stereotypies include attention-deficit hyperactivity disorder, motor coordination problems, tics/Tourette's disorder, and anxiety.

Tic Disorders

Diagnostic Criteria

Note: A tic is a sudden, rapid, recurrent, nonrhythmic motor movement or vocalization.

Tourette's Disorder F95.2

A. Both multiple motor and one or more vocal tics have been present at some time during the illness, although not necessarily concurrently.
B. The tics may wax and wane in frequency but have persisted for more than 1 year since first tic onset.
C. Onset is before age 18 years.
D. The disturbance is not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., Huntington's disease, postviral encephalitis).

Persistent (Chronic) Motor or Vocal Tic Disorder F95.1

A. Single or multiple motor or vocal tics have been present during the illness, but not both motor and vocal.
B. The tics may wax and wane in frequency but have persisted for more than 1 year since first tic onset.
C. Onset is before age 18 years.
D. The disturbance is not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., Huntington's disease, postviral encephalitis).
E. Criteria have never been met for Tourette's disorder.

Specify if:

With motor tics only

With vocal tics only

Provisional Tic Disorder **F95.0**

A. Single or multiple motor and/or vocal tics.
B. The tics have been present for less than 1 year since first tic onset.
C. Onset is before age 18 years.
D. The disturbance is not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., Huntington's disease, postviral encephalitis).
E. Criteria have never been met for Tourette's disorder or persistent (chronic) motor or vocal tic disorder.

Specifiers

The "motor tics only" or "vocal tics only" specifier is only required for persistent (chronic) motor or vocal tic disorder.

Diagnostic Features

Tic disorders comprise five diagnostic categories: Tourette's disorder, persistent (chronic) motor or vocal tic disorder, provisional tic disorder, and the other specified and unspecified tic disorders. Diagnosis for any of the

specific tic disorders is based on the presence of motor and/or vocal tics (Criterion A), duration of tics (Criterion B), age at onset (Criterion C), and absence of any known cause such as another medical condition or substance use (Criterion D). The tic disorder diagnoses are hierarchical in order (i.e., Tourette's disorder, followed by persistent [chronic] motor or vocal tic disorder, followed by provisional tic

disorder, followed by the other specified and unspecified tic disorders). Once a tic disorder at one level of the hierarchy is diagnosed, a lower hierarchy diagnosis cannot be made (Criterion E).

Tics are typically sudden, rapid, recurrent, nonrhythmic motor movements or vocalizations. Some motor tics can be slower twisting or tightening movements that occur over varying lengths of time. An individual may exhibit various tics over time, but, at any point in time, the tic repertoire may recur in a characteristic fashion. Although tics can include almost any muscle group or vocalization, certain tics, such as eye blinking or throat clearing, are common across patient populations. There is often a localized uncomfortable sensation (premonitory sensation) prior to a tic, and most individuals report an "urge" to tic. Consequently, tics are generally experienced as involuntary, but some tics can be voluntarily suppressed for varying lengths of time.

Explicit discussion of tics can serve as a trigger. Likewise, observing a gesture or sound in another person may result in an individual with a tic disorder making a similar gesture or sound, which may be incorrectly perceived by others as purposeful. This can be particularly problematic when the individual is interacting with authority figures who do not have an adequate understanding of tic disorders (e.g., teachers, supervisors, police).

Tics are classically categorized as either simple or complex. *Simple motor tics* are characterized by the limited involvement of specific muscle groups, often are of short duration, and can include eye blinks, facial grimaces, shoulder shrugs, or extension of the extremities. *Simple vocal tics* include throat clearing, sniffs, chirps, barks, or grunting often caused by contraction of the diaphragm or muscles of the oropharynx. *Complex motor tics* are of longer duration and often include a combination of simple tics

such as simultaneous head turning and shoulder shrugging. Complex tics can appear purposeful, such as head gestures or torso movements. They can also include imitations of someone else's movements (*echopraxia*) or sexual or taboo gestures (*copropraxia*). Similarly, *complex vocal tics* have linguistic meaning (words or partial words) and can include repeating one's own sounds or words (*palilalia*), repeating the last-heard word or phrase (*echolalia*), or uttering socially unacceptable words, including obscenities, or ethnic, racial, or religious slurs (*coprolalia*). Importantly, coprolalia is an abrupt, sharp bark or grunt utterance and lacks the prosody of similar inappropriate speech observed in human interactions.

The presence of motor and/or vocal tics varies across the five tic disorders (Criterion A). For Tourette's disorder, both motor and vocal tics must be present (though not necessarily concurrently), whereas for persistent (chronic) motor or vocal tic disorder, only motor or only vocal tics are present. For provisional tic disorder, motor and/or vocal tics may be present. For other specified or unspecified tic disorders, the tics or tic-like symptoms are best characterized as tics but are atypical in presentation or age at onset, or have a known etiology.

The 1-year minimum duration criterion (Criterion B) assures that individuals diagnosed with either Tourette's disorder or persistent (chronic) motor or vocal tic disorder have had persistent symptoms. Tics wax and wane in severity, and some individuals may have tic-free intervals of weeks to months; however, an individual who has had tics of greater than 1 year's duration since first tic onset would be considered to have persistent symptoms regardless of intermittent tic-free periods. For an individual with motor and/or vocal tics for less than 1 year since first tic onset, a provisional tic disorder diagnosis can be considered. The onset of tics must occur prior to age 18 years (Criterion C). Tic disorders typically begin in the prepubertal period, with an average age at onset between 4 and 6 years, and with the incidence of first-onset tic disorders decreasing in the later teen years. First onset of tics in adulthood is exceedingly rare and is often associated with exposures to illicit substances (e.g., excessive cocaine use), is the result of a central nervous system insult, or is related to a functional neurological disorder. Although first onset of tics is uncommon in teenagers and adults, it is not uncommon for adolescents and adults to present for an

initial diagnostic assessment and, when carefully evaluated, provide a history of milder tics dating

back to childhood, even if earlier phases of development included tic-free periods of months or years. First-onset abnormal movements suggestive of tics that occur outside of the usual age range should result in evaluation for other movement disorders, including functional tic-like complex movements or vocalizations.

Tics cannot be attributable to the physiological effects of a substance or another medical condition (Criterion D). When there is strong evidence from the history, physical examination, and/or laboratory results to suggest a plausible, proximal, and probable cause for a tic disorder, a diagnosis of other specified tic disorder should be used.

Having previously met diagnostic criteria for Tourette's disorder negates a possible diagnosis of persistent (chronic) motor or vocal tic disorder (Criterion E). Similarly, a previous diagnosis of persistent (chronic) motor or vocal tic disorder negates a diagnosis of provisional tic disorder or other specified or unspecified tic disorder (Criterion E).

Prevalence

Tics are common in childhood but transient in most cases. A national survey in the United States estimated 3 per 1,000 for the prevalence of clinically identified cases. The frequency of identified cases was lower among African Americans and Latinx individuals, which may be related to differences in access to care. The estimated prevalence of Tourette's disorder in Canada ranges from 3 to 9 per 1,000 in school-age children. Globally, males are more commonly affected than females, with the ratio varying from 2:1 to 4:1. Epidemiological studies have shown tics to be present in children from all continents, but exact prevalence rates are influenced by methodological differences in research.

Development and Course

First onset of tics is typically between ages 4 and 6 years. Eye blinking is highly characteristic as an initial symptom. Peak severity occurs between

ages 10 and 12 years, with a decline in severity during adolescence. Many adults with tic disorders experience diminished symptoms. However, a percentage of individuals will have persistently severe or worsening symptoms in adulthood.

Tics manifest similarly in all age groups and across the lifespan. Tics wax and wane in severity (frequency and intensity) and over time change with regard to the affected muscle groups and nature of vocalizations. Many individuals, including young children, report that their tics are associated with a localized bodily sensation preceding the tic and a premonitory urge to move. It can be difficult to find words to describe these premonitory sensations and urges. Tics associated with a premonitory urge may be experienced as not completely "involuntary" in that the urge and the tic can be resisted. An individual may also feel the need to perform a tic repeatedly or in a specific way until the individual feels that the tic has been done "just right." Often there is a feeling of relief and tension reduction following the expression of the tic or a series of tics.

The vulnerability toward developing co-occurring conditions changes as individuals pass through the age of risk for various co-occurring conditions. For example, prepubertal children with tic disorders are more likely to exhibit co-occurring attention-deficit/hyperactivity disorder (ADHD), obsessive-compulsive disorder (OCD), and separation anxiety disorder. Teenagers and adults are more vulnerable to developing mood and anxiety disorders as well as substance use disorders.

Risk and Prognostic Factors

Environmental. Early in brain development, a number of environmental risk factors have been identified, including advanced paternal age as well as pre- and perinatal adverse events (e.g., impaired fetal growth, maternal intrapartum fever, maternal smoking, severe maternal psychosocial stress, preterm birth, breech presentation, and cesarean delivery).

Genetic and physiological. Genetic factors influence tic expression and severity. The heritability of tic disorders is estimated to be 70%–85%, and there are no differences in familial risk or heritability between males and

females. Important risk alleles for Tourette's disorder and rare genetic variants in families with tic disorders have been identified. Common genetic variants have also been identified. They are shared across tic disorders in a graded fashion that correlates with disease severity. Indeed, tic disorders likely exist along a continuous developmental spectrum, based on both their phenomenology and their genetic background.

Chronic tic disorders have shared genetic variance with OCD, ADHD, and other neurodevelopmental disorders, including autism spectrum disorder. In addition, individuals with tic disorders are at increased risk to develop an autoimmune disorder (e.g., Hashimoto's thyroiditis). It is increasingly evident that the immune system and neuroinflammation play important roles in the pathobiology of tics in at least a subset of affected individuals (e.g., those with Sydenham's chorea). However, more work is needed to understand the biobehavioral underpinnings and the potential causative role of infections for other neuropsychiatric conditions, including pediatric acute-onset neuropsychiatric syndrome and pediatric autoimmune neuropsychiatric disorder associated with streptococcal infections.

Course modifiers. Tics are increased by anxiety, excitement, and exhaustion and are better during calm, focused activities. For example, many individuals typically have fewer tics when engaged in tasks that require focused attention and motor control. Stressful/exciting events (e.g., taking a test, participating in exciting activities) often make tics worse.

Culture-Related Diagnostic Issues

Tic disorders do not appear to vary in clinical characteristics, course, or etiology by ethnic, racialized, and cultural background, but these backgrounds may affect how tic disorders are perceived and managed in the family and community, influencing patterns of help seeking and choices of treatment, such as age at presentation at specialty services. For example, preferred social distance from individuals with tic disorders (e.g., when working or studying together) was greater in a Korean sample than in U.S. studies.

Sex- and Gender-Related Diagnostic Issues

Males are more commonly affected than females, but there are no sex differences in the kinds of tics, age at onset, or course. Women with persistent tic disorders may be more likely to experience anxiety and depression.

Association With Suicidal Thoughts or Behavior

A matched case-cohort study in Sweden from 1969 to 2013 demonstrated that individuals with Tourette's disorder or persistent (chronic) motor or vocal tic disorder have a substantially increased risk of suicide attempts (odds ratio 3.86) and suicide death (odds ratio 4.39), even after adjustment for psychiatric comorbidities, compared with matched general population control subjects. Persistence of tics after young adulthood and a prior suicide attempt were the strongest predictors of suicide death. Case-control data suggest that about 1 in 10 youth with persistent (chronic) motor or vocal tic disorder has suicidal thoughts and/or behaviors, particularly in the context of anger/frustration and associated with anxiety/depression, social problems or withdrawal, aggression and internalizing problems, tic severity, and related impairment.

Functional Consequences of Tic Disorders

Many individuals with mild to moderate tic severity experience no distress or impairment in functioning and may even be unaware of their tics. Individuals with more severe

symptoms generally have more impairment in daily living, but even individuals with moderate or even severe tic disorders may function well. The presence of a co-occurring condition, such as ADHD or OCD, can have greater impact on functioning than the tics themselves. Less commonly, tics disrupt functioning in daily activities and result in social isolation, interpersonal conflict, peer victimization, inability to work or to go to school, and lower quality of life. Often individuals with tics have difficulty focusing their attention on work-related tasks while they are actively trying to suppress their tics. The individual also may experience substantial psychological distress and even suicidal thoughts. Rare complications of

Tourette's disorder include physical injury, such as eye injury (from hitting oneself in the face), and orthopedic and neurological injury (e.g., disc disease related to forceful head and neck movements).

Differential Diagnosis

Abnormal movements that may accompany other medical conditions, including other movement disorders. *Motor stereotypies* are defined as involuntary rhythmic, repetitive, predictable movements that appear purposeful but serve no obvious adaptive function. They are often self-soothing or pleasurable and stop with distraction. Examples include repetitive hand waving/rotating, arm flapping, and finger wiggling. Motor stereotypies can usually be differentiated from tics based on the former's earlier age at onset (often younger than 3 years), prolonged duration (seconds to minutes), being repetitive and rhythmic in form and location, lacking a premonitory sensation or urge, and cessation with distraction (e.g., hearing name called or being touched). *Chorea* represents rapid, random, continual, abrupt, irregular, unpredictable, nonstereotyped actions that are usually bilateral and affect all parts of the body (i.e., face, trunk, and limbs). The timing, direction, and distribution of movements vary from moment to moment, and movements usually worsen during attempted voluntary action. *Dystonia* is the simultaneous sustained contraction of both agonist and antagonist muscles, resulting in a distorted posture or movement of parts of the body. Dystonic postures are often triggered by attempts at voluntary movements and are not seen during sleep.

Paroxysmal dyskinesias. Paroxysmal dyskinesias are characterized by episodic involuntary dystonic or choreoathetoid movements that are precipitated by voluntary movement or exertion and less commonly arise from normal background activity.

Myoclonus. Myoclonus is characterized by a sudden unidirectional movement that is often nonrhythmic. It may be worsened by movement and occur during sleep. Myoclonus is differentiated from tics by its rapidity, lack of suppressibility, and absence of a premonitory sensation or urge.

Obsessive-compulsive and related disorders. Differentiating compulsions in OCD from complex tics may be difficult, especially because they frequently

co-occur in the same individual. The compulsions of OCD are aimed at preventing or reducing anxiety or distress and are usually performed in response to an obsession (e.g., fear of contamination). In contrast, many individuals with a tic disorder feel the need to perform the action in a particular fashion, equally on both sides of the body a certain number of times or until a "just right" feeling is achieved. Body-focused repetitive behavior disorders (i.e., persistent hair-pulling, skin-picking, nail-biting) are more goal-directed and complex than tics.

Functional tic disorder. Functional disorders should also be considered when an individual presents with "tic attacks" that can go on for extended periods of time lasting from 15 minutes to several hours.

Comorbidity

Many medical and psychiatric conditions have been described as co-occurring with tic disorders, and ADHD, disruptive behavior, and OCD and related disorders are particularly

common. Children with ADHD may demonstrate disruptive behavior, social immaturity, and learning difficulties that may interfere with academic progress and interpersonal relationships and lead to greater impairment than that caused by a tic disorder. The obsessive-compulsive symptoms observed in tic disorders tend to have an earlier age at onset and often are characterized by a need for symmetry and exactness and/or forbidden or taboo thoughts (e.g., aggressive, sexual, or religious obsessions and related compulsions). Individuals with tic disorders can also have other movement disorders (e.g., Sydenham's chorea, stereotypic movement disorder) and other neurodevelopmental and psychiatric conditions, such as autism spectrum disorder and specific learning disorder. As noted earlier, teenagers and adults with a tic disorder are at increased risk for developing a mood, anxiety, or substance use disorder.

Other Specified Tic Disorder

F95.8

This category applies to presentations in which symptoms characteristic of a tic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for a tic disorder or any of the disorders in the neurodevelopmental disorders diagnostic class. The other specified tic disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for a tic disorder or any specific neurodevelopmental disorder. This is done by recording "other specified tic disorder" followed by the specific reason (e.g., "with onset after age 18 years").

Unspecified Tic Disorder

F95.9

This category applies to presentations in which symptoms characteristic of a tic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for a tic disorder or for any of the disorders in the neurodevelopmental disorders diagnostic class. The unspecified tic disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a tic disorder or for a specific neurodevelopmental disorder and includes presentations in which there is insufficient information to make a more specific diagnosis.

Other Neurodevelopmental Disorders

Other Specified Neurodevelopmental Disorder

F88

This category applies to presentations in which symptoms characteristic of a neurodevelopmental disorder that cause impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the neurodevelopmental disorders diagnostic class. The other specified neurodevelopmental disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific neurodevelopmental disorder. This is done by recording “other specified neurodevelopmental disorder” followed by the specific reason (e.g., “neurodevelopmental disorder associated with prenatal alcohol exposure”).

An example of a presentation that can be specified using the “other specified” designation is the following:

Neurodevelopmental disorder associated with prenatal alcohol exposure: Neurodevelopmental disorder associated with prenatal alcohol exposure is characterized by a range of developmental disabilities following exposure to alcohol in utero.

Unspecified Neurodevelopmental Disorder

F89

This category applies to presentations in which symptoms characteristic of a neurodevelopmental disorder that cause impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the neurodevelopmental disorders diagnostic class. The unspecified neurodevelopmental disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific neurodevelopmental disorder and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Schizophrenia Spectrum and Other Psychotic Disorders

Schizophrenia spectrum and other psychotic disorders include schizophrenia, other psychotic disorders, and schizotypal (personality) disorder. They are defined by abnormalities in one or more of the following five domains: delusions, hallucinations, disorganized thinking (speech), grossly disorganized or abnormal motor behavior (including catatonia), and negative symptoms.

Key Features That Define the Psychotic Disorders

Delusions

Delusions are fixed beliefs that are not amenable to change in light of conflicting evidence. Their content may include a variety of themes (e.g., persecutory, referential, somatic, religious, grandiose). *Persecutory delusions* (i.e., belief that one is going to be harmed, harassed, and so forth by an individual, organization, or other group) are most common. *Referential delusions* (i.e., belief that certain gestures, comments, environmental cues, and so forth are directed at oneself) are also common. *Grandiose delusions* (i.e., when an individual believes that he or she has exceptional abilities, wealth, or fame) and *erotomanic delusions* (i.e., when an individual believes falsely that another person is in love with him or her) are also seen. *Nihilistic delusions* involve the conviction that a major catastrophe will occur, and *somatic delusions* focus on preoccupations regarding health and organ function.

Delusions are deemed *bizarre* if they are clearly implausible and not understandable to same-culture peers and do not derive from ordinary life experiences. An example of a bizarre delusion is the belief that an outside force has removed his or her internal organs and replaced them with someone else's organs without leaving any wounds or scars. An example of a nonbizarre delusion is the belief that one is under surveillance by the police, despite a lack of convincing evidence. Delusions that express a loss of control over mind or body are generally considered to be bizarre; these include the belief that one's thoughts have been "removed" by some outside force (*thought withdrawal*), that alien thoughts have been put into one's mind (*thought insertion*), or that one's body or actions are being acted on or manipulated by some outside force (*delusions of control*).

The distinction between a delusion and a strongly held idea is sometimes difficult to determine and depends in part on the degree of conviction with which the belief is held despite clear or reasonable contradictory evidence regarding its veracity. Assessing delusions in individuals from a variety of cultural backgrounds can be difficult. Some religious and supernatural beliefs (e.g., evil eye, causing illness through curses, influence of spirits) may be viewed as bizarre and possibly delusional in some cultural contexts but be generally accepted in others. However, elevated religiosity can be a feature of many presentations of psychosis.

Individuals who have experienced torture, political violence, or discrimination can report fears that may be misjudged as persecutory delusions; these may represent instead intense fears of recurrence or posttraumatic symptoms. A careful evaluation of whether the

person's fears are justified given the nature of the trauma can help to differentiate appropriate fears from persecutory delusions.

Hallucinations

Hallucinations are perception-like experiences that occur without an external stimulus. They are vivid and clear, with the full force and impact of normal perceptions, and not under voluntary control. They may occur in

any sensory modality, but auditory hallucinations are the most common in schizophrenia and related disorders. Auditory hallucinations are usually experienced as voices, whether familiar or unfamiliar, that are perceived as distinct from the individual's own thoughts. The hallucinations must occur in the context of a clear sensorium; those that occur while falling asleep (*hypnagogic*) or waking up (*hypnopompic*) are considered to be within the range of normal experience. Hallucinations may be a normal part of religious experience in certain cultural contexts.

Disorganized Thinking (Speech)

Disorganized thinking (*formal thought disorder*) is typically inferred from the individual's speech. The individual may switch from one topic to another (*derailment or loose associations*). Answers to questions may be obliquely related or completely unrelated (*tangentiality*). Rarely, speech may be so severely disorganized that it is nearly incomprehensible and resembles receptive aphasia in its linguistic disorganization (*incoherence* or "word salad"). Because mildly disorganized speech is common and nonspecific, the symptom must be severe enough to substantially impair effective communication. The severity of the impairment may be difficult to evaluate if the person making the diagnosis comes from a different linguistic background than that of the person being examined. For example, some religious groups engage in glossolalia ("speaking in tongues"); others describe experiences of possession trance (trance states in which personal identity is replaced by an external possessing identity). These phenomena are characterized by disorganized speech. These instances do not represent signs of psychosis unless they are accompanied by other clearly psychotic symptoms. Less severe disorganized thinking or speech may occur during the prodromal and residual periods of schizophrenia.

Grossly Disorganized or Abnormal Motor Behavior (Including Catatonia)

Grossly disorganized or abnormal motor behavior may manifest itself in a variety of ways, ranging from childlike "silliness" to unpredictable agitation. Problems may be noted in any form of goal-directed behavior, leading to difficulties in performing activities of daily living.

Catatonic behavior is a marked decrease in reactivity to the environment. This ranges from resistance to instructions (*negativism*); to maintaining a rigid, inappropriate or bizarre posture; to a complete lack of verbal and motor responses (*mutism* and *stupor*). It can also include purposeless and excessive motor activity without obvious cause (*catatonic excitement*). Other features are repeated stereotyped movements, staring, grimacing, and the echoing of speech. Although catatonia has historically been associated with schizophrenia, catatonic symptoms are nonspecific and may occur in other mental disorders (e.g., bipolar or depressive disorders with catatonia) and in medical conditions (catatonic disorder due to another medical condition).

Negative Symptoms

Negative symptoms account for a substantial portion of the morbidity associated with schizophrenia but are less prominent in other psychotic disorders. Two negative symptoms are particularly prominent in schizophrenia: diminished emotional expression and avolition. *Diminished emotional expression* includes reductions in the expression of emotions in the

face, eye contact, intonation of speech (prosody), and movements of the hand, head, and face that normally give an emotional emphasis to speech. *Avolition* is a decrease in motivated self-initiated purposeful activities. The individual may sit for long periods of time and show little interest in participating in work or social activities. Other negative symptoms include alogia, anhedonia, and asociality. *Alogia* is manifested by diminished speech output. *Anhedonia* is the decreased ability to experience pleasure. Individuals with schizophrenia can still enjoy a pleasurable activity in the moment and can recall it, but show a reduction in the frequency of engaging in pleasurable activity. *Asociality* refers to the apparent lack of interest in social interactions and may be associated with avolition, but it can also be a manifestation of limited opportunities for social interactions.

Disorders in This Chapter

This chapter is organized along a gradient of psychopathology. Clinicians should first consider conditions that do not reach full criteria for a psychotic disorder or are limited to one domain of psychopathology. Then they should consider time-limited conditions. Finally, the diagnosis of a schizophrenia spectrum disorder requires the exclusion of another condition that may give rise to psychosis.

Schizotypal personality disorder is noted within this chapter as it is considered within the schizophrenia spectrum, although its full description is found in the chapter “Personality Disorders.” The diagnosis schizotypal personality disorder captures a pervasive pattern of social and interpersonal deficits, including reduced capacity for close relationships; cognitive or perceptual distortions; and eccentricities of behavior, usually beginning by early adulthood but in some cases first becoming apparent in childhood and adolescence. Abnormalities of beliefs, thinking, and perception are below the threshold for the diagnosis of a psychotic disorder.

Two conditions are defined by abnormalities limited to one domain of psychosis: delusions or catatonia. Delusional disorder is characterized by at least 1 month of delusions but no other psychotic symptoms. Catatonia is described later in the chapter and further in this discussion.

Brief psychotic disorder lasts more than 1 day and remits by 1 month. Schizophreniform disorder is characterized by a symptomatic presentation equivalent to that of schizophrenia except for its duration (less than 6 months) and the absence of a requirement for a decline in functioning.

Schizophrenia lasts for at least 6 months and includes at least 1 month of active-phase symptoms. In schizoaffective disorder, a mood episode and the active-phase symptoms of schizophrenia occur together and were preceded or are followed by at least 2 weeks of delusions or hallucinations without prominent mood symptoms.

Psychotic disorders may be induced by substances, medications, toxins, and other medical conditions. In substance/medication-induced psychotic disorder, the psychotic symptoms are judged to be a direct physiological consequence of a drug of abuse, a medication, or toxin exposure and cease after removal of the agent. In psychotic disorder due to another medical

condition, the psychotic symptoms are judged to be a direct physiological consequence of another medical condition.

Catatonia can occur in several disorders, including neurodevelopmental, psychotic, bipolar, depressive, and other mental disorders. This chapter also includes the diagnoses catatonia associated with another mental disorder (catatonia specifier), catatonic disorder due to another medical condition, and unspecified catatonia, and the diagnostic criteria for all three conditions are described together.

Other specified and unspecified schizophrenia spectrum and other psychotic disorders are included for classifying psychotic presentations that do not meet the criteria for any of the specific psychotic disorders, or psychotic symptomatology about which there is inadequate or contradictory information.

Clinician-Rated Assessment of Symptoms and Related Clinical Phenomena in Psychosis

Psychotic disorders are heterogeneous, and the severity of symptoms can predict important aspects of the illness, such as the degree of cognitive or neurobiological deficits. To move the field forward, a detailed framework for the assessment of severity is included in Section III, “Assessment Measures,” which may help with treatment planning, prognostic decision-making, and research on pathophysiological mechanisms. Section III, “Assessment Measures,” also contains dimensional assessments of the primary symptoms of psychosis, including hallucinations, delusions, disorganized speech (except for substance/medication-induced psychotic disorder and psychotic disorder due to another medical condition), abnormal psychomotor behavior, and negative symptoms, as well as dimensional assessments of depression and mania. The severity of mood symptoms in psychosis has prognostic value and guides treatment. Thus, dimensional assessments of depression and mania for all psychotic disorders alert clinicians to mood pathology and the need to treat where appropriate. The Section III scale also includes a dimensional assessment of

cognitive impairment. Many individuals with psychotic disorders have impairments in a range of cognitive domains that predict functional status. Clinical neuropsychological assessment can help guide diagnosis and treatment, but brief assessments without formal neuropsychological assessment can provide useful information that can be sufficient for diagnostic purposes. Formal neuropsychological testing, when conducted, should be administered and scored by personnel trained in the use of testing instruments. If a formal neuropsychological assessment is not conducted, the clinician should use the best available information to make a judgment. Further research on these assessments is necessary to determine their clinical utility; thus, the assessments available in Section III should serve as a prototype to stimulate such research.

Cultural Considerations in the Assessment of Psychotic Symptoms

Diagnostic accuracy and the quality of treatment planning may be enhanced by interview approaches, scales, and tools that have been adapted or validated for the person's culture and by using a cultural formulation interview (see Section III, "Culture and Psychiatric Diagnosis"). Assessing psychosis through interpreters or in a second or third language must avoid the misunderstanding of unfamiliar metaphors as delusions.

Schizotypal (Personality) Disorder

Criteria and text for schizotypal personality disorder can be found in the chapter "Personality Disorders." Because this disorder is considered part of the schizophrenia spectrum of disorders, and is labeled in this section of ICD-10 as schizotypal disorder, it is listed in this chapter and discussed in detail in the DSM-5 chapter "Personality Disorders."

Delusional Disorder

Diagnostic Criteria	F22

A. The presence of one (or more) delusions with a duration of 1 month or longer.

B. Criterion A for schizophrenia has never been met.

Note: Hallucinations, if present, are not prominent and are related to the delusional theme (e.g., the sensation of being infested with insects associated with delusions of infestation).

C. Apart from the impact of the delusion(s) or its ramifications, functioning is not markedly impaired, and behavior is not obviously bizarre or odd.

D. If manic or major depressive episodes have occurred, these have been brief relative to the duration of the delusional periods.

E. The disturbance is not attributable to the physiological effects of a substance or another medical condition and is not better explained by another mental disorder, such as body dysmorphic disorder or obsessive-compulsive disorder.

Specify whether:

Erotomanic type: This subtype applies when the central theme of the delusion is that another person is in love with the individual.

Grandiose type: This subtype applies when the central theme of the delusion is the conviction of having some great (but unrecognized) talent or insight or having made some important discovery.

Jealous type: This subtype applies when the central theme of the individual's delusion is that his or her spouse or lover is unfaithful.

Persecutory type: This subtype applies when the central theme of the delusion involves the individual's belief that he or she is being conspired against, cheated, spied on,

followed, poisoned or drugged, maliciously maligned, harassed, or obstructed in the pursuit of long-term goals.

Somatic type: This subtype applies when the central theme of the delusion involves bodily functions or sensations.

Mixed type: This subtype applies when no one delusional theme predominates.

Unspecified type: This subtype applies when the dominant delusional belief cannot be clearly determined or is not described in the specific types (e.g., referential delusions without a prominent persecutory or grandiose component).

Specify if:

With bizarre content: Delusions are deemed bizarre if they are clearly implausible, not understandable, and not derived from ordinary life experiences (e.g., an individual's belief that a stranger has removed his or her internal organs and replaced them with someone else's organs without leaving any wounds or scars).

Specify if:

The following course specifiers are only to be used after a 1-year duration of the disorder:

First episode, currently in acute episode: First manifestation of the disorder meeting the defining diagnostic symptom and time criteria. An *acute episode* is a time period in which the symptom criteria are fulfilled.

First episode, currently in partial remission: *Partial remission* is a time period during which an improvement after a previous episode is maintained and in which the defining criteria of the disorder are only partially fulfilled.

First episode, currently in full remission: *Full remission* is a period of time after a previous episode during which no disorder-specific symptoms are present.

Multiple episodes, currently in acute episode

Multiple episodes, currently in partial remission

Multiple episodes, currently in full remission

Continuous: Symptoms fulfilling the diagnostic symptom criteria of the disorder are remaining for the majority of the illness course, with subthreshold symptom periods being very brief relative to the overall course.

Unspecified

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor

behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter “Assessment Measures.”)

Note: Diagnosis of delusional disorder can be made without using this severity specifier.

Subtypes

In *erotomanic type,* the central theme of the delusion is that another person is in love with the individual. The person about whom this conviction is held is usually of higher status (e.g., a famous individual or a superior at work) but can be a complete stranger. Efforts to contact the object of the delusion are common. In *grandiose type,* the central theme of the delusion is the conviction of having some great talent or insight or of having made some important discovery. Less commonly, the individual may have the delusion of having a special relationship with a prominent individual or of being a prominent person (in which case the actual individual may be regarded as an impostor). Grandiose delusions may have a religious content. In *jealous type,* the central theme of the delusion is that of an

unfaithful partner. This belief is arrived at without due cause and is based on incorrect inferences supported by small bits of "evidence" (e.g., disarrayed clothing). The individual with the delusion usually confronts the spouse or lover and attempts to intervene in the imagined infidelity. In *persecutory type,* the central theme of the delusion involves the individual's belief of being conspired against, cheated, spied on, followed, poisoned, maliciously maligned, harassed, or obstructed in the pursuit of long-term goals. Small slights may be exaggerated and become the focus of a delusional system. The affected individual may engage in repeated attempts to obtain satisfaction by legal or legislative action. Individuals with persecutory delusions are often resentful and angry and may resort to violence against those they believe are hurting them. In *somatic type,* the central theme of the delusion involves bodily functions or sensations. Somatic delusions can occur in several forms. Most common is the belief that the individual emits a foul odor; that there is an infestation of insects on or in the skin; that there is an internal parasite; or that parts of the body are not functioning.

Diagnostic Features

The essential feature of delusional disorder is the presence of one or more delusions that persist for at least 1 month (Criterion A). A diagnosis of delusional disorder is not given if the individual has ever had a symptom presentation that met Criterion A for schizophrenia (Criterion B). Apart from the direct impact of the delusions, impairments in psychosocial functioning may be more circumscribed than those seen in other psychotic disorders such as schizophrenia, and behavior is not obviously bizarre or odd (Criterion C). If mood episodes occur concurrently with the delusions, the total duration of these mood episodes is brief relative to the total duration of the delusional periods (Criterion D). The delusions are not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., Alzheimer's disease) and are not better explained by another mental disorder, such as body dysmorphic disorder or obsessive-compulsive disorder (Criterion E).

In addition to the delusions identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital

for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders. Whereas delusions are a sine qua non of delusional disorder, hallucinations and negative symptoms are uncommon and disorganization is rare. By definition, the presence of catatonia in conjunction with delusions rules out delusional disorder, because Criterion A for schizophrenia would be met. A subset of cases has prominent depressive symptoms, but cognitive impairment and mania are rarely demonstrated.

Associated Features

Social, marital, or work problems can result from the delusional beliefs of delusional disorder. Individuals with delusional disorder may be able to factually describe that others view their beliefs as irrational but are unable to accept this themselves (i.e., there may be “factual insight” but no true insight). Many individuals develop irritable or dysphoric mood, which can sometimes be understood as a reaction to their delusional beliefs. Anger and violent behavior can occur with persecutory, jealous, and erotomanic types. The individual may engage in litigious or antagonistic behavior (e.g., sending hundreds of letters of protest to the government). Legal difficulties can occur, particularly in jealous and erotomanic types.

Prevalence

The lifetime prevalence of delusional disorder has been estimated at around 0.2% in a Finnish sample, and the most frequent subtype is persecutory. Delusional disorder, jealous type, is probably more common in men than in women, but there are no major sex or gender differences in the overall frequency of delusional disorder or in the content of the delusions.

Development and Course

On average, global functioning is generally better than that observed in schizophrenia. Although the diagnosis is generally stable, a proportion of individuals go on to develop schizophrenia. Whereas about a third of individuals with delusional disorder of 1–3 months’ duration subsequently

receive a diagnosis of schizophrenia, the diagnosis of delusional disorder is much less likely to change if the duration of the disorder is greater than 6–12 months. Although delusional disorder can occur in younger age groups, it may be more prevalent in older individuals.

Risk and Prognostic Factors

Genetic and physiological. Delusional disorder has a significant familial relationship with both schizophrenia and schizotypal personality disorder.

Culture-Related Diagnostic Issues

An individual's cultural and religious background must be taken into account in evaluating the possible presence of delusional disorder; in fact, some traditional beliefs unfamiliar to Western cultures may be wrongly labeled as delusional, so their context must be carefully assessed. The nature and content of delusions also vary among different cultural groups.

Functional Consequences of Delusional Disorder

The functional impairment is usually more circumscribed than that seen with other psychotic disorders, although in some cases, the impairment may be substantial and include poor occupational functioning and social isolation. When poor psychosocial functioning is present, delusional beliefs themselves often play a significant role. A common characteristic of individuals with delusional disorder is the apparent normality of their behavior and appearance when their delusional ideas are not being discussed or acted on. Men with delusional disorder generally have more severe symptoms and worse functional outcomes compared with women.

Differential Diagnosis

Obsessive-compulsive and related disorders. If an individual with obsessive-compulsive disorder is completely convinced that his or her obsessive-compulsive disorder beliefs are true, then the diagnosis of obsessive-compulsive disorder, with absent insight/delusional

beliefs specifier, should be given rather than a diagnosis of delusional disorder. Similarly, if an individual with body dysmorphic disorder is completely convinced that his or her body dysmorphic disorder beliefs are true, then the diagnosis of body dysmorphic disorder, with absent insight/delusional beliefs specifier, should be given rather than a diagnosis of delusional disorder.

Delirium, major neurocognitive disorder, and psychotic disorder due to another medical condition. Individuals with these disorders may present with symptoms that suggest delusional disorder. For example, simple persecutory delusions in the context of major neurocognitive disorder would be diagnosed as major neurocognitive disorder, with behavioral disturbance.

Substance/medication-induced psychotic disorder. A substance/medication-induced psychotic disorder cross-sectionally may be identical in symptomatology to delusional disorder but can be distinguished by the chronological relationship of substance use to the onset and remission of the delusional beliefs.

Schizophrenia and schizophreniform disorder. Delusional disorder can be distinguished from schizophrenia and schizophreniform disorder by the absence of the other characteristic symptoms of the active phase of schizophrenia. Furthermore, the quality of delusions can help distinguish between schizophrenia and delusional disorder. In schizophrenia, delusions show greater disorganization (the degree to which delusions are internally consistent, logical, and systematized), whereas in delusional disorder, they show greater conviction (the degree to which the individual is convinced of the reality of the delusion), greater extension (the degree to which the delusion involves various areas of the individual's life), and greater pressure (the degree to which the individual is preoccupied and concerned with the expressed delusion).

Depressive and bipolar disorders and schizoaffective disorder. These disorders may be distinguished from delusional disorder by the temporal relationship between the mood disturbance and the delusions and by the severity of the mood symptoms. If delusions occur exclusively during mood episodes, the diagnosis is major depressive or bipolar disorder, with psychotic features. Mood symptoms that meet full criteria for a mood episode can be

superimposed on delusional disorder. Delusional disorder can be diagnosed only if the total duration of all mood episodes remains brief relative to the total duration of the delusional disturbance. If not, then a diagnosis of other specified or unspecified schizophrenia spectrum and other psychotic disorder accompanied by other specified depressive disorder, unspecified depressive disorder, other specified bipolar and related disorder, or unspecified bipolar and related disorder is appropriate.

Brief Psychotic Disorder

Diagnostic Criteria F23

A. Presence of one (or more) of the following symptoms. At least one of these must be (1), (2), or (3):
   1. Delusions.
   2. Hallucinations.
   3. Disorganized speech (e.g., frequent derailment or incoherence).
   4. Grossly disorganized or catatonic behavior.

   Note: Do not include a symptom if it is a culturally sanctioned response.

B. Duration of an episode of the disturbance is at least 1 day but less than 1 month, with eventual full return to premorbid level of functioning.

C. The disturbance is not better explained by major depressive or bipolar disorder with psychotic features or another psychotic disorder such as schizophrenia or catatonia, and is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

Specify if:

With marked stressor(s) (brief reactive psychosis): If symptoms occur in response to events that, singly or together, would be markedly stressful to almost anyone in similar circumstances in the individual's culture.

Without marked stressor(s): If symptoms do not occur in response to events that, singly or together, would be markedly stressful to almost anyone in similar circumstances in the individual's culture.

With peripartum onset: If onset is during pregnancy or within 4 weeks postpartum.

Specify if:

With catatonia (refer to the criteria for catatonia associated with another mental disorder, p. 135, for definition).

Coding note: Use additional code F06.1 catatonia associated with brief psychotic disorder to indicate the presence of the comorbid catatonia.

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter "Assessment Measures.")

Note: Diagnosis of brief psychotic disorder can be made without using this severity specifier.

Diagnostic Features

The essential feature of brief psychotic disorder is a disturbance that involves at least one of the following positive psychotic symptoms: delusions, hallucinations, disorganized speech (e.g., frequent derailment or

incoherence), or grossly abnormal psychomotor behavior, including catatonia (Criterion A). An episode of the disturbance lasts at least 1 day but less than 1 month, and the individual eventually has a full return to the premorbid level of functioning (Criterion B). The disturbance is not better explained by a depressive or bipolar disorder with psychotic features, by schizoaffective disorder, or by schizophrenia and is not attributable to the physiological effects of a substance (e.g., a hallucinogen) or another medical condition (e.g., subdural hematoma) (Criterion C).

In addition to the four symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features

Individuals with brief psychotic disorder typically experience emotional turmoil or overwhelming confusion. They may have rapid shifts from one intense affect to another. Although the disturbance is brief, the level of impairment may be severe, and supervision may be required to ensure that nutritional and hygienic needs are met and that the individual is protected from the consequences of poor judgment, cognitive impairment, or acting on the basis of delusions. There appears to be an increased risk of suicidal behavior, particularly during the acute episode.

Prevalence

Brief psychotic disorder may account for 2%–7% of cases of first-onset psychosis in several countries.

Development and Course

Brief psychotic disorder may appear in adolescence or early adulthood, and onset can occur across the life span, with the average age at onset being the mid 30s. By definition, a diagnosis of brief psychotic disorder requires a full remission of all symptoms and an eventual full return to the premorbid level of functioning within 1 month of the onset of the disturbance. In some

individuals, the duration of psychotic symptoms may be quite brief (e.g., a few days).

Although brief psychotic disorder by definition reaches a full remission within 1 month, subsequently more than 50% of the individuals experience a relapse. Despite the possibility of relapse, for most individuals, outcome is favorable in terms of social functioning and symptomatology.

In less than half of cases diagnosed with DSM-IV brief psychotic disorder or ICD-10 acute and transient psychotic disorder, the diagnosis changes—more often to schizophrenia spectrum disorders and less often to affective disorders or to other psychotic disorders.

Culture-Related Diagnostic Issues

It is important to distinguish symptoms of brief psychotic disorder from culturally sanctioned response patterns. For example, in some religious ceremonies, an individual may report hearing voices, but these do not generally persist and are not perceived as abnormal by most members of the individual's community. In a wide range of cultural contexts, it would be common or expected for bereaved relatives to hear, see, or interact with the spirit of a recently deceased loved one without notable pathological sequelae. In addition, cultural and religious background must be taken into account when considering whether beliefs are delusional.

Differential Diagnosis

Other medical conditions. A variety of medical conditions can manifest with psychotic symptoms of short duration. Psychotic disorder due to another medical condition or a delirium is diagnosed when there is evidence from the history, physical examination, or laboratory tests that the delusions or hallucinations are the direct physiological consequence of a specific medical condition (e.g., Cushing's syndrome, brain tumor) (see "Psychotic Disorder Due to Another Medical Condition" later in this chapter).

Substance-related disorders. Substance/medication-induced psychotic disorder, substance-induced delirium, and substance intoxication are distinguished from brief psychotic disorder by the fact that a substance (e.g., a drug of abuse, a medication, exposure to a toxin) is judged to be etiologically related to the psychotic symptoms (see

"Substance/Medication-Induced Psychotic Disorder" later in this chapter). Laboratory tests, such as a urine drug screen or a blood alcohol level, may be helpful in making this determination, as may a careful history of substance use with attention to temporal relationships between substance intake and onset of the symptoms and to the nature of the substance being used.

Depressive and bipolar disorders. The diagnosis of brief psychotic disorder cannot be made if the psychotic symptoms are better explained by a mood episode (i.e., the psychotic symptoms occur exclusively during a full major depressive, manic, or mixed episode).

Other psychotic disorders. If the psychotic symptoms persist for 1 month or longer, the diagnosis is either schizophreniform disorder, delusional disorder, depressive disorder with psychotic features, bipolar disorder with psychotic features, or other specified or

unspecified schizophrenia spectrum and other psychotic disorder, depending on the other symptoms in the presentation. The differential diagnosis between brief psychotic disorder and schizophreniform disorder is difficult when the psychotic symptoms have remitted before 1 month in response to successful treatment with medication. Careful attention should be given to the possibility that a recurrent disorder (e.g., bipolar disorder, recurrent acute exacerbations of schizophrenia) may be responsible for any recurring psychotic episodes.

Malingering and factitious disorders. An episode of factitious disorder with predominantly psychological signs and symptoms may have the appearance of brief psychotic disorder, but in such cases there is evidence that the symptoms are intentionally produced. When malingering involves apparently psychotic symptoms, there is usually evidence that the illness is being feigned for an understandable goal.

Personality disorders. In certain individuals with personality disorders, psychosocial stressors may precipitate brief periods of psychotic symptoms. These symptoms are usually transient and do not warrant a separate

diagnosis. If psychotic symptoms persist for at least 1 day, an additional diagnosis of brief psychotic disorder may be appropriate.

Schizophreniform Disorder

Diagnostic Criteria F20.81

A. Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated). At least one of these must be (1), (2), or (3):
   1. Delusions.
   2. Hallucinations.
   3. Disorganized speech (e.g., frequent derailment or incoherence).
   4. Grossly disorganized or catatonic behavior.
   5. Negative symptoms (i.e., diminished emotional expression or avolition).

B. An episode of the disorder lasts at least 1 month but less than 6 months. When the diagnosis must be made without waiting for recovery, it should be qualified as “provisional.”

C. Schizoaffective disorder and depressive or bipolar disorder with psychotic features have been ruled out because either 1) no major depressive or manic episodes have occurred concurrently with the active-phase symptoms, or 2) if mood episodes have occurred during active-phase symptoms, they have been present for a minority of the total duration of the active and residual periods of the illness.

D. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

Specify if:

With good prognostic features: This specifier requires the presence of at least two of the following features: onset of prominent psychotic symptoms within 4 weeks of the first noticeable change in usual behavior or functioning; confusion or perplexity; good premorbid social and occupational functioning; and absence of blunted or flat affect.

Without good prognostic features: This specifier is applied if two or more of the above features have not been present.

Specify if:

With catatonia (refer to the criteria for catatonia associated with another mental disorder, p. 135, for definition).

Coding note: Use additional code F06.1 catatonia associated with schizophreniform disorder to indicate the presence of the comorbid catatonia.

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter “Assessment Measures.”)

Note: Diagnosis of schizophreniform disorder can be made without using this severity specifier.

Note: For additional information on Associated Features, Development and Course (age-related factors), Culture-Related Diagnostic Issues, Sex- and Gender-Related Diagnostic Issues, Differential Diagnosis, and Comorbidity, see the corresponding sections in Schizophrenia.

Diagnostic Features

The characteristic symptoms of schizophreniform disorder are identical to those of schizophrenia (Criterion A). Schizophreniform disorder is distinguished by its difference in duration: the total duration of the illness, including prodromal, active, and residual phases, is at least 1 month but less than 6 months (Criterion B). The duration requirement for schizophreniform disorder is intermediate between that for brief psychotic disorder, which lasts more than 1 day and remits by 1 month, and schizophrenia, which lasts for at least 6 months. The diagnosis of schizophreniform disorder is made under two conditions: 1) when an episode of illness lasts between 1 and 6 months and the individual has already recovered, and 2) when an individual is symptomatic for less than the 6 months' duration required for the diagnosis of schizophrenia but has not yet recovered. In this case, the diagnosis should be noted as "schizophreniform disorder (provisional)" because it is uncertain if the individual will recover from the disturbance within the 6-month period. If the disturbance persists beyond 6 months, the diagnosis should be changed to schizophrenia.

Another distinguishing feature of schizophreniform disorder is the lack of a criterion requiring impaired social and occupational functioning. While such impairments may potentially be present, they are not necessary for a diagnosis of schizophreniform disorder.

In addition to the five symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features

As with schizophrenia, currently there are no laboratory or psychometric tests for schizophreniform disorder. There are multiple brain regions where neuroimaging, neuropathological, and neurophysiological research has indicated abnormalities, but none are diagnostic.

Prevalence

Incidence of schizophreniform disorder across sociocultural settings is likely similar to that observed in schizophrenia. In the United States and other high-income countries, the incidence is low, possibly fivefold less than that of schizophrenia. In lower-income countries, the incidence may be higher, especially for the specifier “with good prognostic features”; in some of these settings schizophreniform disorder may be as common as schizophrenia.

Development and Course

The development of schizophreniform disorder is similar to that of schizophrenia. About one-third of individuals with an initial diagnosis of schizophreniform disorder (provisional) recover within the 6-month period and schizophreniform disorder is their final diagnosis. The majority of the remaining two-thirds of individuals will eventually receive a diagnosis of schizophrenia or schizoaffective disorder.

Risk and Prognostic Factors

Genetic and physiological. Relatives of individuals with schizophreniform disorder have an increased risk for schizophrenia.

Functional Consequences of Schizophreniform Disorder

For the majority of individuals with schizophreniform disorder who eventually receive a diagnosis of schizophrenia or schizoaffective disorder, the functional consequences are similar to the consequences of those disorders. Most individuals experience dysfunction in several areas of daily functioning, such as school or work, interpersonal relationships, and self-care. Individuals who recover from schizophreniform disorder have better functional outcomes.

Differential Diagnosis

Other mental disorders and medical conditions. A wide variety of mental disorders and medical conditions can manifest with psychotic symptoms that must be considered in the differential diagnosis of schizophreniform

disorder. These include psychotic disorder due to another medical condition or its treatment; delirium or major neurocognitive disorder; substance/medication-induced psychotic disorder or delirium; major depressive or bipolar disorder with psychotic features; schizoaffective disorder; other specified or unspecified bipolar and related disorder; major depressive or bipolar disorder with catatonic features; schizophrenia; delusional disorder; other specified or unspecified schizophrenia spectrum and other psychotic disorder; schizotypal, schizoid, or paranoid personality disorders; autism spectrum disorder; disorders presenting in childhood with disorganized speech; attention-deficit/hyperactivity disorder; obsessive-compulsive disorder; posttraumatic stress disorder; and traumatic brain injury.

Since the diagnostic criteria for schizophreniform disorder and schizophrenia differ primarily in duration of illness, the discussion of the differential diagnosis of schizophrenia also applies to schizophreniform disorder.

Brief psychotic disorder. Schizophreniform disorder differs in duration from brief psychotic disorder, which has a duration of less than 1 month.

Schizophrenia

Diagnostic Criteria F20.9

A. Two (or more) of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated). At least one of these must be (1), (2), or (3):
   1. Delusions.
   2. Hallucinations.
   3. Disorganized speech (e.g., frequent derailment or incoherence).

4. Grossly disorganized or catatonic behavior.
5. Negative symptoms (i.e., diminished emotional expression or avolition).

B. For a significant portion of the time since the onset of the disturbance, level of functioning in one or more major areas, such as work, interpersonal relations, or self-care, is markedly below the level achieved prior to the onset (or when the onset is in childhood or adolescence, there is failure to achieve expected level of interpersonal, academic, or occupational functioning).

C. Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least 1 month of symptoms (or less if successfully treated) that meet Criterion A (i.e., active-phase symptoms) and may include periods of prodromal or residual symptoms. During these prodromal or residual periods, the signs of the disturbance may be manifested by only negative symptoms or by two or more symptoms listed in Criterion A present in an attenuated form (e.g., odd beliefs, unusual perceptual experiences).

D. Schizoaffective disorder and depressive or bipolar disorder with psychotic features have been ruled out because either 1) no major depressive or manic episodes have occurred concurrently with the active-phase symptoms, or 2) if mood episodes have occurred during active-phase symptoms, they have been present for a minority of the total duration of the active and residual periods of the illness.

E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

F. If there is a history of autism spectrum disorder or a communication disorder of childhood onset, the additional diagnosis of schizophrenia is made only if prominent delusions or hallucinations, in addition to the other required

symptoms of schizophrenia, are also present for at least 1 month (or less if successfully treated).

Specify if:

The following course specifiers are only to be used after a 1-year duration of the disorder and if they are not in contradiction to the diagnostic course criteria.

First episode, currently in acute episode: First manifestation of the disorder meeting the defining diagnostic symptom and time criteria. An *acute episode* is a time period in which the symptom criteria are fulfilled.

First episode, currently in partial remission: *Partial remission* is a period of time during which an improvement after a previous episode is maintained and in which the defining criteria of the disorder are only partially fulfilled.

First episode, currently in full remission: *Full remission* is a period of time after a previous episode during which no disorder-specific symptoms are present.

Multiple episodes, currently in acute episode: Multiple episodes may be determined after a minimum of two episodes (i.e., after a first episode, a remission and a minimum of one relapse).

Multiple episodes, currently in partial remission

Multiple episodes, currently in full remission

Continuous: Symptoms fulfilling the diagnostic symptom criteria of the disorder are remaining for the majority of the illness course, with subthreshold symptom periods being very brief relative to the overall course.

Unspecified

Specify if:

With catatonia (refer to the criteria for catatonia associated with another mental disorder, p. 135, for definition).

Coding note: Use additional code F06.1 catatonia associated with schizophrenia to indicate the presence of

the comorbid catatonia.

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Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter “Assessment Measures.”)

Note: Diagnosis of schizophrenia can be made without using this severity specifier.

Diagnostic Features

The characteristic symptoms of schizophrenia involve a range of cognitive, behavioral, and emotional dysfunctions, but no single symptom is pathognomonic of the disorder. The diagnosis involves the recognition of a constellation of signs and symptoms associated with impaired occupational or social functioning. Individuals with the disorder will vary substantially on most features, as schizophrenia is a heterogeneous clinical syndrome.

At least two Criterion A symptoms must be present for a significant portion of time during a 1-month period or longer. At least one of these symptoms must be the clear presence of delusions (Criterion A1), hallucinations (Criterion A2), or disorganized speech (Criterion A3). Grossly disorganized or catatonic behavior (Criterion A4) and negative symptoms (Criterion A5) may also be present. In those situations in which the active-phase symptoms remit within a month in response to treatment, Criterion A is still met if the clinician estimates that they would have persisted in the absence of treatment.

Schizophrenia involves impairment in one or more major areas of functioning (Criterion B). If the disturbance begins in childhood or adolescence, the expected level of function is not attained. Comparing the

individual with unaffected siblings may be helpful. The dysfunction persists for a substantial period during the course of the disorder and does not appear to be a direct result of any single feature. Avolition (i.e., reduced drive to pursue goal-directed behavior; Criterion A5) is linked to the social dysfunction described under Criterion B. There is also strong evidence for a relationship between cognitive impairment (see the section “Associated Features” for this disorder) and functional impairment in individuals with schizophrenia.

Some signs of the disturbance must persist for a continuous period of at least 6 months (Criterion C). Prodromal symptoms often precede the active phase, and residual symptoms may follow it, characterized by mild or subthreshold forms of hallucinations or delusions. Individuals may express a variety of unusual or odd beliefs that are not of delusional proportions (e.g., ideas of reference or magical thinking); they may have unusual perceptual experiences (e.g., sensing the presence of an unseen person); their speech may be generally understandable but vague; and their behavior may be unusual but not grossly disorganized (e.g., mumbling in public). Negative symptoms are common in the prodromal and residual phases and can be severe. Individuals who had been socially active may become withdrawn from previous routines. Such behaviors are often the first sign of a disorder.

Mood symptoms and full mood episodes are common in schizophrenia and may be concurrent with active-phase symptomatology. However, as distinct from a psychotic mood disorder, a schizophrenia diagnosis requires the presence of delusions or hallucinations in the absence of mood episodes. In addition, mood episodes, taken in total, should be present for only a minority of the total duration of the active and residual periods of the illness.

In addition to the five symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features

Individuals with schizophrenia may display inappropriate affect (e.g., laughing in the absence of an appropriate stimulus); a dysphoric mood that can take the form of depression, anxiety, or anger; a disturbed sleep pattern (e.g., daytime sleeping and nighttime activity); and a lack of interest in eating or food refusal. Depersonalization, derealization, and somatic concerns may occur and sometimes reach delusional proportions. Anxiety and phobias are common. Cognitive deficits in schizophrenia are common and are strongly linked to vocational and functional impairments. These deficits can include decrements in declarative memory, working memory, language function, and other executive functions, as well as slower processing speed. Abnormalities in sensory processing and inhibitory capacity, as well as reductions in attention, are also found. Some individuals with schizophrenia show social cognition deficits, including deficits in the ability to infer the intentions of other people (theory of mind), and may attend to and then interpret irrelevant events or stimuli as meaningful, perhaps leading to the generation of explanatory delusions. These impairments frequently persist during symptomatic remission.

Some individuals with psychosis may lack insight or awareness of their disorder (i.e., anosognosia). This lack of “insight” includes unawareness of symptoms of schizophrenia and may be present throughout the entire course of the illness. Unawareness of illness is typically a symptom of schizophrenia itself rather than a coping strategy. It is comparable to the lack of awareness of neurological deficits following brain damage, termed *anosognosia.* This symptom is the most common predictor of nonadherence to treatment, and it predicts higher relapse rates, increased number of involuntary treatments, poorer psychosocial functioning, aggression, and a poorer course of illness.

Hostility and aggression can be associated with schizophrenia, although spontaneous or random assault is uncommon. Aggression is more frequent for younger males and for individuals with a past history of violence, nonadherence to treatment, substance abuse, and impulsivity. It should be noted that the vast majority of persons with schizophrenia are not aggressive and are more frequently victimized than are individuals in the general population.

Currently, there are no radiological, laboratory, or psychometric tests for the disorder. Differences are evident in multiple brain regions between

groups of healthy individuals and persons with schizophrenia, including evidence from neuroimaging, neuropathological, and neurophysiological studies. Differences are also evident in cellular architecture, white matter connectivity, and gray matter volume in a variety of regions such as the prefrontal and temporal cortices. Reduced overall brain volume has been observed, as well as increased brain volume reduction with age. Brain volume reductions with age are more pronounced in individuals with schizophrenia than in healthy individuals. Finally, individuals with schizophrenia appear to differ from individuals without the disorder in eye-tracking and electrophysiological indices.

Neurological soft signs common in individuals with schizophrenia include impairments in motor coordination, sensory integration, and motor sequencing of complex movements; left-right confusion; and disinhibition of associated movements. In addition, minor physical anomalies of the face and limbs may occur.

Prevalence

The estimated lifetime prevalence of schizophrenia is approximately 0.3%–0.7%, with variation over a fivefold range in meta-analyses of nationally representative surveys. Studies have shown increased prevalence and incidence of schizophrenia for some groups based on migration and refugee status, urbanicity, and the economic status and latitude of the country. It is important to note that the reported prevalence and incidence of schizophrenia may be affected by the fact that some groups are more likely to be misdiagnosed or overdiagnosed.

The sex ratio differs across samples and populations: for example, presentations with prominent negative symptoms and longer duration of disorder (associated with poorer outcome) show higher incidence rates for men, whereas definitions allowing for the inclusion of more mood symptoms and brief presentations (associated with better outcome) show equivalent risks for both sexes. A large worldwide study, which was based on a range of definitions of schizophrenia, found no difference in prevalence between the sexes.

Development and Course

The requisite psychotic features of the schizophrenia diagnosis typically emerge between the late teens and the mid-30s; onset prior to adolescence is rare. The peak onset age occurs in the early- to mid-20s for men and in the late-20s for women. The onset may be abrupt or insidious, but the majority of individuals manifest a slow and gradual development of a variety of clinically significant signs and symptoms, particularly social withdrawal, emo-tional changes, and cognitive changes producing a deterioration in role functioning. Half of these individuals display depressive symptoms. Prognosis is influenced both by dura-tion and by severity of illness and gender. Men, especially those with long duration of psychosis before treatment and lower premorbid adjustment, have more prominent negative symptoms, cognitive impairment, and generally worse functional outcomes than women. Sociocognitive deficits may manifest during development and precede the emergence of psychosis, taking the form of stable impairments during adulthood, refractory to antipsychotic medications.

Course and outcome in schizophrenia are heterogeneous, and prognosis is uncertain at the onset of psychosis. Although most individuals with schizophrenia remain vulnerable to exacerbation of psychotic symptoms and a chronic course defined by symptoms and functional impairment is common, many individuals experience periods of remission and even recovery. According to a meta-analysis of 79 longitudinal studies of first-episode psychosis with more than 1 year of follow-up, the pooled remission rate (qualitatively defined as mild or absent symptoms for at least 6 months) for first-episode schizophrenia was 56% and the pooled recovery rate (qualitatively defined as symptomatic and functional improvement for greater than 2 years) was 30%. A different meta-analysis of 50 studies of individuals with broadly defined schizophrenia (i.e., schizophrenia, schizophreniform, schizoaffective, or delusional disorder) found that the median proportion of individuals who met recovery criteria (at most mild symptoms and improvements in social and/or occupational functioning persisting for at least 2 years) was 13.5%. There is a tendency for reduced psychotic experiences during late life. In addition to psychosis, cognitive impairment and negative symptom pathology are core features of schizophrenia, and the course for these characteristic features is different

from that of positive psychotic symptoms. Cognition tends to decline during development prior to full psychosis and is relatively stable over the longer term. Negative symptoms, if present during development, also tend to be relatively stable traits over time. Negative symptoms that begin after psychosis onset are more variable and may reflect secondary causes. A degree of chronicity is required for a diagnosis of schizophrenia, and long-term course reflects a need for mental health care and living support in many individuals. While schizophrenia is generally not a progressive neurodegenerative disorder, life challenges, changing lifestyle, and persistent symptoms may lead to progressive dysfunction in more severe chronic cases.

The essential features of schizophrenia are the same in childhood, but it is more difficult to make the diagnosis. In children, delusions and hallucinations may be less elaborate than in adults, and visual hallucinations are more common and should be distinguished from normal fantasy play. Disorganized speech occurs in many disorders with childhood onset (e.g., autism spectrum disorder), as does disorganized behavior (e.g., attention-deficit/hyperactivity disorder). These symptoms should not be attributed to schizophrenia

without due consideration of the more common disorders of childhood. Childhood-onset cases tend to resemble poor-outcome adult cases, with gradual onset and prominent negative symptoms. Children who later receive the diagnosis of schizophrenia are more likely to have experienced nonspecific emotional-behavioral disturbances and psychopathology, intellectual and language alterations, and subtle motor delays.

Late-onset cases (i.e., onset after age 40 years) are overrepresented by women, who may have married. Often, the course is characterized by a predominance of psychotic symptoms with preservation of affect and social functioning. Such late-onset cases can still meet the diagnostic criteria for schizophrenia, but it is not yet clear whether this is the same condition as schizophrenia diagnosed prior to midlife (e.g., prior to age 55 years).

Risk and Prognostic Factors

Environmental. Season of birth has been linked to the incidence of schizophrenia, including late winter/early spring in some locations and summer for the deficit form of the disease. The incidence of schizophrenia and related disorders may be higher for children growing up in an urban environment, for refugees, for some migrant groups, and for socially oppressed groups facing discrimination. There is evidence that social deprivation, social adversity, and socioeconomic factors may be associated with increased rates of this disorder. Among individuals with schizophrenia and other psychotic disorders, the severity of positive and negative symptoms appears to be correlated with the severity of adverse childhood experiences, such as trauma and neglect. Higher rates of schizophrenia for some ethnic and racialized groups have been documented when they live in areas with lower proportions of people from the same ethnicity or racialized group. The reasons for this are not completely clear but appear related to several factors, including the following: 1) higher levels of discrimination or fear of discrimination; 2) less social support and more stigmatization of those with schizophrenia; 3) higher social isolation; and 4) decreased availability of and access to normalizing explanations of perceptual experiences and abnormal beliefs reported by individuals at high risk for developing schizophrenia.

Genetic and physiological. There is a strong contribution for genetic factors in determining risk for schizophrenia, although most individuals who have been diagnosed with schizophrenia have no family history of psychosis. Liability is conferred by a spectrum of risk alleles, common and rare, with each allele contributing only a small fraction to the total population variance. The risk alleles identified to date are also associated with other mental disorders, including bipolar disorder, depression, and autism spectrum disorder.

Pregnancy and birth complications with hypoxia and greater paternal age are associated with a higher risk of schizophrenia for the developing fetus. In addition, other prenatal and perinatal adversities, including stress, infection, malnutrition, maternal diabetes, and other medical conditions, have been linked with schizophrenia. However, the vast majority of offspring with these risk factors do not develop schizophrenia.

Culture-Related Diagnostic Issues

The form and content of schizophrenia symptoms can vary cross-culturally, including the following ways: the relative proportion of visual and auditory hallucinations (e.g., while auditory hallucinations tend to be more common than visual hallucinations around the world, the relative proportion of visual hallucinations may be particularly higher in some regions compared with others); the specific content of the delusions (e.g., persecutory, grandiose, somatic) and hallucinations (e.g., command, abusive, religious); and the level of fear associated with them. Cultural and socioeconomic factors must be considered, particularly when the individual and the clinician do not share the same cultural and socioeconomic background. Ideas that appear to be delusional in one cultural context (e.g., evil eye, causing illness through curses, influences of spirits) may be commonly held in others.

In some cultural contexts, visual or auditory hallucinations with a religious content (e.g., hearing God's voice) are a normal part of religious experience. In addition, the assessment of disorganized speech may be made difficult by linguistic variation in narrative styles across cultures. The assessment of affect requires sensitivity to differences in styles of emotional expression, eye contact, and body language, which vary across cultures. If the assessment is conducted in a language that is different from the individual's primary language, care must be taken to ensure that alogia is not related to linguistic barriers. In certain cultures, distress may take the form of hallucinations or pseudo-hallucinations and overvalued ideas that may present clinically similar to true psychosis but are normative to the individual's subgroup. Misdiagnosis of schizophrenia in individuals with mood disorders with psychotic features or with other psychiatric disorders is more likely to occur in members of underserved ethnic and racialized groups (in the United States, especially among African Americans). This may be attributable to clinical bias, racism, or discrimination leading to limited quality of information and potential misinterpretation of symptoms.

Sex- and Gender-Related Diagnostic Issues

A number of features distinguish the clinical expression of schizophrenia in women and men. The age at onset is later in women, with a second midlife peak. Symptoms tend to be more affect-laden among women, and there are more psychotic symptoms, as well as a greater propensity for psychotic symptoms to worsen in later life. Other symptom differences include less frequent negative symptoms and disorganization. Finally, social functioning tends to remain better preserved in women. There are, however, frequent exceptions to these general caveats.

Psychotic symptoms have been observed to worsen during the premenstrual time period when estrogen levels are dropping; consequently, increased psychiatric admission rates are seen in women with schizophrenia just before and during menses. Lower estrogen levels resulting from menopause may be another factor associated with the second peak of onset in women in midlife. Similarly, psychotic symptoms appear to improve during pregnancy when estrogen levels are high and worsen again postpartum when estrogen levels precipitously drop.

Association With Suicidal Thoughts or Behavior

Approximately 5%–6% of individuals with schizophrenia die by suicide, about 20% attempt suicide on one or more occasions, and many more have significant suicidal ideation. Suicidal behavior is sometimes in response to command hallucinations to harm oneself or others. Suicide risk remains high over the whole lifespan for men and women, although it may be especially high for younger men with comorbid substance use. Other risk factors include depressive symptoms, hopelessness, being unemployed, the period after a psychotic episode or hospital discharge, number of psychiatric admissions, closeness to onset of illness, and older age at illness onset. A systematic review and meta-analysis of longitudinal studies found that the odds of suicidal behavior during follow-up after first-episode psychosis were higher among individuals with depressive symptoms during first-episode psychosis compared with those without. A meta-analysis of a large number of studies of the relationship of schizophrenia with suicidal behavior found that alcohol, tobacco, and drug abuse; depression; number of hospitalizations; physical comorbidity; and family history of depression and suicidal behavior increased the risk of suicide attempt. Risk factors for

suicide included male sex, being younger, having a higher IQ, history of attempts, hopelessness, and poor adherence to treatment.

Functional Consequences of Schizophrenia

Schizophrenia is associated with significant social and occupational dysfunction. Among individuals with schizophrenia, deficits in reading ability are more severe than what would

be predicted by the general cognitive impairments associated with the disorder. Such deficits can be conceptualized as a secondary or acquired dyslexia that underlies the academic impairment observed in schizophrenia. Making educational progress and maintaining employment are frequently impaired by avolition or other disorder manifestations, even when the cognitive skills are sufficient for the tasks at hand. Most individuals are employed at a lower level than their parents, and most, particularly men, do not marry or have limited social contacts outside of their family.

Differential Diagnosis

Major depressive or bipolar disorder with psychotic or catatonic features. The distinction between schizophrenia and major depressive or bipolar disorder with psychotic features or with catatonia depends on the temporal relationship between the mood disturbance and the psychosis, and on the severity of the depressive or manic symptoms. If delusions or hallucinations occur exclusively during a major depressive or manic episode, the diagnosis is depressive or bipolar disorder with psychotic features.

Schizoaffective disorder. A diagnosis of schizoaffective disorder requires that a major depressive or manic episode occur concurrently with the active-phase symptoms and that the mood symptoms be present for a majority of the total duration of the active periods.

Schizophreniform disorder and brief psychotic disorder. These disorders are of shorter duration than schizophrenia as specified in Criterion C, which requires 6 months of symptoms. In schizophreniform disorder, the

disturbance is present less than 6 months, and in brief psychotic disorder, symptoms are present at least 1 day but less than 1 month.

Delusional disorder. Delusional disorder can be distinguished from schizophrenia by the absence of the other symptoms characteristic of schizophrenia (e.g., delusions, prominent auditory or visual hallucinations, disorganized speech, grossly disorganized or catatonic behavior, negative symptoms).

Schizotypal personality disorder. Schizotypal personality disorder may be distinguished from schizophrenia by subthreshold symptoms that are associated with persistent personality features.

Obsessive-compulsive disorder and body dysmorphic disorder. Individuals with obsessive-compulsive disorder and body dysmorphic disorder may present with poor or absent insight, and the preoccupations may reach delusional proportions. But these disorders are distinguished from schizophrenia by their prominent obsessions, compulsions, preoccupations with appearance or body odor, hoarding, or body-focused repetitive behaviors.

Posttraumatic stress disorder. Posttraumatic stress disorder may include flashbacks that have a hallucinatory quality, and hypervigilance may reach paranoid proportions. But a traumatic event and characteristic symptom features relating to reliving or reacting to the event are required to make the diagnosis of posttraumatic stress disorder.

Autism spectrum disorder or communication disorders. These disorders may also have symptoms resembling a psychotic episode but are distinguished by their respective deficits in social interaction with repetitive and restricted behaviors and other cognitive and communication deficits. An individual with autism spectrum disorder or communication disorder must have symptoms that meet full criteria for schizophrenia, with prominent hallucinations or delusions for at least 1 month, in order to be diagnosed with schizophrenia as a comorbid condition.

Other mental disorders associated with a psychotic episode. The diagnosis of schizophrenia is made only when the psychotic episode is persistent and not attributable to the physiological effects of a substance or another medical condition. Individuals with a delirium or major or minor neurocognitive disorder may present with psychotic symptoms,

but these would have a temporal relationship to the onset of cognitive changes consistent with those disorders.

Substance/medication-induced psychotic disorder. Individuals with substance/medication-induced psychotic disorder may present with symptoms characteristic of Criterion A for schizophrenia, but the substance/medication-induced psychotic disorder can usually be distinguished by the chronological relationship of substance use to the onset and remission of the psychosis in the absence of substance use.

Comorbidity

Rates of comorbidity with substance-related disorders are high in schizophrenia. Over half of individuals with schizophrenia have tobacco use disorder and smoke cigarettes regularly. Comorbidity with anxiety disorders is increasingly recognized in schizophrenia. Rates of obsessive-compulsive disorder and panic disorder are elevated in individuals with schizophrenia compared with the general population. Schizotypal or paranoid personality disorder may sometimes precede the onset of schizophrenia.

Life expectancy is reduced in individuals with schizophrenia because of associated medical conditions. Weight gain, diabetes, metabolic syndrome, and cardiovascular and pulmonary disease are more common in schizophrenia than in the general population. Poor engagement in health maintenance behaviors (e.g., cancer screening, exercise) increases the risk of chronic disease, but other disorder factors, including medications, lifestyle, cigarette smoking, and diet, may also play a role. A shared vulnerability for psychosis and medical conditions may explain some of the medical comorbidity of schizophrenia.

Schizoaffective Disorder

Diagnostic Criteria

A. An uninterrupted period of illness during which there is a major mood episode (major depressive or manic) concurrent with Criterion A of schizophrenia.

   Note: The major depressive episode must include Criterion A1: Depressed mood.

B. Delusions or hallucinations for 2 or more weeks in the absence of a major mood episode (depressive or manic) during the lifetime duration of the illness.

C. Symptoms that meet criteria for a major mood episode are present for the majority of the total duration of the active and residual portions of the illness.

D. The disturbance is not attributable to the effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

Specify whether:

F25.0 Bipolar type: This subtype applies if a manic episode is part of the presentation. Major depressive episodes may also occur.

F25.1 Depressive type: This subtype applies if only major depressive episodes are part of the presentation.

Specify if:

With catatonia (refer to the criteria for catatonia associated with another mental disorder, p. 135, for definition).

Coding note: Use additional code F06.1 catatonia associated with schizoaffective disorder to indicate the presence of the comorbid catatonia.

Specify if:

The following course specifiers are only to be used after a 1-year duration of the disorder and if they are not in contradiction to the diagnostic course criteria.

First episode, currently in acute episode: First manifestation of the disorder meeting the defining diagnostic symptom and time criteria. An *acute episode* is a time period in which the symptom criteria are fulfilled.

First episode, currently in partial remission: *Partial remission* is a time period during which an improvement after a previous episode is maintained and in which the defining criteria of the disorder are only partially fulfilled.

First episode, currently in full remission: *Full remission* is a period of time after a previous episode during which no disorder-specific symptoms are present.

Multiple episodes, currently in acute episode: Multiple episodes may be determined after a minimum of two episodes (i.e., after a first episode, a remission and a minimum of one relapse).

Multiple episodes, currently in partial remission

Multiple episodes, currently in full remission

Continuous: Symptoms fulfilling the diagnostic symptom criteria of the disorder are remaining for the majority of the illness course, with subthreshold symptom periods being very brief relative to the overall course.

Unspecified

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, disorganized speech, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter “Assessment Measures.”)

Note: Diagnosis of schizoaffective disorder can be made without using this severity specifier.

Diagnostic Features

The diagnosis of schizoaffective disorder is based on the assessment of an uninterrupted period of illness during which the individual continues to display active or residual symptoms of psychotic illness. The diagnosis is usually, but not necessarily, made during the period of psychotic illness. At some time during the period, Criterion A for schizophrenia has to be met. Criteria B (social dysfunction), C (6-month duration), and F (exclusion of autism spectrum disorder or other communication disorder of childhood onset) for schizophrenia do not have to be met. In addition to meeting Criterion A for schizophrenia, there is a major mood episode (major depressive or manic) (Criterion A for schizoaffective disorder). Because loss of interest or pleasure is common in schizophrenia, to meet Criterion A for schizoaffective disorder, the major depressive episode must include pervasive depressed mood (i.e., the presence of markedly diminished interest or pleasure is not sufficient). Episodes of depression or mania are present for the majority of the total duration of the illness (i.e., after Criterion A has been met) (Criterion C for schizoaffective disorder). To separate schizoaffective disorder from a depressive or bipolar disorder with psychotic features, delusions or hallucinations must be present for at least 2 weeks in the absence of a major mood episode (depressive or manic) at some point during the lifetime duration of the illness (Criterion B for schizoaffective disorder). The symptoms must not be attributable to the effects of a substance or another medical condition (Criterion D for schizoaffective disorder).

Criterion C for schizoaffective disorder specifies that mood symptoms meeting criteria for a major mood episode must be present for the majority of the total duration of the active and residual portion of the illness. Criterion C requires the assessment of mood symptoms for the entire lifetime course of a psychotic illness. If the mood symptoms are present

for only a relatively brief period, the diagnosis is schizophrenia, not schizoaffective disorder. When deciding whether an individual’s presentation meets Criterion C, the clinician should review the total

duration of psychotic illness (i.e., both active and residual symptoms) and determine when significant mood symptoms (untreated or in need of treatment with antidepressant and/or mood-stabilizing medication) accompanied the psychotic symptoms. This determination requires sufficient historical information and clinical judgment. For example, an individual with a 4-year history of active and residual symptoms of schizophrenia develops depressive and manic episodes that, taken together, do not occupy more than 1 year during the 4-year history of psychotic illness. This presentation would not meet Criterion C.

In addition to the five symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features

Occupational and social functioning is frequently impaired, but this is not a defining criterion (in contrast to schizophrenia). Restricted social contact and difficulties with self-care are associated with schizoaffective disorder, but negative symptoms may be less severe and less persistent than those seen in schizophrenia. Anosognosia (i.e., poor insight) is also common in schizoaffective disorder, but the deficits in insight may be less severe and pervasive than those in schizophrenia. Individuals with schizoaffective disorder may be at increased risk for later developing episodes of major depressive disorder or bipolar disorder if mood symptoms continue following the remission of symptoms meeting Criterion A for schizophrenia. There may be associated alcohol and other substance-related disorders.

There are no tests or biological measures that can provide definitive assistance in making the diagnosis of schizoaffective disorder. Neuropsychological testing typically shows cognitive deficits in areas such as executive function, verbal memory, and speed of processing, and these may be less pronounced than in schizophrenia. Schizoaffective disorder is often characterized by gray matter volume loss on brain imaging, in much the same way that schizophrenia is.

Prevalence

Schizoaffective disorder appears to be about one-third as common as schizophrenia. Lifetime prevalence of schizoaffective disorder was estimated to be 0.3% in a Finnish sample and is higher in women than in men when DSM-IV diagnostic criteria were used. This rate would be expected to be lower because of the more stringent requirement of DSM-5 Criterion C (i.e., mood symptoms meeting criteria for a major mood episode must be present for the majority of the total duration of the active and residual portion of the illness).

Development and Course

The typical age at onset of schizoaffective disorder is early adulthood, although onset can occur anytime from adolescence to late in life. A significant number of individuals diagnosed with another psychotic illness initially will receive the diagnosis schizoaffective disorder later when the pattern of mood episodes has become more apparent, whereas others may be diagnosed with mood disorders before independent psychotic symptoms are detected.

Conversely, some individuals will have a change in diagnosis from schizoaffective disorder to a mood disorder or to schizophrenia over time. A change in diagnosis from schizoaffective disorder to schizophrenia was more common than a change to mood disorder

under DSM-IV criteria, and that difference is expected to be more pronounced under DSM-5 as the current Criterion C for schizoaffective disorder has become more stringent, requiring mood symptoms to be present for the majority of the illness as compared with the DSM-IV definition, which only required mood symptoms to be present for a “substantial” portion. The prognosis for schizoaffective disorder is somewhat better than the prognosis for schizophrenia but worse than the prognosis for mood disorders.

Schizoaffective disorder may occur in a variety of temporal patterns. The following is a typical pattern: An individual may have pronounced auditory hallucinations and persecutory delusions for 2 months before the onset of a prominent major depressive episode. The psychotic symptoms

and the full major depressive episode are then present for 4 months. Then, the individual recovers completely from the major depressive episode, but the psychotic symptoms persist for another month before they too disappear. During this period of illness, the individual's symptoms concurrently met criteria for a major depressive episode and Criterion A for schizophrenia, and during this same period of illness, auditory hallucinations and delusions were present both before and after the depressive phase. The total period of illness lasted for about 7 months, with psychotic symptoms alone present during the initial 2 months, both depressive and psychotic symptoms present during the next 4 months, and psychotic symptoms alone present during the last month. In this instance, the depressive episode was present for a majority of the total duration of the psychotic disturbance, and thus the presentation qualifies for a diagnosis of schizoaffective disorder.

The temporal relationship between the mood symptoms and the psychotic symptoms across the lifespan is variable. Depressive or manic symptoms can occur before the onset of psychosis, during acute psychotic episodes, during residual periods, and after cessation of psychosis. For example, an individual might present with prominent mood symptoms during the prodromal stage of schizophrenia. This pattern is not necessarily indicative of schizoaffective disorder, since it is the co-occurrence of psychotic and mood symptoms that is diagnostic. For an individual with symptoms that clearly meet the criteria for schizoaffective disorder but who on further follow-up only presents with residual psychotic symptoms (such as subthreshold psychosis and/or prominent negative symptoms), the diagnosis may be changed to schizophrenia, as the total proportion of psychotic illness compared with mood symptoms becomes more prominent. Schizoaffective disorder, bipolar type, may be more common in young adults, whereas schizoaffective disorder, depressive type, may be more common in older adults.

Risk and Prognostic Factors

Genetic and physiological. Among individuals with schizophrenia, there may be an increased risk for schizoaffective disorder in first-degree relatives. The risk for schizoaffective disorder may also be increased among

individuals who have a first-degree relative with bipolar disorder or schizoaffective disorder itself. The molecular genetic composite signatures known as polygenic risk scores for schizophrenia, bipolar disorder, and major depressive disorder may all be elevated in schizoaffective disorder.

Culture-Related Diagnostic Issues

Cultural and socioeconomic factors must be considered, particularly when the individual and the clinician do not share the same cultural and economic background. Ideas that appear to be delusional in one cultural context (e.g., evil eye, causing illness through curses, influences of spirits) may be commonly held in others. There is also some evidence in the literature that African American and Hispanic populations whose symptoms meet criteria for schizoaffective disorder are more likely to be diagnosed with schizophrenia. To mitigate the impact of clinician bias, care must be taken to ensure a comprehensive evaluation that includes both psychotic and mood symptoms.

Association With Suicidal Thoughts or Behavior

The lifetime risk of suicide for schizophrenia and schizoaffective disorder is 5%, and the presence of depressive symptoms is correlated with a higher risk for suicide. There is evidence that suicide rates are higher in North American populations than in European, Eastern European, South American, and Indian populations of individuals with schizophrenia or schizoaffective disorder.

Functional Consequences of Schizoaffective Disorder

Schizoaffective disorder is associated with global dysfunction, including in social and occupational domains, but dysfunction is not a diagnostic criterion (as it is for schizophrenia), and there is substantial variability between individuals diagnosed with schizoaffective disorder.

Differential Diagnosis

Other mental disorders and medical conditions.

A wide variety of psychiatric and medical conditions can manifest with psychotic and mood symptoms and must be considered in the differential diagnosis of schizoaffective disorder. These include delirium; major neurocognitive disorder; substance/medication-induced psychotic disorder or neurocognitive disorder; bipolar disorders, with psychotic features; major depressive disorder, with psychotic features; depressive or bipolar disorders, with catatonic features; schizotypal, schizoid, or paranoid personality disorder; brief psychotic disorder; schizophreniform disorder; schizophrenia; delusional disorder; and other specified and unspecified schizophrenia spectrum and other psychotic disorders.

Psychotic disorder due to another medical condition. Other medical conditions and substance use can manifest with a combination of psychotic and mood symptoms, and thus psychotic disorder due to another medical condition needs to be excluded.

Schizophrenia, bipolar, and depressive disorders. Distinguishing schizoaffective disorder from schizophrenia and from depressive and bipolar disorders with psychotic features is often difficult. Criterion C is designed to separate schizoaffective disorder from schizophrenia, and Criterion B is designed to distinguish schizoaffective disorder from a depressive or bipolar disorder with psychotic features. More specifically, schizoaffective disorder can be distinguished from a major depressive or bipolar disorder with psychotic features based on the presence of prominent delusions and/or hallucinations for at least 2 weeks in the absence of a major mood episode. In contrast, in depressive or bipolar disorder with psychotic features, the psychotic features only occur during the mood episode(s). Because the relative proportion of mood to psychotic symptoms may change over time, the appropriate diagnosis may change from and to schizoaffective disorder. (For example, a diagnosis of schizoaffective disorder for a severe and prominent major depressive episode lasting 4 months during the first 6 months of a chronic psychotic illness would be changed to schizophrenia if active psychotic or prominent residual symptoms persist over several years without a recurrence of another mood episode.) Achieving greater clarity about the relative proportion of mood to psychotic symptoms over time and about their concurrence may require collateral information from medical records and from informants.

Comorbidity

Many individuals diagnosed with schizoaffective disorder are also diagnosed with other mental disorders, especially substance use disorders and anxiety disorders. Similarly, the incidence of medical conditions, including metabolic syndrome, is increased above base rate for the general population and leads to decreased life expectancy.

Substance/Medication-Induced Psychotic Disorder

Diagnostic Criteria

A. Presence of one or both of the following symptoms:
   1. Delusions.
   2. Hallucinations.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):
   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a psychotic disorder that is not substance/medication-induced. Such evidence of an independent psychotic disorder could include the following:

   The symptoms preceded the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the

cessation of acute withdrawal or severe intoxication; or there is other evidence of an independent non-substance/medication-induced psychotic disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance/medication]-induced psychotic disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given. If a mild substance use disorder is comorbid with the substance-induced psychotic disorder, the 4th position character is “1,” and the clinician should record “mild [substance] use disorder” before the substance-induced psychotic disorder (e.g., “mild cocaine use disorder with cocaine-induced psychotic disorder”). If a moderate or severe substance use disorder is comorbid with the substance-induced psychotic disorder, the 4th position character is “2,” and the clinician should record “moderate [substance] use disorder” or “severe [substance] use disorder,” depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is “9,” and the clinician should record only the substance-induced psychotic disorder.

	ICD-10-CM		
	With mild use disorder	**With moderate or severe use disorder**	**Without use disorder**
Alcohol	F10.159	F10.259	F10.959
Cannabis	F12.159	F12.259	F12.959
Phencyclidine	F16.159	F16.259	F16.959
Other hallucinogen	F16.159	F16.259	F16.959
Inhalant	F18.159	F18.259	F18.959
Sedative, hypnotic, or anxiolytic	F13.159	F13.259	F13.959
Amphetamine-type substance (or other stimulant)	F15.159	F15.259	F15.959
Cocaine	F14.159	F14.259	F14.959
Other (or unknown) substance	F19.159	F19.259	F19.959

Specify (see Table 1 in the chapter “Substance-Related and Addictive Disorders,” which indicates whether “with onset during intoxication” and/or “with onset during withdrawal” applies to a given substance class; or *specify* “with onset after medication use”):

With onset during intoxication: If criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: If symptoms developed at initiation of medication, with a change in use of medication, or during withdrawal of medication.

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter “Assessment Measures.”)

Note: Diagnosis of substance/medication-induced psychotic disorder can be made without using this severity specifier.

Recording Procedures

The name of the substance/medication-induced psychotic disorder begins with the specific substance (e.g., cocaine, dexamethasone) that is presumed to be causing the delusions or hallucinations. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., dexamethasone), the code for “other (or unknown) substance” should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the same code should also be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word “with,” followed by the name of the substance-induced psychotic disorder, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal). For example, in the case of delusions occurring during intoxication in a man with a severe cocaine use disorder, the diagnosis is F14.259 severe cocaine use disorder with cocaine-induced psychotic disorder, with onset during intoxication. A separate diagnosis of the comorbid severe cocaine use disorder is not given. If the substance-induced psychotic disorder occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance

use disorder is noted (e.g., F16.959 phencyclidine-induced psychotic disorder, with onset during intoxication). When more than one substance is judged to play a significant role in the development of psychotic symptoms, each should be listed separately (e.g., F12.259 severe cannabis use disorder with cannabis-induced psychotic disorder, with onset during intoxication; F16.159 mild phencyclidine use disorder with phencyclidine-induced psychotic disorder, with onset during intoxication).

Diagnostic Features

The essential features of substance/medication-induced psychotic disorder are prominent delusions and/or hallucinations (Criterion A) that are judged to be due to the physiological effects of a substance/medication (i.e., a drug of abuse, a medication, or a toxin exposure) (Criterion B). Hallucinations that the individual realizes are substance/medication-induced are not included here and instead would be diagnosed as substance intoxication or substance withdrawal with the accompanying specifier "with perceptual disturbances" (applies to alcohol withdrawal; cannabis intoxication; sedative, hypnotic, or anxiolytic withdrawal; and stimulant intoxication).

A substance/medication-induced psychotic disorder is distinguished from an independent psychotic disorder by considering the onset, course, and other factors. For drugs of abuse, there must be evidence from the history, physical examination, or laboratory findings of substance use, intoxication, or withdrawal. Substance/medication-induced psychotic disorders arise during or soon after exposure to or withdrawal from a medication or after substance intoxication or withdrawal but can persist for weeks, whereas independent psychotic disorders may precede the onset of substance/medication use or may occur during times of sustained abstinence. Once initiated, the psychotic symptoms may continue as long as the substance/medication use continues. Another consideration is the presence of features that are atypical of an independent psychotic disorder (e.g., atypical age at onset or course). For example, the appearance of delusions de novo in a male person older than 35 years without a known history of an independent psychotic disorder should suggest the possibility of a substance/medication-induced psychotic disorder. Even a prior history of an independent psychotic disorder does not rule out the possibility of a

substance/medication-induced psychotic disorder. In contrast, factors that suggest that the psychotic symptoms are better accounted for by an independent psychotic disorder include persistence of psychotic symptoms for a substantial period of time (i.e., a month or more) after the end of substance intoxication or acute substance withdrawal or after cessation of medication use; or a history of prior recurrent independent psychotic disorders. Other causes of psychotic symptoms must be considered even in an individual with substance intoxication or withdrawal, because substance use problems are not uncommon among individuals with non-substance/medication-induced psychotic disorders.

In addition to the two symptom domain areas identified in the diagnostic criteria (i.e., delusions and hallucinations), the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Associated Features

Psychotic disorders can occur in association with intoxication with the following classes of substances: alcohol; cannabis; hallucinogens, including phencyclidine and related substances; inhalants; sedatives, hypnotics, and anxiolytics; stimulants (including cocaine); and other (or unknown) substances. Psychotic disorders can occur in association with withdrawal from the following classes of substances: alcohol; sedatives, hypnotics, and anxiolytics; and other (or unknown) substances.

Some of the medications reported to evoke psychotic symptoms include anesthetics and analgesics, anticholinergic agents, anticonvulsants, antihistamines, antihypertensive and cardiovascular medications, antimicrobial medications, antiparkinsonian medications, chemotherapeutic agents (e.g., cyclosporine, procarbazine), corticosteroids, gastrointestinal medications, muscle relaxants, nonsteroidal anti-inflammatory medications, other over-the-counter medications (e.g., phenylephrine, pseudoephedrine), antidepressant medication, and disulfiram. Toxins reported to induce psychotic symptoms include anticholinesterase, organophosphate

insecticides, sarin and other nerve gases, carbon monoxide, carbon dioxide, and volatile substances such as fuel or paint.

Prevalence

Prevalence of substance/medication-induced psychotic disorder in the general population is unknown. Between 7% and 25% of individuals presenting with a first episode of psychosis in different settings are reported to have substance/medication-induced psychotic disorder.

Development and Course

The initiation of the psychotic symptoms may vary considerably with the substance. For example, smoking a high dose of cocaine may produce psychosis within minutes, whereas days or weeks of high-dose alcohol or sedative use may be required to produce psychosis. Alcohol-induced psychotic disorder, with hallucinations, usually occurs only after prolonged, heavy ingestion of alcohol in individuals who have moderate to severe alcohol use disorder, and the hallucinations are generally auditory in nature.

Psychotic disorders induced by amphetamine-type substances and cocaine share similar clinical features. Persecutory delusions may rapidly develop shortly after use of amphetamine or a similarly acting sympathomimetic. The hallucination of bugs or vermin crawling in or under the skin (formication) can lead to scratching and extensive skin excoriations. Cannabis-induced psychotic disorder may develop shortly after high-dose cannabis use and usually involves persecutory delusions, marked anxiety, emotional lability, and depersonalization. The disorder usually remits within a day but in some cases may persist longer.

Substance/medication-induced psychotic disorder may at times persist when the offending agent is removed, such that it may be difficult initially to distinguish it from an independent psychotic disorder. Agents such as amphetamine-type substances, phencyclidine, and cocaine have been reported to evoke temporary psychotic states that can sometimes persist for weeks or longer despite removal of the agent and treatment with neuroleptic medication. In later life, polypharmacy for medical conditions and exposure to medications for parkinsonism, cardiovascular disease, and other medical

disorders may be associated with a greater likelihood of psychosis induced by prescription medications as opposed to substances of abuse.

According to data from a Danish registry study that followed cases of substance-induced psychosis longitudinally over 20 years, roughly one-third (32%) of individuals with substance-induced psychosis are later diagnosed with a schizophrenia spectrum disorder (26%) or a bipolar disorder (8%), with the highest rate (44%) for cannabis-induced psychotic disorder.

Diagnostic Markers

With substances for which relevant blood levels are available (e.g., blood alcohol level, other quantifiable blood levels such as digoxin), the presence of a level consistent with toxicity may increase diagnostic certainty.

Functional Consequences of Substance/Medication-Induced Psychotic Disorder

Substance/medication-induced psychotic disorder is typically severely disabling and consequently is observed most frequently in emergency departments, as individuals are often brought to the acute-care setting when it occurs. However, the disability is typically self-limited and resolves upon removal of the offending agent.

Differential Diagnosis

Substance intoxication or substance withdrawal. Individuals intoxicated with stimulants, cannabis, the opioid meperidine, or phencyclidine, or those withdrawing from alcohol or sedatives, may experience altered perceptions that they recognize as drug effects. If reality testing for these experiences remains intact (i.e., the individual recognizes that the perception is substance induced and neither believes in nor acts on it), the diagnosis is not substance/medication-induced psychotic disorder. Instead, substance intoxication or substance withdrawal, with perceptual disturbances, is diagnosed (e.g., cocaine intoxication, with perceptual disturbances). “Flashback” hallucinations that can occur long after the use of

hallucinogens has stopped are diagnosed as hallucinogen persisting perception disorder. If substance/medication-induced psychotic symptoms occur exclusively during the course of a delirium, as in severe forms of alcohol withdrawal, the psychotic symptoms are considered to be an associated feature of the delirium and are not diagnosed separately. Delusions in the context of a major or mild neurocognitive disorder would be diagnosed as major or mild neurocognitive disorder, with behavioral disturbance.

Independent psychotic disorder. A substance/medication-induced psychotic disorder is distinguished from an independent psychotic disorder, such as schizophrenia, schizoaffective disorder, delusional disorder, brief psychotic disorder, other specified schizophrenia spectrum and other psychotic disorder, or unspecified schizophrenia spectrum and other psychotic disorder, by the fact that a substance is judged to be etiologically related to the symptoms.

Psychotic disorder due to another medical condition. A substance/medication-induced psychotic disorder due to a prescribed treatment for a mental disorder or medical condition must have its onset while the individual is receiving the medication (or during withdrawal, if there is a withdrawal syndrome associated with the medication). Because individuals with medical conditions often take medications for those conditions, the clinician must consider the possibility that the psychotic symptoms are caused by the physiological consequences of the medical condition itself rather than the medication, in which case psychotic disorder due to another medical condition is diagnosed. The history often provides the primary basis for such a judgment. At times, a change in the treatment for the medical condition (e.g., medication substitution or discontinuation) may be needed to determine empirically for that individual whether the medication is the causative agent. If the clinician has ascertained that the disturbance is attributable to both a medical condition and substance/medication use, both diagnoses (i.e., psychotic disorder due to another medical condition and substance/medication-induced psychotic disorder) may be given.

Other specified or unspecified schizophrenia spectrum and other psychotic disorder.

The psychotic symptoms included in the diagnosis of substance/medication-induced psy-chotic disorder are limited to either delusions or hallucinations. Individuals with other

substance-induced psychotic symptoms (e.g., disorganized or catatonic behavior; disorganized speech; incoherence or irrational content) should be classified in the category other specified or unspecified schizophrenia spectrum and other psychotic disorder.

Psychotic Disorder Due to Another Medical Condition

Diagnostic Criteria

A. Prominent hallucinations or delusions.
B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.
C. The disturbance is not better explained by another mental disorder.
D. The disturbance does not occur exclusively during the course of a delirium.
E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify whether:

Code based on predominant symptom:

F06.2 With delusions: If delusions are the predominant symptom.

F06.0 With hallucinations: If hallucinations are the predominant symptom.

Coding note: Include the name of the other medical condition in the name of the mental disorder (e.g., F06.2 psychotic disorder due to malignant lung neoplasm, with delusions). The other medical condition should be coded and listed separately immediately before the psychotic disorder due to the medical condition (e.g., C34.90 malignant lung neoplasm; F06.2 psychotic disorder due to malignant lung neoplasm, with delusions).

Specify current severity:

Severity is rated by a quantitative assessment of the primary symptoms of psychosis, including delusions, hallucinations, abnormal psychomotor behavior, and negative symptoms. Each of these symptoms may be rated for its current severity (most severe in the last 7 days) on a 5-point scale ranging from 0 (not present) to 4 (present and severe). (See Clinician-Rated Dimensions of Psychosis Symptom Severity in the chapter "Assessment Measures.")

Note: Diagnosis of psychotic disorder due to another medical condition can be made without using this severity specifier.

Specifiers

In addition to the symptom domain areas identified in the diagnostic criteria, the assessment of cognition, depression, and mania symptom domains is vital for making critically important distinctions between the various schizophrenia spectrum and other psychotic disorders.

Diagnostic Features

The essential features of psychotic disorder due to another medical condition are prominent delusions or hallucinations that are judged to be attributable to the physiological effects of another medical condition and are not better explained by another mental disorder (e.g., the symptoms are not a psychologically mediated response to a severe medical condition, in

which case a diagnosis of brief psychotic disorder, with marked stressor, would be appropriate).

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Hallucinations can occur in any sensory modality (i.e., visual, olfactory, gustatory, tactile, or auditory), but certain etiological factors are likely to evoke specific hallucinatory phenomena. Olfactory hallucinations are suggestive of temporal lobe epilepsy, for example. Hallucinations may vary from simple and unformed to highly complex and organized, depending on etiological and environmental factors. Psychotic disorder due to another medical condition is generally not diagnosed if the individual maintains reality testing for the hallucinations and appreciates that they result from the medical condition. Delusions may have a variety of themes, including somatic, grandiose, religious, and, most commonly, persecutory. On the whole, however, associations between delusions and particular medical conditions appear to be less specific than is the case for hallucinations.

Although there are no infallible guidelines to determine whether the psychotic disturbance is etiologically attributable to another medical condition, three considerations can provide some guidance: biological plausibility, temporality, and typicality. First, the presence of a medical condition that has the potential to cause psychotic symptoms through a putative physiological mechanism (e.g., severe, generalized infection; porphyria; lupus; temporal lobe epilepsy) must be identified (biological plausibility). The second consideration is whether there is a temporal association between the onset, exacerbation, or remission of the medical condition and that of the psychotic disturbance (temporality). The third consideration in favor of a medical etiology of the psychotic symptoms is the presence of features that would be atypical for an independent psychotic disorder (e.g., atypical age at onset, presence of visual or olfactory hallucinations) (typicality). Finally, causes of psychotic symptoms other than the physiological effects of a medical condition need to be considered and ruled out (e.g., substance/medication-induced psychotic disorder, psychotic symptoms occurring as side effects of the treatment of the medical condition).

The temporal association of the onset or exacerbation of the medical condition offers the greatest diagnostic certainty that the delusions or hallucinations are attributable to a medical condition. Additional factors may include concomitant treatments for the underlying medical condition that confer a risk for psychosis independently, such as steroid treatment for autoimmune disorders.

The diagnosis of psychotic disorder due to another medical condition depends on the clinical condition of each individual, and the diagnostic tests will vary according to that condition. A wide variety of medical conditions may cause psychotic symptoms. These include neurological conditions (e.g., neoplasms, cerebrovascular disease, Huntington's disease, Parkinson's disease, multiple sclerosis, epilepsy, auditory or visual nerve injury or impairment, deafness, migraine, central nervous system infections), endocrine conditions (e.g., hyper- and hypothyroidism, hyper- and hypoparathyroidism, hyper- and hypoadrenocorticism), metabolic conditions (e.g., hypoxia, hypercarbia, hypoglycemia), vitamin B_{12} deficiency, fluid or electrolyte imbalances, hepatic or renal diseases, and autoimmune disorders with central nervous system involvement (e.g., systemic lupus erythematosus, *N*-methyl-D-aspartate [NMDA] receptor autoimmune encephalitis). The associated physical examination findings, laboratory findings, and patterns of prevalence or onset reflect the etiological medical condition.

Prevalence

Prevalence rates for psychotic disorder due to another medical condition are difficult to estimate given the wide variety of underlying medical etiologies. Lifetime prevalence has been estimated to range from 0.21% to 0.54% in studies in Sweden and Finland. When the prevalence findings are stratified by age group, individuals older than 65 years have a significantly greater prevalence of 0.74% compared with those in younger age groups in Finland. Rates of psychosis also vary according to the underlying medical condition; conditions most commonly associated with psychosis include untreated endocrine and metabolic disorders, autoimmune disorders (e.g., systemic lupus erythematosus, NMDA receptor autoimmune encephalitis), or temporal lobe epilepsy. Psychosis attributable to epilepsy

has been further differentiated into ictal, postictal, and interictal psychosis. The most common of these is postictal psychosis, observed in 2%–7.8% of individuals with epilepsy. Among older individuals, there may be a higher prevalence of the disorder in women, although additional sex- or gender-related features are not clear and vary considerably with the sex and gender distributions of the underlying medical conditions. An estimated 60% of older individuals with new-onset psychosis have a medical etiology for their psychotic symptoms.

Development and Course

Psychotic disorder due to another medical condition may be a single transient state or it may be recurrent, cycling with exacerbations and remissions of the underlying medical condition. Although treatment of the underlying medical condition often results in a resolution of the psychosis, this is not always the case, and psychotic symptoms may persist long after the medical event (e.g., psychotic disorder due to focal brain injury). In the context of chronic conditions such as multiple sclerosis or chronic interictal psychosis of epilepsy, the psychosis may assume a long-term course.

The expression of psychotic disorder due to another medical condition does not differ substantially in phenomenology depending on age at occurrence. However, older age groups have a higher prevalence of the disorder, which is most likely due to the increasing medical burden associated with advanced age and the cumulative effects of deleterious exposures and age-related processes (e.g., atherosclerosis). The nature of the underlying medical conditions is likely to change across the lifespan, with younger age groups more affected by epilepsy, head trauma, autoimmune, and neoplastic diseases of early to midlife, and older age groups more affected by a neurodegenerative disease (e.g., Alzheimer’s), stroke disease, anoxic events, and multiple system comorbidities. Underlying factors with increasing age, such as preexisting cognitive impairment as well as vision and hearing impairments, may incur a greater risk for psychosis, possibly by serving to lower the threshold for experiencing psychosis.

Risk and Prognostic Factors

Course modifiers. Identification and treatment of the underlying medical condition has the greatest impact on course, although preexisting central nervous system injury may confer a worse course outcome (e.g., head trauma, cerebrovascular disease).

Association With Suicidal Thoughts or Behavior

Suicide risk in the context of psychotic disorder due to another medical condition is not clearly delineated, although certain conditions such as epilepsy and multiple sclerosis are associated with increased rates of suicide, which may be further increased in the presence of psychosis.

Functional Consequences of Psychotic Disorder Due to Another Medical Condition

Functional disability is typically severe in the context of psychotic disorder due to another medical condition but will vary considerably by the type of condition and likely improve with successful resolution of the condition.

Differential Diagnosis

Delirium and major or mild neurocognitive disorder. Hallucinations and delusions commonly occur in the context of a delirium; a separate diagnosis of psychotic disorder due to

another medical condition is not given if the delusions and/or hallucinations occur exclusively during the course of a delirium. On the other hand, a diagnosis of psychotic disorder due to another medical condition may be given in addition to a diagnosis of major or mild neurocognitive disorder if the delusions or hallucinations are judged to be a physiological consequence of the pathological process causing the neurocognitive disorder (e.g., psychotic disorder due to Lewy body disease, with delusions).

Substance/medication-induced psychotic disorder. If there is evidence of recent or prolonged substance use (including medications with psychoactive

effects), withdrawal from a substance or medication that can cause psychotic symptoms on withdrawal, or exposure to a toxin (e.g., LSD [lysergic acid diethylamide] intoxication, alcohol withdrawal), a substance/medication-induced psychotic disorder should be considered. Symptoms that occur during or shortly after (i.e., within 4 weeks) of substance intoxication or withdrawal or after medication use may be especially indicative of a substance-induced psychotic disorder, depending on the character, duration, or amount of the substance used. If the clinician has ascertained that the disturbance is due to both a medical condition and substance use, both diagnoses (i.e., psychotic disorder due to another medical condition and substance/medication-induced psychotic disorder) can be given.

Psychotic disorder. Psychotic disorder due to another medical condition must be distinguished from a psychotic disorder that is not due to another medical condition (e.g., schizophrenia, delusional disorder, schizoaffective disorder) or a major depressive or bipolar disorder, with psychotic features. In psychotic disorders and in depressive or bipolar disorders, with psychotic features, no specific and direct causative physiological mechanisms associated with a medical condition can be demonstrated. Late age at onset and the absence of a personal or family history of schizophrenia or delusional disorder suggest the need for a thorough assessment to rule out the diagnosis of psychotic disorder due to another medical condition. Auditory hallucinations that involve voices speaking complex sentences are more characteristic of schizophrenia than of psychotic disorder due to a medical condition. While certain symptoms suggest a medical or toxic etiology (e.g., visual or olfactory hallucinations, dreamlike quality of delusions [individual as uninvolved observer]), there are no pathognomonic signs or symptoms that unequivocally point clinicians either way. Visual hallucinations are not uncommon in schizophrenia or bipolar disorder, and olfactory hallucinations (e.g., unpleasant smells) are also consistent with a diagnosis of schizophrenia. Thus, clinicians should not give undue weight to any one particular hallucination alone when deciding between a psychiatric and a medical cause for psychopathology.

Comorbidity

Psychotic disorder due to another medical condition in individuals older than 80 years is associated with concurrent major neurocognitive disorder (dementia). Alzheimer's disease is commonly accompanied by psychosis, and psychosis is a defining feature in Lewy body disease.

Catatonia

Catatonia can occur in the context of several disorders, including neurodevelopmental, psychotic, bipolar, and depressive disorders, and other medical conditions (e.g., cerebral folate deficiency, rare autoimmune and paraneoplastic disorders). The manual does not treat catatonia as an independent class but recognizes a) catatonia associated with another

mental disorder (i.e., a neurodevelopmental, psychotic disorder, a bipolar disorder, a depressive disorder, or other mental disorder), b) catatonic disorder due to another medical condition, and c) unspecified catatonia.

Catatonia is defined by the presence of 3 or more of 12 psychomotor features in the diagnostic criteria for catatonia associated with another mental disorder and catatonic disorder due to another medical condition. The essential feature of catatonia is a marked psychomotor disturbance that may involve decreased motor activity, decreased engagement during interview or physical examination, or excessive and peculiar motor activity. The clinical presentation of catatonia can be puzzling, as the psychomotor disturbance may range from marked unresponsiveness to marked agitation. Motoric immobility may be severe (stupor) or moderate (catalepsy and waxy flexibility). Similarly, decreased engagement may be severe (mutism) or moderate (negativism). Excessive and peculiar motor behaviors can be complex (e.g., stereotypy) or simple (agitation) and may include echolalia and echopraxia. In extreme cases, the same individual may wax and wane between decreased and excessive motor activity. The seemingly opposing clinical features, variable manifestations of the diagnosis, and overemphasis in teaching on rare, severe signs such as waxy flexibility contribute to a lack of awareness and decreased recognition of catatonia. During severe

stages of catatonia, the individual may need careful supervision to avoid self-harm or harming others. There are potential risks from malnutrition, exhaustion, thromboembolism, pressure ulcers, muscle contractions, hyperpyrexia and self-inflicted injury.

Catatonia Associated With Another Mental Disorder (Catatonia Specifier)

F06.1

A. The clinical picture is dominated by three (or more) of the following symptoms:

1. Stupor (i.e., no psychomotor activity; not actively relating to environment).
2. Catalepsy (i.e., passive induction of a posture held against gravity).
3. Waxy flexibility (i.e., slight, even resistance to positioning by examiner).
4. Mutism (i.e., no, or very little, verbal response [exclude if known aphasia]).
5. Negativism (i.e., opposition or no response to instructions or external stimuli).
6. Posturing (i.e., spontaneous and active maintenance of a posture against gravity).
7. Mannerism (i.e., odd, circumstantial caricature of normal actions).
8. Stereotypy (i.e., repetitive, abnormally frequent, non-goal-directed movements).
9. Agitation, not influenced by external stimuli.
10. Grimacing.
11. Echolalia (i.e., mimicking another’s speech).
12. Echopraxia (i.e., mimicking another’s movements).

Coding note: Indicate the name of the associated mental disorder when recording the name of the condition (i.e., F06.1 catatonia associated with major depressive disorder). Code first the associated mental disorder (e.g., neurodevelopmental disorder, brief psychotic disorder, schizophreniform disorder, schizophrenia, schizoaffective disorder, bipolar disorder, major depressive disorder, or other mental disorder) (e.g., F25.1 schizoaffective disorder, depressive type; F06.1 catatonia associated with schizoaffective disorder).

Diagnostic Features

Catatonia associated with another mental disorder (catatonia specifier) may be used when criteria are met for catatonia during the course of a neurodevelopmental, psychotic, bipolar, depressive, or other mental disorder. The catatonia specifier is appropriate when the clinical picture is characterized by marked psychomotor disturbance and involves at least three of the 12 diagnostic features listed in Criterion A. Catatonia is typically diagnosed in an inpatient setting and occurs in up to 35% of individuals with schizophrenia, but the majority of catatonia cases involve individuals with depressive or bipolar disorders. Meta-analysis of clinical samples indicated that approximately 9% of patients had catatonia. Before the catatonia specifier is used in neurodevelopmental, psychotic, bipolar, depressive, or other mental disorders, a wide variety of other medical conditions need to be ruled out; these conditions include, but are not limited to, medical conditions due to infectious, metabolic, or neurological conditions (see “Catatonic Disorder Due to Another Medical Condition”). Catatonia can also be a side effect of a medication (see the chapter “Medication-Induced Movement Disorders and Other Adverse Effects of Medication”). Because of the seriousness of the complications, particular attention should be paid to the possibility that the catatonia is attributable to G21.0 neuroleptic malignant syndrome.

Culture-Related Diagnostic Issues

The association between catatonia and mood disorders has been found in a wide range of cultural contexts.

Catatonic Disorder Due to Another Medical Condition

Diagnostic Criteria F06.1

A. The clinical picture is dominated by three (or more) of the following symptoms:
   1. Stupor (i.e., no psychomotor activity; not actively relating to environment).
   2. Catalepsy (i.e., passive induction of a posture held against gravity).
   3. Waxy flexibility (i.e., slight, even resistance to positioning by examiner).
   4. Mutism (i.e., no, or very little, verbal response [**Note:** not applicable if there is an established aphasia]).
   5. Negativism (i.e., opposition or no response to instructions or external stimuli).
   6. Posturing (i.e., spontaneous and active maintenance of a posture against gravity).
   7. Mannerism (i.e., odd, circumstantial caricature of normal actions).
   8. Stereotypy (i.e., repetitive, abnormally frequent, non-goal-directed movements).
   9. Agitation, not influenced by external stimuli.
   10. Grimacing.
   11. Echolalia (i.e., mimicking another's speech).
   12. Echopraxia (i.e., mimicking another's movements).

B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.
C. The disturbance is not better explained by another mental disorder (e.g., a manic episode).
D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Coding note: Include the name of the medical condition in the name of the mental disorder (e.g., F06.1 catatonic disorder due to hepatic encephalopathy). The other medical condition should be coded and listed separately immediately before the catatonic disorder due to the medical condition (e.g., K72.90 hepatic encephalopathy; F06.1 catatonic disorder due to hepatic encephalopathy).

Diagnostic Features

The essential feature of catatonic disorder due to another medical condition is the presence of catatonia that is judged to be attributed to the physiological effects of another medical condition. Catatonia can be diagnosed by the presence of at least 3 of the 12 clinical features in Criterion A. There must be evidence from the history, physical examination, or laboratory findings that the catatonia is attributable to another medical condition (Criterion B). The diagnosis is not given if the catatonia is better explained by another mental disorder (e.g., manic episode) (Criterion C) or if it occurs exclusively during the course of a delirium (Criterion D).

Associated Features

A variety of medical conditions may cause catatonia, especially neurological conditions (e.g., neoplasms, head trauma, cerebrovascular disease, encephalitis) and metabolic conditions (e.g., hypercalcemia, hepatic encephalopathy, homocystinuria, diabetic ketoacidosis). The associated physical examination findings, laboratory findings, and patterns of prevalence and onset reflect those of the etiological medical condition.

Differential Diagnosis

A separate diagnosis of catatonic disorder due to another medical condition is not given if the catatonia occurs exclusively during the course of a delirium or neuroleptic malignant syndrome. However, even though a separate diagnosis of catatonia cannot be made, research suggests that catatonia symptoms occur in a significant proportion of delirium cases. If the individual is currently taking neuroleptic medication, consideration should be given to medication-induced movement disorders (e.g., abnormal positioning may be due to neuroleptic-induced acute dystonia) or neuroleptic malignant syndrome (e.g., catatonic-like features may be present, along with associated vital sign and/or laboratory abnormalities). Catatonic symptoms may be present in any of the following five psychotic disorders: brief psychotic disorder, schizophreniform disorder, schizophrenia, schizoaffective disorder, and substance/medication-induced psychotic disorder. It may also be present in some of the neurodevelopmental disorders, in all of the bipolar and depressive disorders, and in other mental disorders.

Unspecified Catatonia

This category applies to presentations in which symptoms characteristic of catatonia cause clinically significant distress or impairment in social, occupational, or other important areas of functioning but either the nature of the underlying mental disorder or other medical condition is unclear, full criteria for catatonia are not met, or there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Coding note: Code first **R29.818** other symptoms involving nervous and musculoskeletal systems, followed by **F06.1** unspecified catatonia.

Other Specified Schizophrenia Spectrum and Other Psychotic Disorder

F28

This category applies to presentations in which symptoms characteristic of a schizophrenia spectrum and other psychotic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the schizophrenia spectrum and other psychotic disorders diagnostic class. The other specified schizophrenia spectrum and other psychotic disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific schizophrenia spectrum and other psychotic disorder. This is done by recording “other specified schizophrenia spectrum and other psychotic disorder” followed by the specific reason (e.g., “persistent auditory hallucinations”).

Examples of presentations that can be specified using the “other specified” designation include the following:

1. **Persistent auditory hallucinations** occurring in the absence of any other features.
2. **Delusions with significant overlapping mood episodes:** This includes persistent delusions with periods of overlapping mood episodes that are present for a substantial portion of

the delusional disturbance (such that the criterion stipulating only brief mood disturbance in delusional disorder is not met).

3. **Attenuated psychosis syndrome:** This syndrome is characterized by psychotic-like symptoms that are below a threshold for full psychosis (e.g., the symptoms are less severe and more transient, and insight is relatively maintained).
4. **Delusional symptoms in the context of relationship with an individual with prominent delusions:** In the context of a relationship, the delusional material from the individual with a psychotic disorder provides content for the same delusions held by the other person who may not otherwise have symptoms that meet criteria for a psychotic disorder.

Unspecified Schizophrenia Spectrum and Other Psychotic Disorder

F29

This category applies to presentations in which symptoms characteristic of a schizophrenia spectrum and other psychotic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the schizophrenia spectrum and other psychotic disorders diagnostic class. The unspecified schizophrenia spectrum and other psychotic disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific schizophrenia spectrum and other psychotic disorder and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Bipolar and Related Disorders

Bipolar and related disorders are found between the chapters on schizophrenia spectrum and other psychotic disorders and depressive disorders in DSM-5-TR in recognition of their place as a bridge between those two diagnostic classes in terms of symptomatology, family history, and genetics. The diagnoses included in this chapter are bipolar I disorder, bipolar II disorder, cyclothymic disorder, substance/medication-induced bipolar and related disorder, bipolar and related disorder due to another medical condition, other specified bipolar and related disorder, and unspecified bipolar and related disorder.

The bipolar I disorder criteria represent the modern understanding of the classic manic-depressive disorder or affective psychosis described in the nineteenth century, differing from that classic description only to the extent that neither psychosis nor the lifetime experience of a major depressive episode is a requirement. However, the vast majority of individuals whose symptoms meet the criteria for a fully syndromal manic episode also experience major depressive episodes during the course of their lives.

Bipolar II disorder, requiring the lifetime experience of at least one major depressive episode and at least one hypomanic episode (but no history of mania), is no longer thought to be a less severe condition than bipolar I disorder, largely because of the burden of depression in bipolar II disorder and because the instability of mood experienced by individuals with bipolar II disorder is often accompanied by serious impairment in work and social functioning.

The diagnosis of cyclothymic disorder is given to adults who experience at least 2 years (for children, a full year) of both hypomanic and depressive periods without ever fulfilling the criteria for an episode of mania, hypomania, or major depression.

A large number of substances of abuse, some prescribed medications, and several medical conditions can be associated with manic-like phenomena. This fact is recognized in the diagnoses of substance/medication-induced bipolar and related disorder and bipolar and related disorder due to another medical condition.

The recognition that there are individuals who experience bipolar-like phenomena with symptoms that do not meet the criteria for bipolar I, bipolar II, or cyclothymic disorder is reflected in the availability of the other specified bipolar and related disorder category. Specific criteria for a disorder involving short-duration hypomania are provided in Section III in the hope of encouraging further study of this presentation of bipolar disorder symptomatology and its course.

Bipolar I Disorder

Diagnostic Criteria

For a diagnosis of bipolar I disorder, it is necessary to meet the following criteria for a manic episode. The manic episode may have been preceded by and may be followed by hypomanic or major depressive episodes.

Manic Episode

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy, lasting at least 1 week and present most of the day, nearly every day (or any duration if hospitalization is necessary).

B. During the period of mood disturbance and increased energy or activity, three (or more) of the following symptoms (four if the mood is only irritable) are present to a significant degree and represent a noticeable change from usual behavior:

1. Inflated self-esteem or grandiosity.
2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep).
3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed.
6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation (i.e., purposeless non-goal-directed activity).
7. Excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

C. The mood disturbance is sufficiently severe to cause marked impairment in social or occupational functioning or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.

D. The episode is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment) or another medical condition.

   Note: A full manic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a manic episode and, therefore, a bipolar I diagnosis.

Note: Criteria A–D constitute a manic episode. At least one lifetime manic episode is required for the diagnosis of bipolar I disorder.

Hypomanic Episode

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy, lasting at least 4 consecutive days and present most of the day, nearly every day.

B. During the period of mood disturbance and increased energy and activity, three (or more) of the following symptoms (four if the mood is only irritable) have persisted, represent a noticeable change from usual behavior, and have been present to a significant degree:

   1. Inflated self-esteem or grandiosity.
   2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep).
   3. More talkative than usual or pressure to keep talking.
   4. Flight of ideas or subjective experience that thoughts are racing.
   5. Distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed.
   6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation.
   7. Excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

C. The episode is associated with an unequivocal change in functioning that is uncharacteristic of the individual when not symptomatic.

D. The disturbance in mood and the change in functioning are observable by others.

E. The episode is not severe enough to cause marked impairment in social or occupational functioning or to

necessitate hospitalization. If there are psychotic features, the episode is, by definition, manic.

F. The episode is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment) or another medical condition.

Note: A full hypomanic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a hypomanic episode diagnosis. However, caution is indicated so that one or two symptoms (particularly increased irritability, edginess, or agitation following antidepressant use) are not taken as sufficient for diagnosis of a hypomanic episode, nor necessarily indicative of a bipolar diathesis.

Note: Criteria A–F constitute a hypomanic episode. Hypomanic episodes are common in bipolar I disorder but are not required for the diagnosis of bipolar I disorder.

Major Depressive Episode

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly attributable to another medical condition.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, or hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).

3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**Note:** In children, consider failure to make expected weight gain.)
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The episode is not attributable to the physiological effects of a substance or another medical condition.

Note: Criteria A–C constitute a major depressive episode. Major depressive episodes are common in bipolar I disorder but are not required for the diagnosis of bipolar I disorder.

Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight

loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.[1]

Bipolar I Disorder

A. Criteria have been met for at least one manic episode (Criteria A–D under "Manic Episode" above).
B. At least one manic episode is not better explained by schizoaffective disorder and is not superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.

Coding and Recording Procedures

The diagnostic code for bipolar I disorder is based on type of current or most recent episode and its status with respect to current severity, presence of psychotic features, and remission status. Current severity and psychotic features are only indicated if full criteria are currently met for a manic or major depressive episode. Remission specifiers are only indicated if the full criteria are not currently met for a manic, hypomanic, or major depressive episode. Codes are as follows:

Bipolar I disorder	Current or most recent episode manic	Current or most recent episode hypomanic*	Current or most recent episode depressed	Current or most recent episode unspecified**
Mild (p. 175)	F31.11	NA	F31.31	NA
Moderate (p. 175)	F31.12	NA	F31.32	NA

Bipolar I disorder	Current or most recent episode manic	Current or most recent episode hypomanic*	Current or most recent episode depressed	Current or most recent episode unspecified**
Severe (p. 175)	F31.13	NA	F31.4	NA
143	F31.2	NA	F31.5	NA
With psychotic features*** (p. 173)				
In partial remission (p. 175)	F31.73	F31.71	F31.75	NA
In full remission (p. 175)	F31.74	F31.72	F31.76	NA
Unspecified	F31.9	F31.9	F31.9	NA

*Severity and psychotic specifiers do not apply; code F31.0 for cases not in remission.
**Severity, psychotic, and remission specifiers do not apply. Code F31.9.
***If psychotic features are present, code the "with psychotic features" specifier irrespective of episode severity.

In recording the name of a diagnosis, terms should be listed in the following order: bipolar I disorder, type of current episode (or most recent episode if bipolar I disorder is in partial or full remission), severity/psychotic/remission specifiers, followed by as many of the following specifiers without codes as apply to the current episode (or the most recent episode if bipolar I disorder is in partial or full remission). **Note:** The specifiers "with rapid cycling" and "with seasonal pattern" describe the pattern of mood episodes.

Specify if:

With anxious distress (pp. 169–170)

With mixed features (pp. 170–171)

With rapid cycling (p. 171)

With melancholic features (pp. 171–172)

With atypical features (pp. 172–173)

With mood-congruent psychotic features (p. 173; *applies to manic episode and/or major depressive episode*)

With mood-incongruent psychotic features (p. 173; *applies to manic episode and/or major depressive episode*)

With catatonia (p. 173). **Coding note:** Use additional code F06.1.

With peripartum onset (pp. 173–174)

With seasonal pattern (pp. 174–175)

Diagnostic Features

Bipolar I disorder is characterized by a clinical course of recurring mood episodes (manic, depressive, and hypomanic), but the occurrence of at least one manic episode is necessary for the diagnosis of bipolar I disorder. The essential feature of a manic episode is a distinct period during which there is an abnormally, persistently elevated, expansive, or irritable mood and persistently increased activity or energy that is present for most of the day, nearly every day, for a period of at least 1 week (or any duration if hospitalization is necessary), accompanied by at least three additional symptoms from Criterion B. If the mood is irritable rather than elevated or expansive, at least four Criterion B symptoms must be present.

Mood in a manic episode is often described as euphoric, excessively cheerful, high, or "feeling on top of the world." In some cases, the mood is of such a highly infectious quality that it is easily recognized as excessive and may be characterized by unlimited and

haphazard enthusiasm for interpersonal, sexual, or occupational interactions. For example, the individual may spontaneously start extensive conversations with strangers in public. Often the predominant mood is irritable rather than elevated, particularly when the individual's wishes are

denied or if the individual has been using substances. Rapid shifts in mood over brief periods of time may occur and are referred to as lability (i.e., the alternation among euphoria, dysphoria, and irritability). In children, happiness, silliness, and “goofiness” are normal in many social contexts; however, if these symptoms are recurrent, inappropriate to the context, and beyond what is expected for the developmental level of the child, they may meet the Criterion A mood requirement of abnormally elevated mood. For the happiness or silliness of a child to meet Criterion A, it must be distinctly increased from the child’s baseline and accompanied by persistently increased activity or energy levels that to those who know the child well are clearly unusual for that child. For a child’s symptoms to meet criteria for a manic episode, the symptoms must also meet Criterion B for mania and must also represent a change from the child’s usual baseline.

During the manic episode, the individual may engage in multiple overlapping new projects. The projects are often initiated with little knowledge of the topic, and nothing seems out of the individual’s reach. The increased activity or energy levels may manifest at unusual hours of the day, such as during the individual’s normal sleep phase.

Inflated self-esteem is typically present, ranging from uncritical self-confidence to marked grandiosity, and may reach delusional proportions (Criterion B1). Despite lack of any particular experience or talent, the individual may embark on complex tasks such as writing a novel or seeking publicity for some impractical invention. Grandiose delusions (e.g., of having a special relationship to a famous person) are common. In children, overestimation of abilities and belief that, for example, they are the best at a sport or the smartest in the class is normal; however, when such beliefs are present despite clear evidence to the contrary or the child attempts feats that are clearly dangerous and, most important, represent a change from the child’s normal behavior, the grandiosity criterion should be considered satisfied.

One of the most common features is a decreased need for sleep (Criterion B2), which is distinct from insomnia (during which the individual wants to sleep or feels the need to sleep but is unable to). The individual may sleep little, if at all, or may awaken several hours earlier than usual, feeling rested and full of energy. When the sleep disturbance is severe, the

individual may go for days without sleep, yet not feel tired. Often decreased need for sleep heralds the onset of a manic episode.

Speech can be rapid, pressured, loud, and difficult to interrupt (Criterion B3). Individuals may talk continuously and without regard for others' wishes to communicate, often in an intrusive manner or without concern for the relevance of what is said. Speech is sometimes characterized by jokes, puns, amusing irrelevancies, and theatricality, with dramatic mannerisms, singing, and excessive gesturing. Loudness and forcefulness of speech often become more important than what is conveyed. If the individual's mood is more irritable than expansive, speech may be marked by complaints, hostile comments, or angry tirades, particularly if attempts are made to interrupt the individual. Both Criterion A and Criterion B symptoms may be accompanied by symptoms of the opposite (i.e., depressive) pole (see "with mixed features" specifier, pp. 170–171).

Often the individual's thoughts race at a rate faster than can be expressed through speech (Criterion B4). Frequently there is flight of ideas evidenced by a nearly continuous flow of accelerated speech, with abrupt shifts from one topic to another. When flight of ideas is severe, speech may become disorganized, incoherent, and particularly distressing to the individual. Sometimes thoughts are experienced as so crowded that it is very difficult to speak.

Distractibility (Criterion B5) is evidenced by an inability to censor immaterial external stimuli (e.g., the interviewer's attire, background noises or conversations, furnishings in the room) and often prevents individuals experiencing mania from holding a rational conversation or attending to instructions.

The increase in goal-directed activity (Criterion B6) often consists of excessive planning and participation in multiple activities, including sexual, occupational, political, or religious activities. Increased sexual drive, fantasies, and behavior are often present. Individuals in a manic episode usually show increased sociability (e.g., renewing old acquaintances or calling or contacting friends or even strangers), without regard to the intrusive, domineering, and demanding nature of these interactions. They

often also display psychomotor agitation or restlessness (i.e., purposeless activity) by pacing or by holding multiple conversations simultaneously. Some individuals write excessive letters, e-mails, text messages, and so forth, on many different topics to friends, public figures, or the media.

The increased activity criterion can be difficult to ascertain in children; however, when the child takes on many tasks simultaneously, starts devising elaborate and unrealistic plans for projects, develops previously absent and developmentally inappropriate sexual preoccupations (not accounted for by sexual abuse or exposure to sexually explicit material), then Criterion B might be met based on clinical judgment. It is essential to determine whether the behavior represents a change from the child's baseline behavior; occurs most of the day, nearly every day for the requisite time period; and occurs in temporal association with other symptoms of mania.

The expansive mood, excessive optimism, grandiosity, and poor judgment often lead to reckless involvement in activities such as spending sprees, giving away possessions, reckless driving, foolish business investments, and sexual indiscretions that are unusual for the individual, even though these activities are likely to have catastrophic consequences (Criterion B7). The individual may purchase many unneeded items without the money to pay for them and, in some cases, give them away. Sexual indiscretions may include infidelity or indiscriminate sexual encounters with strangers, often disregarding the risk of sexually transmitted diseases or interpersonal consequences.

The manic episode must result in marked impairment in social or occupational functioning (e.g., financial losses, loss of employment, school failure, divorce) or require hospitalization to prevent harm to self or others (e.g., physical exhaustion or hyperthermia from manic excitement, self-injurious behavior). By definition, the presence of psychotic features during a manic episode also satisfies Criterion C.

Manic symptoms or syndromes that are attributable to the direct physiological effects of a drug of abuse (e.g., in the context of cocaine or amphetamine intoxication), the side effects of medications or treatments (e.g., steroids, L-dopa, antidepressants, stimulants), or another medical condition do not count toward the diagnosis of bipolar I disorder. However,

a fully syndromal manic episode that arises during treatment (e.g., with medications, electroconvulsive therapy, light therapy) and persists beyond the physiological effect of the inducing agent (e.g., after a medication is fully out of the individual's system or the effects of electroconvulsive therapy would be expected to have dissipated completely) is sufficient evidence for a manic episode that is considered due to bipolar I disorder (Criterion D). Caution is indicated so that one or two symptoms (particularly increased irritability, edginess, or agitation following antidepressant use) are not taken as sufficient for diagnosis of a manic or hypomanic episode, nor necessarily an indication of a bipolar disorder diathesis. Although not essential to a diagnosis of bipolar I disorder, hypomanic or depressive episodes often precede or follow a manic episode. Full descriptions of the diagnostic features of a hypomanic episode may be found within the text for bipolar II disorder, and the features of a major depressive episode are described within the text for major depressive disorder.

Associated Features

During a manic episode, individuals often do not perceive that they are ill or in need of treatment and vehemently resist efforts to be treated. Individuals may change their dress, makeup, or personal appearance to a more sexually suggestive or flamboyant style. Some perceive a sharper sense of smell, hearing, or vision. Gambling and antisocial behaviors

may accompany the manic episode. Mood may shift very rapidly to anger or depression; some individuals may become hostile and physically threatening to others and, when delusional, become physically assaultive or suicidal. Serious consequences of a manic episode (e.g., involuntary hospitalization, difficulties with the law, serious financial difficulties) often result from poor judgment, loss of insight, and hyperactivity. Depressive symptoms occur in some 35% of manic episodes (see "with mixed features" specifier, p. 170), and mixed features are associated with poorer outcome

and increased suicide attempts. Bipolar I disorder is also associated with significant decrements in quality of life and well-being.

Trait-like features associated with the diagnosis include hyperthymic, depressive, cyclothymic, anxious, and irritable temperaments, sleep and circadian rhythm disturbances, reward sensitivity, and creativity. Having a first-degree relative with bipolar disorder increases the risk of diagnosis approximately 10-fold.

Prevalence

The 12-month prevalence of DSM-5 bipolar I disorder in a nationally representative U.S. adult sample was 1.5% and did not differ between men (1.6%) and women (1.5%). Compared with non-Hispanic Whites, prevalence of bipolar I disorder appears to be higher among Native Americans and lower among African Americans, Hispanics, and Asians/Pacific Islanders. Twelve-month prevalence of DSM-IV bipolar I disorder across 11 countries ranged from 0.0% to 0.6% and was greater in high-income countries than in low- and middle-income countries, except in Japan, where prevalence was low (0.01%). The lifetime prevalence ratio in men to women is approximately 1.1:1.

Development and Course

The peak age at onset of bipolar I disorder across studies is between 20 and 30 years, but onset occurs throughout the life cycle. In the United States, mean age at onset of DSM-5 bipolar I disorder is 22 years and slightly younger for women (21.5 years) than for men (23.0 years). In a comparison of six international sites, median age at onset of DSM-IV-TR bipolar I disorder was 24.3 years. Special considerations are necessary to apply the diagnosis in children. Because children of the same chronological age may be at different developmental stages, it is difficult to define with precision what is “normal” or “expected” at any given point. Therefore, each child should be judged according to his or her own baseline in determining whether a particular behavior is “normal” or evidence of a manic episode. Although age at first onset may occur in the 60s or 70s, onset of manic symptoms (e.g., sexual or social disinhibition) in late mid-life or late-life

should prompt consideration of medical conditions (e.g., frontotemporal neurocognitive disorder) and of substance ingestion or withdrawal.

More than 90% of individuals who have a single manic episode go on to have recurrent mood episodes. Approximately 60% of manic episodes occur immediately before a major depressive episode. Individuals with bipolar I disorder who have multiple (four or more) mood episodes (major depressive, manic, or hypomanic) occurring in the prior 12 months receive the specifier “with rapid cycling,” a common variant associated with poorer outcomes. About half of individuals diagnosed with bipolar disorder exhibit a predominant polarity (relapse tending to be either depressive or manic), with one international study of bipolar I disorder finding 31.3% with predominant mania, 21.4% with predominant depression, and 47.3% without polarity predominance.

The course of bipolar I disorder is highly heterogeneous. Some patterns have been noted across episodes (e.g., a manic episode with psychotic features may be associated with psychotic features in subsequent manic episodes). Polarity of first episode tends to be associated with predominant polarity of future episodes and clinical features (e.g., depressive onset is associated with greater density of depressive episodes and suicidal behavior). The

presence of mixed features in a manic episode is associated with a poorer prognosis, poorer lithium response, and suicidal behavior.

Risk and Prognostic Factors

Environmental. Childhood adversity (including early emotional trauma, parental psychopathology, and family conflict) is a known risk factor for bipolar disorder and appears to predispose to early onset of bipolar disorder. Childhood adversity is also associated with poorer prognosis and a worse clinical picture that may include medical or psychiatric comorbidities, suicide, and associated psychotic features. More proximally, recent life stress and other negative life events increase depressive relapse risk in individuals diagnosed with bipolar disorder, whereas manic relapse appears

to be specifically linked to goal-attainment life events (e.g., getting married, completing a degree). Cannabis and other substance use is associated with exacerbation of manic symptoms among individuals diagnosed with bipolar disorder, as well as first onset of manic symptoms in the general population. There is some evidence that becoming married is less common among individuals with bipolar disorder than in the general population and that a diagnosis of bipolar disorder is associated with being previously as opposed to currently married.

Genetic and physiological. Genetic processes strongly affect predisposition to bipolar disorder, with heritability estimates around 90% in some twin studies. Risk of bipolar disorder in the general population is around 1%, while risk in a first-degree relative is 5%–10%. However, monozygotic concordance rates are significantly less than 100% (40%–70%), indicating that much risk is left unexplained by genes alone. The mechanism of heritability is not Mendelian, and involves multiple genes (or more complex genetic mechanisms) of small effect, interacting with each other, the environment, and random factors. Emerging genetic findings suggest that mania- and depression-proneness are inherited separately, and bipolar disorder shares a genetic origin with schizophrenia.

Culture-Related Diagnostic Issues

Bipolar I disorder symptoms tend to be consistent across cultural contexts, but some variation exists in symptom expression and interpretation. For example, individuals from different cultural backgrounds with bipolar I disorder, with psychotic features, may vary in the prevalence of flight of ideas or types of delusions (e.g., grandiose, persecutory, sexual, religious, or somatic). Cultural factors may affect disorder prevalence. For example, countries with reward-oriented cultural values that place significance on individual pursuit of reward have a relatively higher prevalence of bipolar disorder. In the United States, individuals with bipolar disorder had an earlier age at onset than those in Europe and were more likely to have a family history of psychiatric disorder.

Culture also influences clinician diagnostic practices regarding bipolar disorder. Compared with non-Latinx Whites in the United States, African Americans with bipolar I disorder are at higher risk of being misdiagnosed

with schizophrenia. Possible reasons include underrecognition of mood symptoms, cultural and linguistic misunderstanding between clinicians and the individuals presenting for treatment (e.g., misinterpretation of cultural mistrust as paranoia), more florid psychotic symptoms at presentation due to delay in receiving services, and diagnoses based on shorter clinical assessments. These factors may result in discriminatory misdiagnosis of schizophrenia, particularly in African Americans with mood disorders who present with psychotic features.

Sex- and Gender-Related Diagnostic Issues

Women may be more likely to experience rapid cycling and mixed states, and to have patterns of comorbidity that differ from those of men, including higher rates of lifetime eating

disorders. Women with bipolar I or II disorder are more likely to experience depressive symptoms than are men. They also have a higher lifetime risk of alcohol use disorder than do men and a much greater likelihood of alcohol use disorder than do women in the general population.

Some women with bipolar disorder experience exacerbation of mood symptoms during the premenstrual time period, and this has been associated with a worse course of illness. Many women with bipolar disorder also report severe emotional disturbances during perimenopause when estrogen levels are decreasing. There does not appear to be an increased risk of mood episodes in pregnant women with bipolar disorder except in those who discontinue medications for pregnancy. In contrast, there is strong and consistent evidence for an increased risk of mood episodes (both depression and mania) in women with bipolar I disorder in the postpartum period. The specifier “with peripartum onset” should be used for mood episodes that begin during pregnancy or within 4 weeks of delivery. “Postpartum psychosis” typically resembles a manic or mixed mood episode with psychotic symptoms and is strongly associated with bipolar I disorder.

Association With Suicidal Thoughts or Behavior

The lifetime risk of suicide in individuals with bipolar disorder is estimated to be 20- to 30-fold greater than in the general population. An estimated 5%–6% of individuals with bipolar disorders die by suicide. While suicide attempts are higher in women, lethal suicide is more common in men with bipolar disorder. A past history of suicide attempt and percent days spent depressed in the past year are associated with greater risk of suicide attempts or completions. Nearly half of individuals whose symptoms meet criteria for bipolar disorder have an alcohol use disorder, and those with both disorders are at greater risk for suicide attempt and suicide death.

Functional Consequences of Bipolar I Disorder

Approximately 30% of individuals with bipolar disorder show severe impairment in work role functioning, although many individuals return to a fully functional level between episodes. Functional recovery lags substantially behind recovery from symptoms, especially with respect to occupational recovery, resulting in lower socioeconomic status despite equivalent levels of education when compared with the general population. Cognitive impairments persist through the lifespan, even during euthymic periods, and may contribute to vocational and interpersonal difficulties. Higher level of self-perceived stigma is associated with lower level of functioning.

Differential Diagnosis

Major depressive disorder. There is a risk of misdiagnosing bipolar I disorder as unipolar depression because of the prominence of depression in the presentation of bipolar I disorder: 1) the first episode of bipolar disorder is often depressive, 2) depressive symptoms are the most frequent symptoms experienced across the long-term course of bipolar I disorder, and 3) the problem for which individuals typically seek help is depression. When the individual presents in an episode of major depression, it is therefore important to actively probe for a history of mania or hypomania. Factors that might indicate that the diagnosis is bipolar I disorder rather than major depressive disorder in an individual presenting with a current depressive episode include family history of bipolar disorder, onset of illness in early 20s, numerous past episodes, presence of psychotic symptoms, and a

history of lack of response to antidepressant treatment or the emergence of a manic episode during antidepressant treatment (e.g., medication, electroconvulsive therapy).

Other bipolar disorders. Bipolar II disorder, cyclothymic disorder, and other specified bipolar and related disorder are similar to bipolar I disorder by virtue of their including

periods of hypomanic symptoms in their presentations but are differentiated from bipolar I disorder by the absence of any manic episodes.

Generalized anxiety disorder, panic disorder, posttraumatic stress disorder, or other anxiety disorders.
A careful history of symptoms is needed to differentiate generalized anxiety disorder from bipolar disorder, as anxious ruminations may be mistaken for racing thoughts (and vice versa), and efforts to minimize anxious feelings may be taken as impulsive behavior. Similarly, symptoms of posttraumatic stress disorder need to be differentiated from bipolar disorder. It is helpful to assess the episodic nature of the symptoms described (classical bipolar I is episodic), as well as to consider symptom triggers, in making this differential diagnosis.

Bipolar and related disorder due to another medical condition. The diagnosis of bipolar and related disorder due to another medical condition should be made instead of bipolar I disorder if the manic episodes are judged, based on history, laboratory findings, or physical examination, to be the direct physiological consequence of another medical condition (e.g., Cushing's disease, multiple sclerosis).

Substance/medication-induced bipolar and related disorder. A substance/medication-induced bipolar and related disorder is distinguished from bipolar I disorder by the fact that a substance (e.g., stimulants, phencyclidine) or medication (e.g., steroids) is judged to be etiologically related to the manic episode. Because individuals with a manic episode have a tendency to overuse substances during an episode, it is important to determine whether the substance use is a consequence of a primary manic episode or whether the manic-like episode has been caused by the substance

use. In some cases, a definitive diagnosis may involve establishing that the manic symptoms remain once the individual is no longer using the substance. Note that manic episodes emerging in the context of treatment with an antidepressant medication but that persist at a fully syndromal level beyond the physiological effect of the medication warrant a diagnosis of bipolar I disorder rather than substance/medication-induced bipolar and related disorder.

Schizoaffective disorder. Schizoaffective disorder is characterized by periods in which manic and major depressive episodes are concurrent with the active phase symptoms of schizophrenia and periods in which delusions or hallucinations occur for at least 2 weeks in the absence of a manic or major depressive episode. The diagnosis is "bipolar I disorder, with psychotic features" if the psychotic symptoms have occurred exclusively during manic and major depressive episodes.

Attention-deficit/hyperactivity disorder. Attention-deficit/hyperactivity disorder is characterized by persistent symptoms of inattention, hyperactivity, and impulsivity, which may resemble the symptoms of a manic episode (e.g., distractibility, increased activity, impulsive behavior) and have their onset by age 12. In contrast, the symptoms of mania in bipolar I disorder occur in distinct episodes and typically begin in late adolescence or early adulthood.

Disruptive mood dysregulation disorder. In individuals with severe irritability, particularly children and adolescents, care must be taken to apply the diagnosis of bipolar I disorder only to those who have had a clear episode of mania or hypomania—that is, a distinct time period, of the required duration, during which the irritability was clearly different from the individual's baseline and was accompanied by the onset of the other characteristic symptoms of mania (e.g., grandiosity, decreased need for sleep, pressured speech, involvement in activities with a high potential for painful consequences). When a child's irritability is persistent and particularly severe, the diagnosis of disruptive mood dysregulation disorder would be more appropriate. Indeed, when any child is being assessed for mania, it is essential that the symptoms represent a clear change from the child's typical behavior.

Personality disorders. Personality disorders such as borderline personality disorder may have substantial symptomatic overlap with bipolar I disorder, since mood lability and impulsivity are common in both conditions. In order to make a diagnosis of bipolar I disorder, symptoms of mood lability and impulsivity must represent a distinct episode of illness, or there must be a noticeable increase in these symptoms over the individual's baseline in order to justify an additional diagnosis of bipolar I disorder.

Comorbidity

Co-occurring mental disorders are the norm in bipolar I disorder, with the majority of individuals having a history of three or more disorders. The most frequently comorbid disorders are anxiety disorders, alcohol use disorder, other substance use disorder, and attention-deficit/hyperactivity disorder. Sociocultural factors influence the pattern of comorbid conditions in bipolar disorder. For example, countries with cultural prohibitions against alcohol or other substance use may have a lower prevalence of substance use comorbidity. Bipolar I disorder is frequently associated with borderline, schizotypal, and antisocial personality disorder. In particular, although the underlying nature of the relationship between bipolar I disorder and borderline personality disorder is unclear, the substantial comorbidity between the two may reflect similarities in phenomenology (i.e., misdiagnosing the emotional extremes of borderline personality disorder as bipolar I disorder), the influence of borderline personality features on vulnerability to bipolar I disorder, and the impact of early childhood adversity on the development of both bipolar I and borderline personality disorder.

Individuals with bipolar I disorder also have high rates of serious co-occurring and often untreated medical conditions, which largely explain the shortened life expectancy of those with bipolar disorder. Comorbidities appear in multiple organ systems, with cardiovascular and autoimmune diseases, obstructive sleep apnea, metabolic syndrome, and migraine more common among individuals with bipolar disorder than in the general population. Comorbid overweight/obesity is a particular concern for individuals with bipolar disorder and is associated with poor treatment outcomes.

Bipolar II Disorder

Diagnostic Criteria F31.81

For a diagnosis of bipolar II disorder, it is necessary to meet the following criteria for a current or past hypomanic episode *and* the following criteria for a current or past major depressive episode:

Hypomanic Episode

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy, lasting at least 4 consecutive days and present most of the day, nearly every day.

B. During the period of mood disturbance and increased energy and activity, three (or more) of the following symptoms have persisted (four if the mood is only irritable), represent a noticeable change from usual behavior, and have been present to a significant degree:

1. Inflated self-esteem or grandiosity.
2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep).
3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.

5. Distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed.
6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation.

7. Excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

C. The episode is associated with an unequivocal change in functioning that is uncharacteristic of the individual when not symptomatic.

D. The disturbance in mood and the change in functioning are observable by others.

E. The episode is not severe enough to cause marked impairment in social or occupational functioning or to necessitate hospitalization. If there are psychotic features, the episode is, by definition, manic.

F. The episode is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment) or another medical condition.

Note: A full hypomanic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a hypomanic episode diagnosis. However, caution is indicated so that one or two symptoms (particularly increased irritability, edginess, or agitation following antidepressant use) are not taken as sufficient for diagnosis of a hypomanic episode, nor necessarily indicative of a bipolar diathesis.

Major Depressive Episode

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

Note: Do not include symptoms that are clearly attributable to a medical condition.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, or hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**Note:** In children, consider failure to make expected weight gain.)
4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The episode is not attributable to the physiological effects of a substance or another medical condition.

Note: Criteria A–C constitute a major depressive episode.

Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.[1]

Bipolar II Disorder

A. Criteria have been met for at least one hypomanic episode (Criteria A–F under "Hypomanic Episode" above) and at least one major depressive episode (Criteria A–C under "Major Depressive Episode" above).
B. There has never been a manic episode.
C. At least one hypomanic episode and at least one major depressive episode are not better explained by schizoaffective disorder and are not superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.
D. The symptoms of depression or the unpredictability caused by frequent alternation between periods of depression and hypomania causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Coding and Recording Procedures

Bipolar II disorder has one diagnostic code: F31.81. Its status with respect to current severity, presence of psychotic features,

course, and other specifiers cannot be coded but should be indicated in writing (e.g., F31.81 bipolar II disorder, current episode depressed, moderate severity, with mixed features; F31.81 bipolar II disorder, most recent episode depressed, in partial remission).

Specify current or most recent episode:

Hypomanic

Depressed

If current episode is **hypomanic** (or most recent episode if bipolar II disorder is in partial or full remission):

In recording the diagnosis, terms should be listed in the following order: bipolar II disorder, current or most recent episode hypomanic, in partial remission/in full remission (p. 175) (if full criteria for a hypomanic episode are not currently met), plus any of the following hypomanic episode specifiers that are applicable. **Note:** The specifiers “with rapid cycling” and “with seasonal pattern” describe the pattern of mood episodes.

Specify if:

With anxious distress (p. 169–170)

With mixed features (pp. 170–171)

With rapid cycling (p. 171)

With peripartum onset (pp. 173–174)

With seasonal pattern (pp. 174–175)

If current episode is **depressed** (or most recent episode if bipolar II disorder is in partial or full remission):

In recording the diagnosis, terms should be listed in the following order: bipolar II disorder, current or most recent episode depressed, mild/moderate/severe (if full criteria for a major depressive episode are currently met), in partial

remission/in full remission (if full criteria for a major depressive episode are not currently met) (p. 175), plus any of the following major depressive episode specifiers that are applicable. **Note:** The specifiers “with rapid cycling” and “with seasonal pattern” describe the pattern of mood episodes.

Specify if:

With anxious distress (pp. 169–170)

With mixed features (pp. 170–171)

With rapid cycling (p. 171)

With melancholic features (pp. 171–172)

With atypical features (pp. 172–173)

With mood-congruent psychotic features (p. 173)

With mood-incongruent psychotic features (p. 173)

With catatonia (p. 173). **Coding note:** Use additional code F06.1.

With peripartum onset (pp. 172–174)

With seasonal pattern (pp. 174–175)

Specify course if full criteria for a mood episode are not currently met:

In partial remission (p. 175)

In full remission (p. 175)

Specify severity if full criteria for a major depressive episode are currently met:

Mild (p. 175)

Moderate (p. 175)

Severe (p. 175)

Diagnostic Features

Bipolar II disorder is characterized by a clinical course of recurring mood episodes consisting of one or more major depressive episodes (Criteria A–C

under “Major Depressive Episode”) and at least one hypomanic episode (Criteria A–F under “Hypomanic Episode”). A diagnosis of a major depressive episode requires that there be a period of depressed mood, or as an alternative, marked diminished interest or pleasure, for most of the day nearly every day, lasting for a minimum of 2 weeks. The depressed mood or loss of interest must be accompanied by additional symptoms occurring nearly every day (e.g., sleep disturbance, psychomotor agitation or retardation) for a total of at least five symptoms. The diagnosis of a hypomanic episode requires that there be a distinct period of

abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy for most of the day, nearly every day, for at least 4 consecutive days accompanied by three (or four if mood is only irritable) additional symptoms (e.g., inflated self-esteem, decreased need for sleep, distractibility) that persist and represent a noticeable change from usual behavior and functioning. By definition, psychotic symptoms do not occur in hypomanic episodes, and they appear to be less frequent in the major depressive episodes in bipolar II disorder than in those of bipolar I disorder. The presence of a manic episode during the course of illness precludes the diagnosis of bipolar II disorder (Criterion B under “Bipolar II Disorder”). Moreover, for depressive and hypomanic episodes to count toward the diagnosis of bipolar II disorder, at least one of the depressive episodes and at least one of the hypomanic episodes must not be attributable to the physiological effects of a substance (i.e., medication, drug of abuse, or toxin exposure) or another medical condition. Note that hypomanic episodes that emerge during antidepressant treatment and persist for at least 4 days at a fully syndromal level beyond the physiological effects of the treatment are not considered to be substance-induced and do count toward the diagnosis of bipolar II disorder. In addition, at least one hypomanic episode and at least one major depressive episode are not explained by a diagnosis of schizoaffective disorder and are not superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum or other psychotic disorder (Criterion C under “Bipolar II Disorder”). The

depressive episodes or the pattern of unpredictable mood changes must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion D under “Bipolar II Disorder”). The recurrent major depressive episodes are often more frequent and lengthier than those occurring in bipolar I disorder.

Individuals with bipolar II disorder typically present to a clinician during a major depressive episode. They are unlikely to complain initially of hypomania, because either they do not recognize the symptoms of hypomania or they consider hypomania desirable. Hypomanic episodes by definition do not cause significant impairment. Instead, the impairment results from the major depressive episodes or from a persistent pattern of unpredictable mood changes and fluctuating, unreliable interpersonal or occupational functioning. Individuals with bipolar II disorder may not view the hypomanic episodes as pathological or disadvantageous, although others may be troubled by the individual’s erratic behavior. Clinical information from other informants, such as close friends or relatives, is often useful in establishing the diagnosis of bipolar II disorder.

A hypomanic episode should not be confused with the several days of euthymia and restored energy or activity that may follow remission of a major depressive episode. Despite the substantial differences in duration and severity between a manic and hypomanic episode, bipolar II disorder is not a “milder form” of bipolar I disorder. Compared to individuals with bipolar I disorder, individuals with bipolar II disorder have greater chronicity of illness and spend, on average, more time in the depressive phase of their illness, which can be severe and/or disabling.

Although the diagnostic requirements for major depressive episodes are identical whether they occur in the context of bipolar II disorder or major depressive disorder, certain clinical features of the episodes may hint at possible differential diagnosis. For instance, the coexistence of both insomnia and hypersomnia is not uncommon in major depressive episodes in both bipolar II disorder and major depressive disorder; however, both insomnia and hypersomnia are overrepresented among women with bipolar II disorder. Similarly, atypical depressive symptoms (hypersomnia, hyperphagia) are common in both disorders, but more so in those with bipolar II disorder.

Depressive symptoms co-occurring with a hypomanic episode or hypomanic symptoms co-occurring with a depressive episode are common in individuals with bipolar II disorder and are overrepresented in females, particularly hypomania with mixed features.

Individuals experiencing hypomania with mixed features may not label their symptoms as hypomania, but instead experience them as depression with increased energy or irritability.

Associated Features

A common feature of bipolar II disorder is impulsivity, which can contribute to suicide attempts and substance use disorders.

There may be heightened levels of creativity during hypomanic episodes in some individuals with a bipolar II disorder. However, that relationship may be nonlinear; that is, greater lifetime creative accomplishments have been associated with milder forms of bipolar disorder, and higher creativity has been found in unaffected family members. The individual's attachment to the prospect of heightened creativity during hypomanic episodes may contribute to ambivalence about seeking treatment or undermine adherence to treatment.

Prevalence

The 12-month prevalence of bipolar II disorder in the United States is 0.8%. The 12-month prevalence internationally is 0.3%. The prevalence rate of pediatric bipolar II disorder is difficult to establish. DSM-IV bipolar I, bipolar II, and bipolar disorder not otherwise specified yield a combined prevalence rate of 1.8% in U.S. and non-U.S. community samples, with higher rates (2.7% inclusive) in youth age 12 years or older.

Development and Course

Although bipolar II disorder can begin in late adolescence and throughout adulthood, average age at onset is the mid-20s, which is slightly later than for bipolar I disorder but earlier than for major depressive disorder. Age at

onset does not reliably distinguish between bipolar I and II disorder. The illness most often begins with a depressive episode and is not recognized as bipolar II disorder until a hypomanic episode occurs; this happens in about 12% of individuals with the initial diagnosis of major depressive disorder. Anxiety, substance use, or eating disorders may also precede the diagnosis, complicating its detection. Many individuals experience several episodes of major depression prior to the first recognized hypomanic episode, with typically a more than 10-year lag between illness onset and the diagnosis of a bipolar disorder.

Bipolar II disorder is a highly recurrent disorder, with over 50% of individuals experiencing a new episode within a year after their first episode. Individuals with bipolar II disorder also have more seasonal variation in mood compared to those with bipolar I disorder.

The number of lifetime episodes (both hypomanic and major depressive episodes) tends to be higher for bipolar II disorder than for major depressive disorder or bipolar I disorder. However, individuals with bipolar I disorder are actually more likely to experience hypomanic symptoms than are individuals with bipolar II disorder. The interval between mood episodes in the course of bipolar II disorder tends to decrease as the individual ages. While the hypomanic episode is the feature that defines bipolar II disorder, depressive episodes are more enduring and disabling over time. Despite the predominance of depression, once a hypomanic episode has occurred, the diagnosis becomes bipolar II disorder and never reverts to major depressive disorder.

Approximately 5%–15% of individuals with bipolar II disorder have multiple (four or more) mood episodes (hypomanic or major depressive) within the previous 12 months. If this pattern is present, it is noted by the specifier “with rapid cycling.” Rapid cycling is more common in women and may reflect an overall worsening of the bipolar disorder.

Switching from a depressive episode to a manic or hypomanic episode (with or without mixed features) may occur, both spontaneously and during treatment for depression.

About 5%–15% of individuals with bipolar II disorder will ultimately develop a manic episode, which changes the diagnosis to bipolar I disorder, regardless of subsequent course.

Making the diagnosis in children is often a challenge, especially in those with irritability and hyperarousal that is *nonepisodic* (i.e., lacks the well-demarcated periods of altered mood). Nonepisodic irritability in youth is associated with an elevated risk for anxiety disorders and major depressive disorder, but not bipolar disorder, in adulthood. Persistently irritable youth have lower familial rates of bipolar disorder than do youth who have bipolar disorder. For a hypomanic episode to be diagnosed, the child's symptoms must exceed what is expected in a given environment and culture for the child's developmental stage. Similar to adults, youth with bipolar II disorder spend less time hypomanic compared to those with bipolar I disorder, and the initial presenting episode is typically depression. Compared with adult onset of bipolar II disorder, childhood or adolescent onset of the disorder may be associated with a more severe lifetime course.

The 3-year incidence rate of first-onset bipolar II disorder in adults older than 60 years is 0.34%. However, distinguishing individuals older than 60 years with bipolar II disorder by late versus early age at onset does not appear to have any clinical utility. The presence of co-occurring hypomanic symptoms during a depressive episode is more common during bipolar II depressive episodes relative to depressive episodes occurring in the context of major depression and may help distinguish older individuals with bipolar II disorder from those with major depressive disorder. In any later life presentation of bipolar disorder, it is important to consider medical factors, including possible medical and neurological causes of new symptoms.

Risk and Prognostic Factors

Genetic and physiological. The risk of bipolar II disorder tends to be highest among relatives of individuals with bipolar II disorder, as opposed to individuals with bipolar I disorder or major depressive disorder. About a third of individuals with bipolar II disorder reported a family history of bipolar disorder. There may be genetic factors influencing the age at onset for bipolar disorders. Th ere is also evidence that bipolar II disorder may

have a genetic architecture that is at least partially distinct from bipolar I disorder and from schizophrenia.

Course modifiers. A rapid-cycling pattern is associated with a poorer prognosis. Return to previous level of social function for individuals with bipolar II disorder is more likely for individuals of younger age and with less severe depression, suggesting adverse effects of prolonged illness on recovery. More education, fewer years of illness, and being married are independently associated with functional recovery in individuals with bipolar disorder, even after diagnostic type (I vs. II), current depressive symptoms, and presence of psychiatric comorbidity are taken into account.

Sex- and Gender-Related Diagnostic Issues

Whereas the gender ratio for bipolar I disorder is equal, findings on gender differences in bipolar II disorder are mixed, differing by type of sample (i.e., registry, community, or clinical) and country of origin. There is little to no evidence of bipolar gender differences in the general population, whereas some, but not all, clinical samples suggest that bipolar II disorder is more common in women than in men, which may reflect gender differences in treatment seeking or other factors.

Patterns of illness and comorbidity, however, seem to differ by sex, with females being more likely than males to report hypomania with mixed depressive features and a rapid-cycling course. Childbirth may also be a specific trigger for a hypomanic episode, which can occur in 10%–20% of females in nonclinical populations and most typically in the early postpartum period. Distinguishing hypomania from the elated mood and reduced sleep

that normally accompany the birth of a child may be challenging. Postpartum hypomania may foreshadow the onset of a depression that occurs in about half of females who experience postpartum “highs.” The perimenopause transition can also be a time of mood instability in bipolar II disorder. No major sex differences have been found in several clinical

variables, including rates of depressive episodes, age at and polarity of onset, symptoms, and severity of the illness.

Association With Suicidal Thoughts or Behavior

Approximately one-third of individuals with bipolar II disorder report a lifetime history of suicide attempt. The risk and incidence of attempted suicide in bipolar II and bipolar I disorder appear to be similar. Overall there appears to be about equal rates of suicide attempts and suicide deaths across individuals with bipolar II and bipolar I disorder, although overall the rates for both attempts and deaths are significantly higher than in the general population. Time spent in a depressive episode is associated more significantly with the diagnosis of bipolar I or bipolar II in terms of suicide attempt risk. However, the lethality of attempts, as defined by a lower ratio of attempts to suicide deaths, may be higher in individuals with bipolar II disorder compared to individuals with bipolar I disorder. There may be an association between genetic markers and increased risk for suicidal behavior in individuals with bipolar disorder, including a 6.5-fold higher risk of suicide among first-degree relatives of bipolar II probands compared with first-degree relatives of bipolar I probands.

Functional Consequences of Bipolar II Disorder

Although many individuals with bipolar II disorder return to a fully functional level between mood episodes, at least 15% continue to have some interepisode dysfunction, and 20% transition directly into another mood episode without interepisode recovery. Functional recovery lags substantially behind recovery from symptoms of bipolar II disorder, especially in regard to occupational recovery, resulting in lower socioeconomic status despite equivalent levels of education with the general population. Individuals with bipolar II disorder perform more poorly than healthy individuals on cognitive tests. Cognitive impairments associated with bipolar II disorder may contribute to vocational difficulties. Prolonged unemployment in individuals with bipolar disorder is associated with more episodes of depression, older age, increased rates of current panic disorder, and lifetime history of alcohol use disorder.

Differential Diagnosis

Major depressive disorder. Major depressive disorder is characterized by the absence of both manic episodes and hypomanic episodes. Given that the presence of some manic or hypomanic symptoms (e.g., fewer symptoms or shorter duration than required for hypomania) may still be compatible with a diagnosis of major depressive disorder, it is important to ascertain whether the symptoms meet criteria for a hypomanic episode to determine whether it is more appropriate to make the diagnosis of bipolar II disorder. Depressive episodes dominate the overall course of illness for most individuals with bipolar II disorder, contributing to the decade-long lag between illness onset and the diagnosis of bipolar II disorder. Because the diagnostic criteria for major depressive episode are identical in major depressive disorder and bipolar II disorder, the diagnosis of bipolar II disorder can be made only by eliciting information about at least one prior hypomanic episode in order to distinguish the bipolar II disorder from major depressive disorder.

Cyclothymic disorder. In cyclothymic disorder, there are numerous periods of hypomanic symptoms that do not meet symptom or duration criteria for a hypomanic episode and numerous periods of depressive symptoms that do not meet symptom or duration criteria for a major depressive episode. Bipolar II disorder is distinguished from cyclothymic disorder by the presence of one or more hypomanic episodes and one or more major depressive episodes.

Schizophrenia. Schizophrenia is characterized by active-phase psychotic symptoms that may be accompanied by major depressive episodes. The diagnosis of schizophrenia is made if no major depressive episodes have occurred concurrently with the active-phase symptoms. If they have occurred concurrently, the diagnosis of schizophrenia is made if the major depressive episodes have been present for only a minority of the time. The diagnosis is bipolar II disorder, with psychotic features, if the psychotic symptoms have occurred exclusively during major depressive episodes.

Schizoaffective disorder. Schizoaffective disorder is characterized by periods in which depressive symptoms are concurrent with the active-phase

symptoms of schizophrenia and periods in which delusions or hallucinations occur for at least 2 weeks in the absence of a major depressive episode. The diagnosis is bipolar II disorder, with psychotic features, if the psychotic symptoms have occurred exclusively during major depressive episodes.

Bipolar and related disorder due to another medical condition. The diagnosis of bipolar and related disorder due to another medical condition should be made instead of bipolar II disorder if the hypomanic episodes are judged, based on history, laboratory findings, or physical examination, to be the direct physiological consequence of another medical condition (e.g., Cushing's disease, multiple sclerosis).

Substance/medication-induced bipolar and related disorder. A substance/medication-induced bipolar and related disorder is distinguished from bipolar II disorder by the fact that a substance (e.g., stimulants, phencyclidine) or medication (e.g., steroids) is judged to be etiologically related to the hypomanic and major depressive episodes. Because individuals with a hypomanic episode have a tendency to overuse substances during an episode, it is important to determine whether the substance use is a consequence of a primary hypomanic episode or whether the hypomanic-like episode has been caused by the substance use. In some cases, a definitive diagnosis may involve establishing that the hypomanic symptoms or depressive symptoms remain once the individual is no longer using the substance. Note that hypomanic episodes emerging in the context of treatment with an antidepressant medication but persisting at a fully syndromal level beyond the physiological effect of the medication warrant a diagnosis of bipolar II disorder rather than substance/medication-induced bipolar and related disorder.

Attention-deficit/hyperactivity disorder. Attention-deficit/hyperactivity disorder (ADHD) may be misdiagnosed as bipolar II disorder, especially in adolescents and children. Many symptoms of ADHD, such as excessive talking, distractibility, and less need for sleep, overlap with the symptoms of hypomania. The double counting of symptoms toward both ADHD and bipolar II disorder can be avoided if the clinician clarifies whether the symptoms represent a distinct episode and if the noticeable increase over baseline required for the diagnosis of bipolar II disorder is present.

Personality disorders. The same convention as applies for ADHD also applies when evaluating an individual for a personality disorder such as borderline personality disorder because mood lability and impulsivity are common in both personality disorders and bipolar II disorder. Symptoms must represent a distinct episode, and the noticeable increase over baseline required for the diagnosis of bipolar II disorder must be present. A diagnosis of a personality disorder should not be made during an untreated mood episode unless the lifetime history supports the presence of a personality disorder.

Other bipolar disorders. Diagnosis of bipolar II disorder should be differentiated from bipolar I disorder by carefully considering whether there have been any past episodes of mania and from other specified and unspecified bipolar and related disorders by confirming the presence of fully syndromal hypomania and depression.

Comorbidity

Bipolar II disorder is more often than not associated with one or more co-occurring mental disorders, with anxiety disorders being the most common. Approximately 60% of individuals with bipolar II disorder have three or more co-occurring mental disorders; 75% have an anxiety disorder, most commonly social anxiety (38%), specific phobia (36%), and generalized anxiety (30%). Lifetime prevalence of comorbid anxiety disorder does not differ between bipolar I and bipolar II disorders but is associated with a worse course of illness. Children and adolescents with bipolar II disorder have a higher rate of co-occurring anxiety disorders compared to those with bipolar I disorder, and the anxiety disorder most often predates the bipolar disorder.

Anxiety and substance use disorders occur in individuals with bipolar II disorder at a higher rate than in the general population. It should be noted that co-occurring anxiety and substance use disorder do not seem to follow a course of illness that is truly independent from that of bipolar II disorder, but rather have strong associations with mood states. For example, anxiety

disorders tend to associate most with depressive symptoms, and substance use disorders are moderately associated with hypomanic symptoms.

The prevalence of substance use disorders appears to be similar between bipolar I and bipolar II disorders, most commonly alcohol use (42%) and cannabis use (20%) disorders. Sociocultural factors influence the pattern of comorbid conditions in bipolar II disorder. For example, countries with cultural prohibitions against alcohol or other substance use may have a lower prevalence of substance use comorbidity.

Individuals with bipolar II disorder appear to have lower rates of comorbid posttraumatic stress disorder compared to individuals with bipolar I disorder.

Approximately 14% of individuals with bipolar II disorder have at least one lifetime eating disorder, with binge-eating disorder being more common than bulimia nervosa and anorexia nervosa.

Premenstrual syndrome and premenstrual dysphoric disorder are common in women with bipolar disorder, especially in those with bipolar II disorder. Among women who have premenstrual syndrome and/or premenstrual dysphoric disorder, bipolar mood symptoms and lability may be more severe.

Individuals with bipolar II disorder also have comorbid medical conditions, which have the potential to substantially complicate course and prognosis. These include cardiovascular disease, migraine, and autoimmune disorders.

Cyclothymic Disorder

Diagnostic Criteria F34.0

A. For at least 2 years (at least 1 year in children and adolescents) there have been numerous periods with hypomanic symptoms that do not meet criteria for a hypomanic episode and numerous periods with depressive symptoms that do not meet criteria for a major depressive episode.

B. During the above 2-year period (1 year in children and adolescents), Criterion A symptoms have been present for at least half the time and the individual has not been without the symptoms for more than 2 months at a time.

C. Criteria for a major depressive, manic, or hypomanic episode have never been met.

D. The symptoms in Criterion A are not better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.

E. The symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism).

F. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

With anxious distress (see pp. 169–170)

Diagnostic Features

The essential feature of cyclothymic disorder is a chronic, fluctuating mood disturbance involving numerous periods of hypomanic symptoms and periods of depressive symptoms (Criterion A). The hypomanic symptoms are of insufficient number, severity, pervasiveness, and/or duration to meet full criteria for a hypomanic episode, and the depressive symptoms are of insufficient number, severity, pervasiveness, and/or duration to meet full criteria for a major depressive episode. During the initial 2-year period (1 year for children or adolescents), the symptoms must be persistent (present more days than not), and any symptom-free intervals last no longer than 2 months (Criterion B). The diagnosis of cyclothymic disorder is made only if

the criteria for a major depressive, manic, or hypomanic episode have never been met (Criterion C).

If an individual with cyclothymic disorder subsequently (i.e., after the initial 2 years in adults or 1 year in children or adolescents) experiences a major depressive, manic, or hypomanic episode, the diagnosis changes to major depressive disorder, bipolar I disorder, or other specified or unspecified bipolar and related disorder (subclassified as hypomanic episode without prior major depressive episode), respectively, and the cyclothymic disorder diagnosis is dropped.

The cyclothymic disorder diagnosis is not made if the pattern of mood swings is better explained by schizoaffective disorder, schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders (Criterion D), in which case the mood symptoms are considered associated features of the psychotic disorder. The mood disturbance must also not be attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism) (Criterion E). Although some individuals may function particularly well during some of the periods of hypomania, over the prolonged course of the disorder, there must be clinically significant distress or impairment in social, occupational, or other important areas of functioning as a result of the mood disturbance (Criterion F). The prolonged pattern of repeated, often unpredictable mood changes may lead to impairment attributable to the negative effects of the symptoms themselves combined with negative effects that the pattern of unpredictability and inconsistency has on interpersonal functioning and role performance (i.e., familial, occupational roles).

Prevalence

The lifetime prevalence of cyclothymic disorder in the United States and Europe is approximately 0.4%–2.5%. Prevalence in mood disorders clinics may range from 3% to 5%. In the general population, cyclothymic disorder is apparently equally common in males and females. In clinical settings, females with cyclothymic disorder may be more likely to present for treatment than males.

Development and Course

Cyclothymic disorder usually begins in adolescence or early adult life and is sometimes considered to reflect a temperamental predisposition to other disorders in this chapter. The vast majority of youth with cyclothymic disorder experience the onset of mood symptoms before age 10. Cyclothymic disorder usually has an insidious onset and a persistent course. There is a 15%–50% risk that an individual with cyclothymic disorder will

subsequently develop bipolar I disorder or bipolar II disorder; diagnostic conversion rates are higher in youth than in adults. Onset of persistent, fluctuating hypomanic and depressive symptoms late in adult life needs to be clearly differentiated from bipolar and related disorder due to another medical condition and depressive disorder due to another medical condition (e.g., multiple sclerosis) before the cyclothymic disorder diagnosis is assigned.

Risk and Prognostic Factors

Genetic and physiological. Major depressive disorder, bipolar I disorder, and bipolar II disorder are more common among first-degree biological relatives of individuals with cyclothymic disorder than in the general population. There may also be an increased familial risk of substance-related disorders. Cyclothymic disorder may be more common in the first-degree biological relatives of individuals with bipolar I disorder than in the general population.

Differential Diagnosis

Bipolar and related disorder due to another medical condition. The diagnosis of bipolar and related disorder due to another medical condition is made when the mood disturbance is judged to be attributable to the physiological effect of a specific, usually chronic medical condition (e.g., hyperthyroidism). This determination is based on the history, physical examination, and/or laboratory findings. If it is judged that the hypomanic and depressive symptoms are not the physiological consequence of the medical condition,

then the primary mental disorder (i.e., cyclothymic disorder) and the medical condition are coded. For example, this would be the case if the mood symptoms are considered to be the psychological (not the physiological) consequence of having a chronic medical condition, or if there is no etiological relationship between the hypomanic and depressive symptoms and the medical condition.

Substance/medication-induced bipolar and related disorder and substance/medication-induced depressive disorder. Substance/medication-induced bipolar and related disorder and substance/medication-induced depressive disorder are distinguished from cyclothymic disorder by the judgment that a substance/medication (especially stimulants) is etiologically related to the mood disturbance. The frequent mood swings in these disorders that are suggestive of cyclothymic disorder usually resolve following cessation of substance/medication use.

Bipolar I disorder, with rapid cycling, and bipolar II disorder, with rapid cycling. Both disorders may resemble cyclothymic disorder by virtue of the frequent marked shifts in mood. By definition, in cyclothymic disorder the criteria for a major depressive, manic, or hypomanic episode have never been met, whereas the bipolar I disorder and bipolar II disorder specifier “with rapid cycling” requires that full mood episodes be present.

Borderline personality disorder. Borderline personality disorder is associated with recurrent, brief marked shifts in mood that may suggest cyclothymic disorder. Engagement in potentially self-damaging behaviors can be seen in both conditions but would need to occur in the context of other hypomanic symptoms to be related to cyclothymia. Mood instability in borderline personality disorder occurs in the domains of anxiety, irritability, and sadness, whereas elation, euphoria, and/or increased energy are not characteristic features of borderline personality disorder. If the criteria are met for both disorders, both borderline personality disorder and cyclothymic disorder may be diagnosed.

Comorbidity

Substance-related disorders and sleep disorders (i.e., difficulties in initiating and maintaining sleep) may be present in individuals with cyclothymic disorder. Rates of comorbid

psychiatric disorders in children with cyclothymic disorder treated in outpatient psychiatric settings are greater than those in children with disruptive behavior/attention-deficit/hyperactivity disorder and similar to those in children with bipolar I or II disorder.

Substance/Medication-Induced Bipolar and Related Disorder

Diagnostic Criteria

A. A prominent and persistent disturbance in mood that predominates in the clinical picture and is characterized by abnormally elevated, expansive, or irritable mood and abnormally increased activity or energy.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a bipolar or related disorder that is not substance/medication-induced. Such evidence of an independent bipolar or related disorder could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an

independent non-substance/medication-induced bipolar and related disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance/medication]-induced bipolar and related disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given. If a mild substance use disorder is comorbid with the substance-induced bipolar and related disorder, the 4th position character is “1,” and the clinician should record “mild [substance] use disorder” before the substance-induced bipolar and related disorder (e.g., “mild cocaine use disorder with cocaine-induced bipolar and related disorder”). If a moderate or severe substance use disorder is comorbid with the substance-induced bipolar and related disorder, the 4th position character is “2,” and the clinician should record “moderate [substance] use disorder” or “severe [substance] use disorder,” depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is “9,” and the clinician should record only the substance-induced bipolar and related disorder.

	ICD-10-CM		
	With mild use disorder	**With moderate or severe use disorder**	**Without use disorder**
Alcohol	F10.14	F10.24	F10.94
Phencyclidine	F16.14	F16.24	F16.94
Other hallucinogen	F16.14	F16.24	F16.94
Sedative, hypnotic, or anxiolytic	F13.14	F13.24	F13.94
Amphetamine-type substance (or other stimulant)	F15.14	F15.24	F15.94
Cocaine	F14.14	F14.24	F14.94
Other (or unknown) substance	F19.14	F19.24	F19.94

Specify (see Table 1 in the chapter "Substance-Related and Addictive Disorders," which indicates whether "with onset during intoxication" and/or "with onset during withdrawal" applies to a given substance class; or *specify* "with onset after medication use"):

With onset during intoxication: If criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: If symptoms developed at initiation of medication, with a change in use of medication, or during withdrawal of medication.

Recording Procedures

The name of the substance/medication-induced bipolar and related disorder begins with the specific substance (e.g., cocaine, dexamethasone) that is presumed to be causing the bipolar mood symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder.

For substances that do not fit into any of the classes (e.g., dexamethasone), the code for “other (or unknown) substance” should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the same code should also be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word “with,” followed by the name of the substance-induced bipolar and related disorder, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal). For example, in the case of irritable symptoms occurring during intoxication in a man with a severe cocaine use disorder, the diagnosis is F14.24 severe cocaine use disorder with cocaine-induced bipolar and related disorder, with onset during intoxication. A separate diagnosis of the comorbid severe cocaine use disorder is not given. If the substance-induced bipolar and related disorder occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F15.94 amphetamine-induced bipolar and related disorder, with onset during intoxication). When more than one substance is judged to play a significant role in the development of bipolar mood symptoms, each should be listed separately (e.g., F15.24 severe methylphenidate use disorder with methylphenidate-induced bipolar and related disorder, with onset during intoxication; F19.94 dexamethasone-induced bipolar and related disorder, with onset during intoxication).

Diagnostic Features

The essential feature of substance/medication-induced bipolar and related disorder is a prominent and persistent disturbance in mood that predominates in the clinical picture and is characterized by abnormally elevated, expansive, or irritable mood and abnormally increased activity or energy (Criterion A); these symptoms are judged to be attributable to the effects of a substance (e.g., a drug of abuse, a medication, or a toxin exposure) (Criterion B).

To meet criteria for the diagnosis, the abnormally elevated, expansive, or irritable mood and increased activity or energy must have developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication, as evidenced by clinical history, physical examination, or laboratory findings (Criterion B1), and the involved substance/medication must be capable of producing the abnormally elevated, expansive, or irritable mood and increased activity or energy (Criterion B2). In addition, the abnormally elevated, expansive, or irritable mood and increased activity or energy are not better explained by a non-substance/medication-induced bipolar and related disorder.

Evidence of an independent bipolar and related disorder includes the observation that the abnormally elevated, expansive, or irritable mood and increased activity or energy preceded the onset of substance/medication use, the symptoms persist beyond a substantial period of time after the cessation of acute withdrawal or severe intoxication (i.e., usually longer than 1 month), or there is other evidence that suggests the existence of an independent non-substance/medication-induced bipolar and related disorder (Criterion C), such as a history of recurrent non-substance-induced manic episodes. Diagnosis of substance/medication-induced bipolar and related disorder should not be made when symptoms occur exclusively during the course of a delirium (Criterion D). Finally, the diagnosis requires that the substance/medication-induced symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E). The substance-induced bipolar and related disorder diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and are sufficiently severe to warrant independent clinical attention.

A key exception to the diagnosis of substance/medication-induced bipolar and related disorder is the case of hypomania or mania that occurs after antidepressant medication use or other treatments and persists beyond the physiological effects of the medication. The persistence of hypomania or mania is considered an indicator of true bipolar disorder, not substance/medication-induced bipolar and related disorder. Similarly, individuals with apparent electroconvulsive therapy–induced manic or hypomanic episodes that persist beyond the physiological effects of the

treatment are diagnosed with bipolar disorder, not substance/medication-induced bipolar and related disorder. Furthermore, substance/medication-induced bipolar and related symptoms may suggest an underlying bipolar diathesis in individuals previously not diagnosed with bipolar disorders.

Side effects of some antidepressants and other psychotropic drugs (e.g., edginess, agitation) may resemble the primary symptoms of a manic syndrome, but they are fundamentally distinct from bipolar symptoms and are insufficient for the diagnosis. That is, the criterion symptoms of mania/hypomania have specificity (simple agitation is not the same as excess involvement in purposeful activities), and a sufficient number of symptoms must be present (not just one or two symptoms) to make these diagnoses. In particular, the appearance of one or two nonspecific symptoms—irritability, edginess, or agitation during antidepressant treatment—in the absence of a full manic or hypomanic syndrome should not be taken to support a diagnosis of a bipolar disorder.

Associated Features

Substances/medications that are typically considered to be associated with substance/medication-induced bipolar and related disorder include the stimulant class of drugs, as

well as phencyclidine and steroids; however, a number of potential substances continue to emerge as new compounds are synthesized (e.g., so-called bath salts).

Prevalence

Limited epidemiological data exist regarding the prevalence of substance/medication-induced mania or bipolar disorder. Prevalence of substance-induced bipolar disorder will depend on substance availability and level of substance use in a society; for example, countries with cultural prohibitions against alcohol or other substance use may have a lower prevalence of substance-related disorders.

Development and Course

In phencyclidine-induced mania, the initial presentation may be one of a delirium with affective features, which then becomes an atypically appearing manic or mixed manic state. This condition follows the ingestion or inhalation quickly, usually within hours or, at the most, a few days. In stimulant-induced manic or hypomanic states, the response is in minutes to 1 hour after one or several ingestions or injections. The episode is very brief and typically resolves over 1–2 days. With corticosteroids and some immunosuppressant medications, the mania (or mixed or depressed state) usually follows several days of ingestion, and the higher doses appear to have a much greater likelihood of producing bipolar symptoms.

Diagnostic Markers

Determination of the substance of use can be made through markers in the blood or urine to corroborate diagnosis.

Differential Diagnosis

Substance/medication-induced bipolar and related disorder should be differentiated from other bipolar disorders, substance intoxication, substance withdrawal, substance-induced delirium, and medication side effects (as noted earlier). A full manic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a bipolar I diagnosis. A full hypomanic episode that emerges during antidepressant treatment (e.g., medication, electroconvulsive therapy) but persists at a fully syndromal level beyond the physiological effect of that treatment is sufficient evidence for a bipolar II diagnosis only if preceded by a major depressive episode.

Substance intoxication and substance withdrawal. Euphoria, irritability, and increased energy may occur in substance intoxication (e.g., stimulant intoxication) or substance withdrawal (e.g., cannabis withdrawal). The diagnosis of the substance-specific intoxication or substance-specific withdrawal will usually suffice to categorize the symptom presentation. A diagnosis of substance/medication-induced bipolar and related disorder

either with onset during intoxication or with onset during withdrawal should be made instead of a diagnosis of substance intoxication or substance withdrawal when the euphoric or irritable mood or increased energy symptoms are predominant in the clinical picture and are sufficiently severe to warrant clinical attention.

Comorbidity

Comorbidities are those associated with the use of illicit substances (in the case of illegal stimulants or phencyclidine) or diversion of prescribed stimulants. Comorbidities related to steroid or immunosuppressant medications are those medical indications for these preparations. Delirium can occur before or along with manic symptoms in individuals ingesting phencyclidine or those who are prescribed steroid medications or other immunosuppressant medications.

Bipolar and Related Disorder Due to Another Medical Condition

Diagnostic Criteria

A. A prominent and persistent disturbance in mood that predominates in the clinical picture and is characterized by abnormally elevated, expansive, or irritable mood and abnormally increased activity or energy.
B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.
C. The disturbance is not better explained by another mental disorder.

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning, or necessitates hospitalization to prevent harm to self or others, or there are psychotic features.

Coding note: The ICD-10-CM code depends on the specifier (see below).

Specify if:

F06.33 With manic features: Full criteria are not met for a manic or hypomanic episode.

F06.33 With manic- or hypomanic-like episode: Full criteria are met except Criterion D for a manic episode or except Criterion F for a hypomanic episode.

F06.34 With mixed features: Symptoms of depression are also present but do not predominate in the clinical picture.

Coding note: Include the name of the other medical condition in the name of the mental disorder (e.g., F06.33 bipolar disorder due to hyperthyroidism, with manic features). The other medical condition should also be coded and listed separately immediately before the bipolar and related disorder due to the medical condition (e.g., E05.90 hyperthyroidism; F06.33 bipolar disorder due to hyperthyroidism, with manic features).

Diagnostic Features

The essential features of bipolar and related disorder due to another medical condition are presence of a prominent and persistent period of abnormally elevated, expansive, or irritable mood and abnormally increased activity or energy predominating in the clinical picture (Criterion A) that is attributable to another medical condition (Criterion B). In most cases the manic or hypomanic picture may appear during the initial presentation of the medical condition (i.e., within 1 month); however, there are exceptions, especially in chronic medical conditions that might worsen or relapse and herald the

appearance of the manic or hypomanic picture. Bipolar and related disorder due to another medical condition would not be diagnosed when the manic or hypomanic episodes definitely preceded the medical condition, because the proper diagnosis would be bipolar disorder (except in the unusual circumstance in which all preceding manic or hypomanic episodes—or, when only one such episode has occurred, the preceding manic or hypomanic episode—were associated with ingestion of a substance/medication). The diagnosis of bipolar and related disorder due to another medical condition should not be made during the course of a delirium (Criterion D). The manic or hypomanic episode in bipolar and related disorder due to another medical condition must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning to qualify for this diagnosis (Criterion E).

Associated Features

The listing of medical conditions that are said to be able to induce mania is never complete, and the clinician's best judgment is the essence of this diagnosis. Among the best known of the medical conditions that can cause a bipolar manic or hypomanic condition are Cushing's disease and multiple sclerosis, as well as stroke and traumatic brain injuries. Antibodies to the *N*-methyl-D-aspartate (NMDA) receptor have been associated with manic or mixed mood and psychotic symptoms. In such cases, the causative medical condition would be anti-NMDA receptor encephalitis.

Development and Course

Bipolar and related disorder due to another medical condition usually has its onset acutely or subacutely within the first weeks or month of the onset of the associated medical condition. However, this is not always the case, as a worsening or later relapse of the associated medical condition may precede the onset of the manic or hypomanic syndrome. The clinician must make a clinical judgment in these situations about whether the medical condition is causative, based on temporal sequence as well as plausibility of a causal relationship. Finally, the condition may remit before or just after the

medical condition remits, particularly when treatment of the manic/hypomanic symptoms is effective.

Culture-Related Diagnostic Issues

Culture-related differences, to the extent that there is any evidence, pertain to those associated with the medical condition (e.g., rates of multiple sclerosis and stroke vary around the world based on dietary factors, genetic factors, and other environmental factors).

Sex- and Gender-Related Diagnostic Issues

Gender differences pertain to those associated with the medical condition (e.g., systemic lupus erythematosus is more common in females; stroke is somewhat more common in middle-age males compared with females).

Diagnostic Markers

Diagnostic markers pertain to those associated with the medical condition (e.g., steroid levels in blood or urine to help corroborate the diagnosis of Cushing's disease, which can be associated with manic or depressive syndromes; laboratory tests confirming the diagnosis of multiple sclerosis).

Functional Consequences of Bipolar and Related Disorder Due to Another Medical Condition

Functional consequences of the bipolar symptoms may exacerbate impairments associated with the medical condition and may incur worse outcomes because of interference with medical treatment.

Differential Diagnosis

Delirium and major or mild neurocognitive disorder. A separate diagnosis of bipolar and related disorder due to another medical condition is not given if the mood disturbance occurs exclusively during the course of a delirium. However, a diagnosis of bipolar and related disorder due to another medical condition may be given in addition to a diagnosis of major or mild neurocognitive disorder if the mood disturbance is judged to be a physiological consequence of the pathological process causing the

neurocognitive disorder and if symptoms of irritability or elevated mood are a prominent part of the clinical presentation.

Symptoms of catatonia and acute anxiety. It is important to differentiate symptoms of mania from excited catatonic symptoms and from agitation related to acute anxiety states.

Medication-induced depressive or manic symptoms. An important differential diagnostic observation is that the other medical condition may be treated with medications (e.g., steroids or alpha-interferon) that can induce depressive or manic symptoms. In these cases, clinical judgment using all of the evidence in hand is the best way to try to separate the most likely and/or the most important of two etiological factors (i.e., association with the medical condition vs. a substance/medication-induced syndrome). The differential diagnosis of the associated medical conditions is relevant but largely beyond the scope of the present manual.

Comorbidity

Conditions comorbid with bipolar and related disorder due to another medical condition are those associated with the medical conditions of etiological relevance. Delirium can occur before or along with manic symptoms in individuals with Cushing's disease.

Other Specified Bipolar and Related Disorder

F31.89

This category applies to presentations in which symptoms characteristic of a bipolar and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the bipolar and

related disorders diagnostic class. The other specified bipolar and related disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific bipolar and related disorder. This is done by recording “other specified bipolar and related disorder” followed by the specific reason (e.g., “short-duration cyclothymia”).

Examples of presentations that can be specified using the “other specified” designation include the following:

1. **Short-duration hypomanic episodes (2–3 days) and major depressive episodes:** A lifetime history of one or more major depressive episodes in individuals whose presentation has never met full criteria for a manic or hypomanic episode but who have experienced two or more episodes of short-duration hypomania that meet the full symptomatic criteria for a hypomanic episode but that only last for 2–3 days. The episodes of hypomanic symptoms do not overlap in time with the major depressive episodes, so the disturbance does not meet criteria for major depressive episode, with mixed features.
2. **Hypomanic episodes with insufficient symptoms and major depressive episodes:** A lifetime history of one or more major depressive episodes in individuals whose presentation has never met full criteria for a manic or hypomanic episode but who have experienced one or more episodes of hypomania that do not meet full symptomatic criteria (i.e., at least 4 consecutive days of elevated mood and one or two of the other symptoms of a hypomanic episode, or irritable mood and two or three of the other symptoms of a hypomanic episode). The episodes of hypomanic symptoms do not overlap in time with the major depressive episodes, so the disturbance does not meet criteria for major depressive episode, with mixed features.
3. **Hypomanic episode without prior major depressive episode:** One or more hypomanic episodes in an individual

whose presentation has never met full criteria for a major depressive episode or a manic episode.

4. **Short-duration cyclothymia (less than 24 months):** Multiple episodes of hypomanic symptoms that do not meet criteria for a hypomanic episode and multiple episodes of depressive symptoms that do not meet criteria for a major depressive episode that persist over a period of less than 24 months (less than 12 months for children or adolescents) in an individual whose presentation has never met full criteria for a major depressive, manic, or hypomanic episode and does not meet criteria for any psychotic disorder. During the course of the disorder, the hypomanic or depressive symptoms are present for more days than not, the individual has not been without symptoms for more than 2 months at a time, and the symptoms cause clinically significant distress or impairment.
5. **Manic episode superimposed** on schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorder. **Note:** Manic episodes that are part of schizoaffective disorder do not merit an additional diagnosis of other specified bipolar and related disorder.

Unspecified Bipolar and Related Disorder

F31.9

This category applies to presentations in which symptoms characteristic of a bipolar and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the bipolar and

related disorders diagnostic class. The unspecified bipolar and related disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific bipolar and related disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Unspecified Mood Disorder

F39

This category applies to presentations in which symptoms characteristic of a mood dis-order that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not at the time of the evaluation meet the full criteria for any of the disorders in either the bipolar or the depressive disorders diagnostic classes and in which it is difficult to choose between unspecified bipolar and related disorder and unspecified depressive disorder (e.g., acute agitation).

Specifiers for Bipolar and Related Disorders

Specify if:

With anxious distress: The presence of at least two of the following symptoms during the majority of days of the current manic, hypomanic, or major depressive episode in bipolar I disorder (or the most recent episode if bipolar I disorder is in partial or full remission); or of the current hypomanic or major depressive episode in bipolar II disorder (or the most recent episode if bipolar II disorder is in partial or full remission); or during the majority of symptomatic days in cyclothymic disorder:

1. Feeling keyed up or tense.
2. Feeling unusually restless.

3. Difficulty concentrating because of worry.
4. Fear that something awful may happen.
5. Feeling that the individual might lose control of himself or herself.

Specify current severity:

Mild: Two symptoms.

Moderate: Three symptoms.

Moderate-severe: Four or five symptoms.

Severe: Four or five symptoms with motor agitation.

Note: Anxious distress has been noted as a prominent feature of both bipolar and major depressive disorders in both primary care and specialty mental health settings. High levels of anxiety have been associated with higher suicide risk, longer duration of illness, and greater likelihood of treatment nonresponse. As a result, it is clinically useful to specify accurately the presence and severity levels of anxious distress for treatment planning and monitoring of response to treatment.

With mixed features: The mixed features specifier can apply to the current manic, hypomanic, or major depressive episode in bipolar I disorder (or the most recent episode if bipolar I disorder is in partial or full remission) or to the current hypomanic or major depressive episode in bipolar II disorder (or the most recent episode if bipolar II disorder is in partial or full remission):

Manic or hypomanic episode, with mixed features:

A. Full criteria are met for a manic episode or hypomanic episode, and at least three of the following symptoms

are present during the majority of days of the current or most recent episode of mania or hypomania:

1. Prominent dysphoria or depressed mood as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful).
2. Diminished interest or pleasure in all, or almost all, activities (as indicated by either subjective account or observation made by others).
3. Psychomotor retardation nearly every day (observable by others; not merely subjective feelings of being slowed down).
4. Fatigue or loss of energy.
5. Feelings of worthlessness or excessive or inappropriate guilt (not merely self-reproach or guilt about being sick).
6. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. Mixed symptoms are observable by others and represent a change from the person's usual behavior.

C. For individuals whose symptoms meet full episode criteria for both mania and depression simultaneously, the diagnosis should be manic episode, with mixed features, due to the marked impairment and clinical severity of full mania.

D. The mixed symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication or other treatment).

Depressive episode, with mixed features:

A. Full criteria are met for a major depressive episode, and at least three of the following manic/hypomanic

symptoms are present during the majority of days of the current or most recent episode of depression:

1. Elevated, expansive mood.
2. Inflated self-esteem or grandiosity.

3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Increase in energy or goal-directed activity (either socially, at work or school, or sexually).
6. Increased or excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).
7. Decreased need for sleep (feeling rested despite sleeping less than usual; to be contrasted with insomnia).

B. Mixed symptoms are observable by others and represent a change from the person's usual behavior.

C. For individuals whose symptoms meet full episode criteria for both mania and depression simultaneously, the diagnosis should be manic episode, with mixed features.

D. The mixed symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication or other treatment).

Note: Mixed features associated with a major depressive episode have been found to be a significant risk factor for the development of bipolar I or bipolar II disorder. As a result, it is clinically useful to note the presence of this specifier for treatment planning and monitoring of response to treatment.

With rapid cycling: Presence of at least four mood episodes in the previous 12 months that meet the criteria for manic, hypomanic, or major depressive episode in bipolar I disorder or that meet the criteria for hypomanic or major depressive episode in bipolar II disorder.

Note: Episodes are demarcated by either partial or full remissions of at least 2 months or a switch to an episode of the opposite polarity (e.g., major depressive episode to manic episode).

Note: The essential feature of a rapid-cycling bipolar disorder is the occurrence of at least four mood episodes during the previous 12 months. These episodes can occur in any combination and order. The episodes must meet both the duration and the symptom number criteria for a major depressive, manic, or hypomanic episode and must be demarcated by either a period of full remission or a switch to an episode of the opposite polarity. Manic and hypomanic episodes are counted as being on the same pole. Except for the fact that they occur more frequently, the episodes that occur in a rapid-cycling pattern are no different from those that occur in a non-rapid-cycling pattern. Mood episodes that count toward defining a rapid-cycling pattern exclude those episodes directly caused by a substance (e.g., cocaine, corticosteroids) or another medical condition.

With melancholic features:

A. One of the following is present during the most severe period of the current major depressive episode (or the most recent major depressive episode if bipolar I or bipolar II disorder is currently in partial or full remission):

   1. Loss of pleasure in all, or almost all, activities.
   2. Lack of reactivity to usually pleasurable stimuli (does not feel much better, even temporarily, when something good happens).

B. Three (or more) of the following:

1. A distinct quality of depressed mood characterized by profound despondency, despair, and/or moroseness or by so-called empty mood.
2. Depression that is regularly worse in the morning.

3. Early-morning awakening (i.e., at least 2 hours before usual awakening).
4. Marked psychomotor agitation or retardation.
5. Significant anorexia or weight loss.
6. Excessive or inappropriate guilt.

Note: The specifier “with melancholic features” is applied if these features are present at the most severe stage of the episode. There is a near-complete absence of the capacity for pleasure, not merely a diminution. A guideline for evaluating the lack of reactivity of mood is that even highly desired events are not associated with marked brightening of mood. Either mood does not brighten at all, or it brightens only partially (e.g., up to 20%–40% of normal for only minutes at a time). The “distinct quality” of mood that is characteristic of the “with melancholic features” specifier is experienced as qualitatively different from that during a nonmelancholic depressive episode. A depressed mood that is described as merely more severe, longer lasting, or present without a reason is not considered distinct in quality. Psychomotor changes are nearly always present and are observable by others.

Melancholic features exhibit only a modest tendency to repeat across episodes in the same individual. They are more frequent in inpatients, as opposed to outpatients; are less likely to occur in milder than in

more severe major depressive episodes; and are more likely to occur in individuals with psychotic features.

With atypical features: This specifier is applied when these features predominate during the majority of days of the current major depressive episode (or the most recent major depressive episode if bipolar I or bipolar II disorder is currently in partial or full remission).

A. Mood reactivity (i.e., mood brightens in response to actual or potential positive events).
B. Two (or more) of the following:
    1. Significant weight gain or increase in appetite.
    2. Hypersomnia.
    3. Leaden paralysis (i.e., heavy, leaden feelings in arms or legs).
    4. A long-standing pattern of interpersonal rejection sensitivity (not limited to episodes of mood disturbance) that results in significant social or occupational impairment.
C. Criteria are not met for “with melancholic features” or “with catatonia” during the same episode.

Note: “Atypical depression” has historical significance (i.e., atypical in contradistinction to the more classical agitated, “endogenous” presentations of depression that were the norm when depression was rarely diagnosed in outpatients and almost never in adolescents or younger adults) and today does not connote an uncommon or unusual clinical presentation as the term might imply.

Mood reactivity is the capacity to be cheered up when presented with positive events (e.g., a visit from children, compliments from others). Mood may become euthymic (not sad) even for extended periods of time if the external circumstances remain favorable. Increased appetite may be manifested by an obvious increase in food intake or by

weight gain. Hypersomnia may include either an extended period of nighttime sleep or daytime napping that totals at least 10 hours of sleep per day (or at least 2 hours more than when not depressed). Leaden paralysis is defined as feeling heavy, leaden, or weighted down, usually in the arms or legs. This sensation is generally present for at least an hour a day but often lasts for many

hours at a time. Unlike the other atypical features, pathological sensitivity to perceived interpersonal rejection is a trait that has an early onset and persists throughout most of adult life. Rejection sensitivity occurs both when the person is and is not depressed, though it may be exacerbated during depressive periods.

With psychotic features: Delusions or hallucinations are present at any time in the current manic or major depressive episode in bipolar I disorder (or the most recent manic or major depressive episode if bipolar I disorder is currently in partial or full remission) or in the current major depressive episode in bipolar II disorder (or the most recent major depressive episode if bipolar II disorder is currently in partial or full remission). If psychotic features are present, *specify* if mood-congruent or mood-incongruent:

When applied to current or most recent manic episode (in bipolar I disorder):

With mood-congruent psychotic features: The content of all delusions and hallucinations is consistent with the typical manic themes of grandiosity, invulnerability, etc., but may also include themes of suspiciousness or paranoia, especially with respect to others' doubts about the individual's capacities, accomplishments, and so forth.

With mood-incongruent psychotic features: The content of the delusions and hallucinations does not involve typical manic themes as described above, or the content is a mixture of mood-incongruent and mood-congruent themes.

When applied to current or most recent major depressive episode (in bipolar I disorder or bipolar II disorder):

With mood-congruent psychotic features: The content of all delusions and hallucinations is consistent with the typical depressive themes of personal inadequacy, guilt, disease, death, nihilism, or deserved punishment.

With mood-incongruent psychotic features: The content of the delusions and hallucinations does not involve typical depressive themes of personal inadequacy, guilt, disease, death, nihilism, or deserved punishment, or the content is a mixture of mood-incongruent and mood-congruent themes.

With catatonia: This specifier is applied to the current manic or major depressive episode in bipolar I disorder (or the most recent manic or major depressive episode if bipolar I disorder is currently in partial or full remission) or to the current major depressive episode in bipolar II disorder (or the most recent major depressive episode if bipolar II disorder is currently in partial or full remission) if catatonic features are present during most of the episode. See criteria for catatonia associated with a mental disorder in the chapter “Schizophrenia Spectrum and Other Psychotic Disorders.”

With peripartum onset: This specifier is applied to the current manic, hypomanic, or major depressive episode in bipolar I disorder (or the most recent manic, hypomanic, or major depressive episode if bipolar I disorder is currently in partial or full remission) or to the current hypomanic or

major depressive episode in bipolar II disorder (or the most recent hypomanic or major depressive episode if bipolar II disorder is currently in partial or full remission) if onset of mood symptoms occurs during pregnancy or in the 4 weeks following delivery.

Note: Mood episodes can have their onset either during pregnancy or postpartum. About 50% of postpartum major depressive episodes begin prior to delivery. Thus, these episodes are referred to collectively as *peripartum* episodes.

Between conception and birth, about 9% of women will experience a major depressive episode. The best estimate for prevalence of a major depressive episode between birth and 12 months postpartum is just below 7%.

Peripartum-onset mood episodes can present either with or without psychotic features. Infanticide (a rare occurrence) is most often associated with postpartum

psychotic episodes that are characterized by command hallucinations to kill the infant or delusions that the infant is possessed, but psychotic symptoms can also occur in severe postpartum mood episodes without such specific delusions or hallucinations.

Postpartum mood (major depressive or manic) episodes with psychotic features appear to occur in from 1 in 500 to 1 in 1,000 deliveries and may be more common in primiparous women. The risk of postpartum episodes with psychotic features is particularly increased for women with prior postpartum psychotic mood episodes but is also elevated for those with a prior history of a depressive or bipolar disorder (especially bipolar I disorder) and those with a family history of bipolar disorders.

Once a woman has had a postpartum episode with psychotic features, the risk of recurrence with each subsequent delivery is between 30% and 50%. Postpartum episodes must be differentiated from delirium occurring in the postpartum period, which is distinguished by a fluctuating level of awareness or attention.

Peripartum-onset depressive disorders must be distinguished from the much more common “maternity blues,” or what is known in lay terms as “baby blues.” Maternity blues is not considered to be a mental disorder and is characterized by sudden changes in mood (e.g., the sudden onset of tearfulness in the absence of depression) that do not cause functional impairment and that are likely caused by physiological changes occurring after delivery. It is temporary and self-limited, typically improving quickly (within a week) without the need for treatment. Other symptoms of maternity blues include sleep disturbance and even confusion that can occur shortly after delivery.

Perinatal women may be at higher risk for depressive disorders due to thyroid abnormalities as well as other medical conditions that can cause depressive symptoms. If the depressive symptoms are judged to be due to another medical condition related to the perinatal period, depressive disorder due to another medical condition should be diagnosed instead of a major depressive episode, with peripartum onset.

With seasonal pattern: This specifier applies to the lifetime pattern of mood episodes. The essential feature is a regular seasonal pattern of at least one type of episode (i.e., mania, hypomania, or depression). The other types of episodes may not follow this pattern. For example, an individual may have seasonal manias but have

depressions that do not regularly occur at a specific time of year.

A. There has been a regular temporal relationship between the onset of manic, hypomanic, or major depressive episodes and a particular time of the year (e.g., in the fall or winter) in bipolar I or bipolar II disorder.

   Note: Do not include cases in which there is an obvious effect of seasonally related psychosocial stressors (e.g., regularly being unemployed every winter).

B. Full remissions (or a change from major depression to mania or hypomania or vice versa) also occur at a characteristic time of the year (e.g., depression disappears in the spring).

C. In the last 2 years, the individual's manic, hypomanic, or major depressive episodes have demonstrated a temporal seasonal relationship, as defined above, and no nonseasonal episodes of that polarity have occurred during that 2-year period.

D. Seasonal manias, hypomanias, or depressions (as described above) substantially outnumber any nonseasonal manias, hypomanias, or depressions that may have occurred over the individual's lifetime.

Note: The specifier "with seasonal pattern" can apply to the pattern of major depressive episodes in bipolar I and bipolar II disorder, to the pattern of manic episodes

and hypomanic episodes in bipolar I disorder, and to the pattern of hypomanic episodes in bipolar II disorder. The essential feature is the onset and remission of major depressive, manic, or hypomanic episodes at characteristic times of the year. In most cases, the seasonal major depressive episodes begin in fall or

winter and remit in spring. Less commonly, there may be recurrent summer depressive episodes. This pattern of onset and remission of episodes must have occurred during at least a 2-year period, without any nonseasonal episodes occurring during this period. In addition, the seasonal depressive, manic, or hypomanic episodes must substantially outnumber any nonseasonal depressive, manic, or hypomanic episodes over the individual's lifetime.

This specifier does not apply to those situations in which the pattern is better explained by seasonally linked psychosocial stressors (e.g., seasonal unemployment or school schedule). It is unclear whether a seasonal pattern of major depressive episodes is more likely in recurrent major depressive disorder or in bipolar disorders. However, within the bipolar disorders group, a seasonal pattern of major depressive episodes appears to be more likely in bipolar II disorder than in bipolar I disorder. In some individuals, the onset of manic or hypomanic episodes may also be linked to a particular season, with peak seasonality of mania or hypomania from spring through summer.

The prevalence of winter-type seasonal pattern appears to vary with latitude, age, and sex. Prevalence increases with higher latitudes. Age is also a strong predictor of seasonality, with younger persons at higher risk for winter depressive episodes.

Specify if:

In partial remission: Symptoms of the immediately previous manic, hypomanic, or major depressive episode are present but full criteria are not met, or there is a period lasting less than 2 months without any significant symptoms of a manic, hypomanic, or major depressive episode following the end of such an episode.

In full remission: During the past 2 months, no significant signs or symptoms of the disturbance were present.

Specify current severity of manic episode:

Severity is based on the number of criterion symptoms, the severity of those symptoms, and the degree of functional disability.

Mild: Minimum symptom criteria are met for a manic episode.

Moderate: Very significant increase in activity or impairment in judgment.

Severe: Almost continual supervision is required in order to prevent physical harm to self or others.

Specify current severity of major depressive episode:

Severity is based on the number of criterion symptoms, the severity of those symptoms, and the degree of functional disability.

Mild: Few, if any, symptoms in excess of those required to make the diagnosis are present, the intensity of the symptoms is distressing but manageable, and the symptoms result in minor impairment in social or occupational functioning.

Moderate: The number of symptoms, intensity of symptoms, and/or functional impairment are between those specified for “mild” and “severe.”

Severe: The number of symptoms is substantially in excess of that required to make the diagnosis, the intensity of the symptoms is seriously distressing and unmanageable, and the symptoms markedly interfere with social and occupational functioning.

[1]In distinguishing grief from a major depressive episode (MDE), it is useful to consider that in grief the predominant affect is feelings of emptiness and loss, while in an MDE it is persistent depressed mood and the inability to anticipate happiness or pleasure. The dysphoria in grief is likely to decrease in intensity over days to weeks and occurs in waves, the so-called pangs of grief. These waves tend to be associated with thoughts or reminders of the deceased. The depressed mood of an MDE is more persistent and not tied to specific thoughts or preoccupations. The pain of grief may be accompanied by positive emotions and humor that are uncharacteristic of the pervasive unhappiness and misery characteristic of an MDE. The thought content associated with grief generally features a preoccupation with thoughts and memories of the deceased, rather than the self-critical or pessimistic ruminations seen in an MDE. In grief, self-esteem is generally preserved, whereas in an MDE, feelings of worthlessness and self-loathing are common. If self-derogatory ideation is present in grief, it typically involves perceived failings vis-à-vis the deceased (e.g., not visiting frequently enough, not telling the deceased how much he or she was loved). If a bereaved individual thinks about death and dying, such thoughts are generally focused on the deceased and possibly about "joining" the deceased, whereas in an MDE such thoughts are focused on ending one's own life because of feeling worthless, undeserving of life, or unable to cope with the pain of depression.

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Depressive Disorders

Depressive disorders include disruptive mood dysregulation disorder, major depressive disorder (including major depressive episode), persistent depressive disorder, premenstrual dysphoric disorder, substance/medication-induced depressive disorder, depressive disorder due to another medical condition, other specified depressive disorder, and unspecified depressive disorder. The common feature of all of these disorders is the presence of sad, empty, or irritable mood, accompanied by related changes that significantly affect the individual's capacity to function (e.g., somatic and cognitive changes in major depressive disorder and persistent depressive disorder). What differs among them are issues of duration, timing, or presumed etiology.

In order to address concerns in the United States and, increasingly, internationally about the potential for the overdiagnosis and treatment of bipolar disorder in children, a new diagnosis, disruptive mood dysregulation disorder, referring to the presentation of children with persistent irritability and frequent episodes of extreme behavioral dyscontrol, is added to the depressive disorders for children up to 12 years of age. Its placement in this chapter reflects the finding that children with this symptom pattern typically develop unipolar depressive disorders or anxiety disorders, rather than bipolar disorders, as they mature into adolescence and adulthood.

Major depressive disorder represents the classic condition in this group of disorders. It is characterized by discrete episodes of at least 2 weeks' duration (although most episodes last considerably longer) involving clear-cut changes in affect, cognition, and neurovegetative functions and interepisode remissions. A diagnosis based on a single episode is possible, although the disorder is a recurrent one in the majority of cases. Careful consideration should be given to the delineation of normal sadness and grief from a major depressive episode. Bereavement may induce great suffering,

but it does not typically induce an episode of major depressive disorder. When they do occur together, the depressive symptoms and functional impairment tend to be more severe and the prognosis is worse compared with bereavement that is not accompanied by major depressive disorder. Bereavement-related major depressive episodes tend to occur in persons with other vulnerabilities to depressive disorders.

A more chronic form of depression, persistent depressive disorder, can be diagnosed when the mood disturbance continues for at least 2 years in adults or 1 year in children. This diagnosis, new in DSM-5, includes the DSM-IV diagnostic categories of chronic major depression and dysthymia.

After careful scientific review of the evidence, premenstrual dysphoric disorder has been moved from an appendix of DSM-IV ("Criteria Sets and Axes Provided for Further Study") to Section II of DSM-5. Almost 20 years of additional research on this condition has confirmed a specific and treatment-responsive form of depressive disorder that begins sometime following ovulation and remits within a few days of menses and has a marked impact on functioning.

A large number of substances of abuse, some prescribed medications, and several medical conditions can be associated with depression-like phenomena. This fact is recognized in the diagnoses of substance/medication-induced depressive disorder and depressive disorder due to another medical condition.

Disruptive Mood Dysregulation Disorder

Diagnostic Criteria	**F34.81**

A. Severe recurrent temper outbursts manifested verbally (e.g., verbal rages) and/or behaviorally (e.g., physical aggression toward people or property) that are grossly out of proportion in intensity or duration to the situation or provocation.

B. The temper outbursts are inconsistent with developmental level.

C. The temper outbursts occur, on average, three or more times per week.

D. The mood between temper outbursts is persistently irritable or angry most of the day, nearly every day, and is observable by others (e.g., parents, teachers, peers).

E. Criteria A–D have been present for 12 or more months. Throughout that time, the individual has not had a period lasting 3 or more consecutive months without all of the symptoms in Criteria A–D.

F. Criteria A and D are present in at least two of three settings (i.e., at home, at school, with peers) and are severe in at least one of these.

G. The diagnosis should not be made for the first time before age 6 years or after age 18 years.

H. By history or observation, the age at onset of Criteria A–E is before 10 years.

I. There has never been a distinct period lasting more than 1 day during which the full symptom criteria, except duration, for a manic or hypomanic episode have been met.

   Note: Developmentally appropriate mood elevation, such as occurs in the context of a highly positive event or its anticipation, should not be considered as a symptom of mania or hypomania.

J. The behaviors do not occur exclusively during an episode of major depressive disorder and are not better explained by another mental disorder (e.g., autism spectrum disorder, posttraumatic stress disorder, separation anxiety disorder, persistent depressive disorder).

   Note: This diagnosis cannot coexist with oppositional defiant disorder, intermittent explosive disorder, or bipolar disorder, though it can coexist with others, including major depressive disorder, attention-deficit/hyperactivity disorder, conduct

disorder, and substance use disorders. Individuals whose symptoms meet criteria for both disruptive mood dysregulation disorder and oppositional defiant disorder should only be given the diagnosis of disruptive mood dysregulation disorder. If an individual has ever experienced a manic or hypomanic episode, the diagnosis of disruptive mood dysregulation disorder should not be assigned.

K. The symptoms are not attributable to the physiological effects of a substance or another medical or neurological condition.

Diagnostic Features

The core feature of disruptive mood dysregulation disorder is chronic severe, persistent irritability. This severe irritability has two prominent clinical manifestations, the first of which is frequent temper outbursts. These outbursts typically occur in response to frustration and can be verbal or behavioral (the latter in the form of aggression against property, self, or others). They must occur frequently (i.e., on average, three or more times per week) (Criterion C) over at least 1 year in at least two settings (Criteria E and F), such as in the home and at school, and they must be developmentally inappropriate (Criterion B). The second manifestation of severe irritability consists of chronic, persistently irritable or angry mood that is present between the severe temper outbursts. This irritable or angry mood must be characteristic of the child, being present most of the day, nearly every day, and noticeable by others in the child's environment (Criterion D).

The clinical presentation of disruptive mood dysregulation disorder must be carefully distinguished from presentations of other, related conditions, particularly pediatric bipolar disorder. In fact, disruptive mood dysregulation disorder was added to DSM-5 to address the considerable concern about the appropriate classification and treatment of children who present with chronic, persistent irritability relative to children who present with classic (i.e., episodic) bipolar disorder.

Some researchers view severe, nonepisodic irritability as characteristic of bipolar disorder in children, although both DSM-IV and DSM-5 require that both children and adults have distinct episodes of mania or hypomania to qualify for the diagnosis of bipolar I disorder. During the latter decades of the twentieth century, this contention by researchers that severe, nonepisodic irritability is a manifestation of pediatric mania coincided with an upsurge in the rates at which clinicians assigned the diagnosis of bipolar disorder to their pediatric patients. This sharp increase in rates appears to be attributable to clinicians combining at least two clinical presentations into a single category. That is, both classic, episodic presentations of mania and nonepisodic presentations of severe irritability have been labeled as bipolar disorder in children. In DSM-5, the term *bipolar disorder* is explicitly reserved for episodic presentations of bipolar symptoms. DSM-IV did not include a diagnosis designed to capture youths whose hallmark symptoms consisted of very severe, nonepisodic irritability, whereas DSM-5, with the inclusion of disruptive mood dysregulation disorder, provides a distinct category for such presentations.

Prevalence

Disruptive mood dysregulation disorder is common among children presenting to pediatric mental health clinics. Prevalence estimates of the disorder in the community are unclear. In a population-based cohort study of Brazilian 11-year-olds that used a specific module for disruptive mood dysregulation disorder, the prevalence was 2.5%.

Consistent gender differences in prevalence have not been reported in population samples, although clinic samples report a male preponderance. For example, up to 80% of children presenting to clinics in Turkey with features of disruptive mood dysregulation disorder in a chart review were boys. Data suggest that the diagnosis may be more common in younger age groups (e.g., 8.2% in a community sample of 6-year-olds in the United States).

Development and Course

The onset of disruptive mood dysregulation disorder must be before age 10 years, and the diagnosis should not be applied to children with a

developmental age of younger than 6 years. It is unknown whether the condition presents only in this age-delimited fashion. Because the symptoms of disruptive mood dysregulation disorder are likely to change as children mature, use of the diagnosis should be restricted to age groups similar to those in which validity has been established (6–18 years). Approximately half of children with disruptive mood dysregulation disorder living in a predominantly rural area in a large U.S. study continue to have symptoms that meet criteria for the condition 1 year later, although those children whose symptoms no longer meet the threshold for the diagnosis often have persistent, clinically impairing irritability. Rates of conversion from severe, nonepisodic irritability to bipolar disorder are very low. Instead, children with disruptive mood dysregulation disorder are at increased risk to develop unipolar depressive and/or anxiety disorders in adulthood.

Risk and Prognostic Factors

Temperamental. Children with chronic irritability typically exhibit complicated psychiatric histories. In such children, a relatively extensive history of chronic irritability is common, typically manifesting before full criteria for the syndrome are met. Such prediagnostic presentations may have qualified for a diagnosis of oppositional defiant

disorder. Many children with disruptive mood dysregulation disorder have symptoms that also meet criteria for attention-deficit/hyperactivity disorder (ADHD) and for an anxiety disorder, with such diagnoses often being present from a relatively early age. For some children, the criteria for major depressive disorder may also be met.

Environmental. Factors associated with disrupted family life, such as psychological abuse or neglect, parental psychiatric disorder, limited parental education, single-parent household, early trauma, death of a parent, parental grief, divorce, and malnutrition (e.g., vitamin deficiency), are associated with the core behaviors of disruptive mood dysregulation disorder.

Genetic and physiological. Data suggest that a family history of depression may be a risk factor for disruptive mood dysregulation disorder. Consistent with this, twin data suggest that the association between early irritability and later unipolar depression and anxiety may be, in part, genetically mediated.

Compared with children with pediatric bipolar disorder or other mental illnesses, those with disruptive mood dysregulation disorder exhibit both commonalities and differences in information-processing deficits. For example, face-emotion labeling deficits, as well as perturbed decision-making and cognitive control, are present in children with bipolar disorder as well as those with disruptive mood dysregulation disorder. Importantly, however, the same behavioral deficit may be associated with different patterns of neural dysfunction. There is also evidence for disorder-specific dysfunction, such as during tasks assessing attention deployment in response to emotional stimuli, which has demonstrated unique signs of dysfunction in children with chronic irritability.

Culture-Related Diagnostic Issues

Culture-related data on disruptive mood dysregulation disorder are limited. However, sociocultural factors affect the presentation of core psychological features of the disorder, including impulsivity as well as emotion, reward, threat, and behavior dysregulation, especially in settings characterized by severe social disruption, such as postconflict zones or communities affected by long-standing racism and discrimination. It is important to distinguish disruptive mood dysregulation disorder from adaptive responses to adversity that are context-dependent and transitory.

Sex- and Gender-Related Diagnostic Issues

There is some evidence from twin studies that while irritability has a strong genetic component in both sexes, patterns differ for boys and girls. For boys, genetic factors appear to account for an increasing amount of the variance of the phenotype of irritability throughout childhood. While genetic factors account for a large proportion of the variance of the irritability phenotype in school-age girls, this decreases into adolescence and young adulthood, with environmental influences playing a greater role.

How this genetic risk for irritability translates into risk and prognosis for disruptive mood dysregulation disorder, per se, is not yet known.

Functional Consequences of Disruptive Mood Dysregulation Disorder

Chronic, severe irritability, such as is seen in disruptive mood dysregulation disorder, is associated with marked disruption in a child's family and peer relationships, as well as in school performance. Because of their extremely low frustration tolerance, such children generally have difficulty succeeding in school; they are often unable to participate in the activities typically enjoyed by healthy children; their family life is severely disrupted by their outbursts and irritability; and they have trouble initiating or sustaining friendships. Levels of dysfunction in children with bipolar disorder and disruptive mood

dysregulation disorder are generally comparable. Both conditions cause severe disruption in the lives of the affected individual and his or her family. In both disruptive mood dysregulation disorder and pediatric bipolar disorder, aggression and psychiatric hospitalization are common.

Differential Diagnosis

Because chronically irritable children and adolescents typically present with complex histories, the diagnosis of disruptive mood dysregulation disorder must be made while considering the presence or absence of multiple other conditions. Despite the need to consider many other syndromes, differentiation of disruptive mood dysregulation disorder from bipolar disorder and oppositional defiant disorder requires particularly careful assessment.

Bipolar disorders. The central feature differentiating disruptive mood dysregulation disorder and bipolar disorders in children involves the longitudinal course of the core symptoms. In children, as in adults, bipolar I disorder and bipolar II disorder manifest as an episodic illness with discrete episodes of mood perturbation that can be differentiated from the child's

typical presentation. The mood perturbation that occurs during a manic episode is distinctly different from the child's usual mood. In addition, during a manic episode, the change in mood must be accompanied by the onset, or worsening, of associated cognitive, behavioral, and physical symptoms (e.g., distractibility, increased goal-directed activity), which are also present to a degree that is distinctly different from the child's usual baseline. Thus, in the case of a manic episode, parents (and, depending on developmental level, children) should be able to identify a distinct time period during which the child's mood and behavior were markedly different from usual. In contrast, the irritability of disruptive mood dysregulation disorder is persistent and is present over many months; while it may wax and wane to a certain degree, severe irritability is characteristic of the child with disruptive mood dysregulation disorder. Thus, while bipolar disorders are episodic conditions, disruptive mood dysregulation disorder is not. In fact, the diagnosis of disruptive mood dysregulation disorder cannot be assigned to a child who has ever experienced a full-duration hypomanic or manic episode (irritable or euphoric) or who has ever had a manic or hypomanic episode lasting more than 1 day. Another central differentiating feature between bipolar disorders and disruptive mood dysregulation disorder is the presence of elevated or expansive mood and grandiosity. These symptoms are common features of mania but are not characteristic of disruptive mood dysregulation disorder.

Oppositional defiant disorder. While symptoms of oppositional defiant disorder typically do occur in children with disruptive mood dysregulation disorder, mood symptoms of disruptive mood dysregulation disorder are relatively rare in children with oppositional defiant disorder. The key features that warrant the diagnosis of disruptive mood dysregulation disorder in children whose symptoms also meet criteria for oppositional defiant disorder are the presence of severe and frequently recurrent outbursts and a persistent disruption in mood between outbursts. In addition, the diagnosis of disruptive mood dysregulation disorder requires severe impairment in at least one setting (i.e., home, school, or among peers) and mild to moderate impairment in a second setting. For this reason, while most children whose symptoms meet criteria for disruptive mood dysregulation disorder will also have a presentation that meets criteria for

oppositional defiant disorder, the reverse is not the case. That is, in only approximately 15% of individuals with oppositional defiant disorder would criteria for disruptive mood dysregulation disorder be met. Moreover, even for children in whom criteria for both disorders are met, only the diagnosis of disruptive mood dysregulation disorder should be made. Finally, both the prominent mood symptoms in disruptive mood dysregulation disorder and the high risk for depressive and anxiety disorders in follow-up studies justify placement of disruptive mood dysregulation disorder among the depressive disorders in DSM-5. (Oppositional defiant disorder is included in the chapter “Disruptive, Impulse-Control, and Conduct

Disorders.”) This reflects the more prominent mood component among individuals with disruptive mood dysregulation disorder, as compared with individuals with oppositional defiant disorder. Nevertheless, it also should be noted that disruptive mood dysregulation disorder appears to carry a high risk for behavioral problems as well as mood problems.

Attention-deficit/hyperactivity disorder, major depressive disorder, anxiety disorders, and autism spectrum disorder.

Unlike children diagnosed with bipolar disorder or oppositional defiant disorder—for whom a diagnosis of disruptive mood dysregulation disorder cannot be given even if the symptoms meet diagnostic criteria for that disorder—children whose symptoms meet criteria for disruptive mood dysregulation disorder also can receive a comorbid diagnosis of ADHD, major depressive disorder, and/or anxiety disorder. However, children whose irritability is present only in the context of a major depressive episode or persistent depressive disorder should receive one of those diagnoses rather than disruptive mood dysregulation disorder. Children with disruptive mood dysregulation disorder may have symptoms that also meet criteria for an anxiety disorder and can receive both diagnoses, but children whose irritability is manifest only in the context of exacerbation of an anxiety disorder should receive the relevant anxiety disorder diagnosis rather than disruptive mood dysregulation disorder. In addition, children with autism spectrum disorders frequently present with temper outbursts when, for example, their routines are disturbed. In that instance, the temper

outbursts would be considered secondary to the autism spectrum disorder, and the child should not receive the diagnosis of disruptive mood dysregulation disorder.

Intermittent explosive disorder. Children with symptoms suggestive of intermittent explosive disorder present with instances of severe temper outbursts, much like children with disruptive mood dysregulation disorder. However, unlike disruptive mood dysregulation disorder, intermittent explosive disorder does not require the individual's mood to be persistently irritable or angry between outbursts. In addition, a diagnosis of intermittent explosive disorder involving verbal aggression or physical aggression that does not result in damage to property or physical injury to animals or other individuals occurring at least twice weekly can be made after only 3 months of symptoms, in contrast to the 12-month requirement for disruptive mood dysregulation disorder. Thus, these two diagnoses should not be made in the same child. For children with outbursts and intercurrent, persistent irritability, only the diagnosis of disruptive mood dysregulation disorder should be made.

Comorbidity

Rates of comorbidity in disruptive mood dysregulation disorder are extremely high. It is rare to find individuals whose symptoms meet criteria for disruptive mood dysregulation disorder alone. Comorbidity between disruptive mood dysregulation disorder and other DSM-defined syndromes appears higher than for many other pediatric mental illnesses; the strongest overlap is with oppositional defiant disorder. Not only is the overall rate of comorbidity high in disruptive mood dysregulation disorder, but also the range of comorbid illnesses appears particularly diverse. These children typically present to the clinic with a wide range of disruptive behavior, mood, anxiety, and even autism spectrum symptoms and diagnoses. However, children with disruptive mood dysregulation disorder should not have symptoms that meet criteria for bipolar disorder, as in that context, only the bipolar disorder diagnosis should be made. If children have symptoms that meet criteria for oppositional defiant disorder or intermittent explosive disorder *and* disruptive mood dysregulation disorder, only the diagnosis of disruptive mood dysregulation disorder should be assigned.

Also, as noted earlier, the diagnosis of disruptive mood dysregulation disorder should not be assigned if the symptoms occur only in an anxiety-provoking context, when the routines of a child with autism spectrum disorder or obsessive-compulsive disorder are disturbed, or in the context of a major depressive episode.

Major Depressive Disorder

Diagnostic Criteria

A. Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure.

   Note: Do not include symptoms that are clearly attributable to another medical condition.

   1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)
   2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
   3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**Note:** In children, consider failure to make expected weight gain.)
   4. Insomnia or hypersomnia nearly every day.

5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).
6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The episode is not attributable to the physiological effects of a substance or another medical condition.

Note: Criteria A–C represent a major depressive episode.

Note: Responses to a significant loss (e.g., bereavement, financial ruin, losses from a natural disaster, a serious medical illness or disability) may include the feelings of intense sadness, rumination about the loss, insomnia, poor appetite, and weight loss noted in Criterion A, which may resemble a depressive episode. Although such symptoms may be understandable or considered appropriate to the loss, the presence of a major depressive episode in addition to the normal response to a significant loss should also be carefully considered. This decision inevitably requires the exercise of clinical judgment based on the individual's history and the cultural norms for the expression of distress in the context of loss.[1]

D. At least one major depressive episode is not better explained by schizoaffective disorder and is not superimposed on

schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorders.

E. There has never been a manic episode or a hypomanic episode.

Note: This exclusion does not apply if all of the manic-like or hypomanic-like episodes are substance-induced or are attributable to the physiological effects of another medical condition.

Coding and Recording Procedures

The diagnostic code for major depressive disorder is based on whether this is a single or recurrent episode, current severity, presence of psychotic features, and remission status. Current severity and psychotic features are only indicated if full criteria are currently met for a major depressive episode. Remission specifiers are only indicated if the full criteria are not currently met for a major depressive episode. Codes are as follows:

Severity/course specifier	Single episode	Recurrent episode*
Mild (p. 214)	F32.0	F33.0
Moderate (p. 214)	F32.1	F33.1
Severe (p. 214)	F32.2	F33.2
With psychotic features** (pp. 212–213)	F32.3	F33.3
In partial remission (p. 214)	F32.4	F33.41
In full remission (p. 214)	F32.5	F33.42
Unspecified	F32.9	F33.9

*For an episode to be considered recurrent, there must be an interval of at least 2 consecutive months between separate episodes in which criteria are not met for a major depressive episode. The definitions of specifiers are found on the indicated pages.

**If psychotic features are present, code the "with psychotic features" specifier irrespective of episode severity.

In recording the name of a diagnosis, terms should be listed in the following order: major depressive disorder, single or recurrent episode, severity/psychotic/remission specifiers, followed by as many of the following specifiers without codes that apply to the current episode (or the most recent episode if the major depressive disorder is in partial or full remission). **Note:** The specifier "with seasonal pattern" describes the pattern of recurrent major depressive episodes.

Specify if:

With anxious distress (pp. 210–211)

With mixed features (p. 211)

With melancholic features (pp. 211–212)

With atypical features (p. 212)

With mood-congruent psychotic features (p. 213)

With mood-incongruent psychotic features (p. 213)

With catatonia (p. 213). **Coding note:** Use additional code F06.1.

With peripartum onset (p. 213)

With seasonal pattern (applies to pattern of recurrent major depressive episodes) (p. 214)

Diagnostic Features

Major depressive disorder is defined by the presence of at least one major depressive episode occurring in the absence of a history of manic or hypomanic episodes. The essential feature of a major depressive episode is a period lasting at least 2 weeks during which there is either depressed mood or the loss of interest or pleasure in all or nearly all activities for most of the day nearly every day (Criterion A). The individual must also

experience at least four additional symptoms during the same 2-week period, drawn from a list that includes changes in appetite or weight, sleep, and psychomotor activity; decreased energy; feelings of worthlessness or guilt; difficulty thinking, concentrating, or making decisions; or thoughts of death, suicidal ideation, a suicide attempt, or a specific plan for suicidal behavior. To count toward a diagnosis of a major depressive episode, a symptom must either be newly present or have clearly worsened compared with the individual's pre-episode status. Moreover, the symptoms must occur nearly every day, for at least 2 consecutive weeks, with the exception of thoughts of death and suicidal ideation, which must be recurrent, and attempting suicide or making a specific plan, which only needs to occur once. The episode must be accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning. For some individuals with milder episodes, functioning may appear to be normal but requires markedly increased effort. The presenting complaint is often insomnia or fatigue rather than depressed mood or loss of interest; thus, the failure to probe for accompanying depressive symptoms can result in underdiagnosis. Fatigue and sleep disturbance are present in a high proportion of cases; psychomotor disturbances are much less common but are indicative of greater overall severity, as is the presence of delusional or near-delusional guilt.

The mood in a major depressive episode is often described by the individual as depressed, sad, hopeless, discouraged, or "down in the dumps" (Criterion A1). In some cases, sadness may be denied at first but may subsequently be elicited by interview (e.g., by pointing out that the individual looks as if he or she is about to cry). In some individuals who complain of feeling "blah," having no feelings, or feeling anxious, the presence of a depressed mood can be inferred from the individual's facial expression and demeanor. Some individuals emphasize somatic complaints (e.g., bodily aches and pains) rather than reporting feelings of sadness. Many individuals report or exhibit increased irritability (e.g., persistent anger, a tendency to respond to events with angry outbursts or blaming others, an exaggerated sense of frustration over minor matters). In children and adolescents, an irritable or cranky mood may develop rather than a sad or dejected mood. This presentation should be differentiated from a pattern of irritability when frustrated.

Diminished interest or pleasure in usual activities is nearly always present, at least to some degree. Individuals may report feeling less interested in hobbies, “not caring anymore,” or not feeling any enjoyment in activities that were previously considered pleasurable (Criterion A2). Family members often notice social withdrawal or neglect of pleasurable avocations (e.g., a formerly avid golfer no longer plays, a child who used to enjoy soccer finds excuses not to practice). In some individuals, there is a significant reduction from previous levels of sexual interest or desire.

Appetite change may involve either a reduction or an increase. Some depressed individuals report that they have to force themselves to eat. Others may eat more and may crave specific foods (e.g., sweets or other carbohydrates). When appetite changes are severe (in either direction), there may be a significant loss or gain in weight, or, in children, a failure to make expected weight gains may be noted (Criterion A3).

Sleep disturbance may take the form of either difficulty sleeping or sleeping excessively (Criterion A4). When insomnia is present, it typically takes the form of middle insomnia (i.e., waking up during the night and then having difficulty returning to sleep) or terminal insomnia (i.e., waking too early and being unable to return to sleep). Initial insomnia (i.e., difficulty falling asleep) may also occur. Individuals who present with oversleeping (hypersomnia) may experience prolonged sleep episodes at night or increased daytime sleep. Sometimes the reason that the individual seeks treatment is for the disturbed sleep.

Psychomotor changes include agitation (e.g., the inability to sit still, pacing, hand-wringing; or pulling or rubbing of the skin, clothing, or other objects) or retardation (e.g., slowed speech, thinking, and body movements; increased pauses before answering; speech that is decreased in volume, inflection, amount, or variety of content, or muteness) (Criterion A5). The psychomotor agitation or retardation must be severe enough to be observable by others and not represent merely subjective feelings. Individuals who display either psychomotor disturbance (i.e., psychomotor agitation or retardation) are likely to have histories of the other.

Decreased energy, tiredness, and fatigue are common (Criterion A6). An individual may report sustained fatigue without physical exertion. Even the smallest tasks seem to require substantial effort. The efficiency with which tasks are accomplished may be reduced. For example, an individual may complain that washing and dressing in the morning are exhausting and take twice as long as usual. This symptom accounts for much of the impairment resulting from major depressive disorder, both during acute episodes and when remission is incomplete.

The sense of worthlessness or guilt associated with a major depressive episode may include unrealistic negative evaluations of one's worth or guilty preoccupations or ruminations over minor past failings (Criterion A7). Such individuals often misinterpret neutral or trivial day-to-day events as evidence of personal defects and have an exaggerated sense of responsibility for untoward events. The sense of worthlessness or guilt may be of delusional proportions (e.g., an individual who is convinced that he or she is personally responsible for world poverty). Blaming oneself for being sick and for failing to meet occupational or interpersonal responsibilities as a result of the depression is very common and, unless delusional, is not considered sufficient to meet this criterion.

Many individuals report impaired ability to think, concentrate, or make even minor decisions (Criterion A8). They may appear easily distracted or complain of memory difficulties. Those engaged in cognitively demanding pursuits are often unable to function. In children, a precipitous drop in grades may reflect poor concentration. In elderly individuals, memory difficulties may be the chief complaint and may be mistaken for early signs of a dementia ("pseudodementia"). When the major depressive episode is successfully treated, the memory problems often fully abate. However, in some individuals, particularly elderly persons, a major depressive episode may sometimes be the initial presentation of an irreversible dementia.

Thoughts of death, suicidal ideation, or suicide attempts (Criterion A9) are common. They may range from a passive wish not to awaken in the morning or a belief that others would be better off if the individual were dead, to transient but recurrent thoughts of dying by suicide, to a specific suicide plan. More severely suicidal individuals may have put their affairs in order (e.g., updated wills, settled debts), acquired needed materials (e.g., a rope or a gun), and chosen a location and time to accomplish the suicide.

Motivations for suicide may include a desire to give up in the face of perceived insurmountable obstacles,

an intense wish to end what is perceived as an unending and excruciatingly painful emotional state, an inability to foresee any enjoyment in life, or the wish to not be a burden to others. The resolution of such thinking may be a more meaningful measure of diminished suicide risk than denial of further plans for suicide.

The degree of impairment associated with a major depressive episode varies, but even in milder cases, there must be either clinically significant distress or some interference in social, occupational, or other important areas of functioning (Criterion B). If impairment is severe, the individual may lose the ability to function socially or occupationally. In extreme cases, the individual may be unable to perform minimal self-care (e.g., feeding and clothing self) or to maintain minimal personal hygiene.

The individual's report of symptoms may be compromised by difficulties in concentrating, impaired memory, or a tendency to deny, discount, or explain away symptoms. Information from additional informants can be especially helpful in clarifying the course of current or prior major depressive episodes and in assessing whether there have been any manic or hypomanic episodes. Because major depressive episodes can begin gradually, a review of clinical information that focuses on the worst part of the current episode may be most likely to detect the presence of symptoms.

The evaluation of the symptoms of a major depressive episode is especially difficult when they occur in an individual who also has another medical condition (e.g., cancer, stroke, myocardial infarction, diabetes, pregnancy). Some of the criterion signs and symptoms of a major depressive episode are identical to those of another medical condition (e.g., weight loss with untreated diabetes; fatigue with cancer; hypersomnia early in pregnancy; insomnia later in pregnancy or the postpartum). Such symptoms count toward a major depressive diagnosis except when they are clearly and fully attributable to another medical condition. Nonvegetative symptoms of dysphoria, anhedonia, guilt or worthlessness, impaired

concentration or indecision, and suicidal thoughts should be assessed with particular care in such cases. Definitions of major depressive episodes that have been modified to include only these nonvegetative symptoms appear to identify nearly the same individuals as do the full criteria.

Associated Features

Major depressive disorder is associated with high mortality, much of which is accounted for by suicide; however, it is not the only cause. For example, depressed individuals admitted to nursing homes have a markedly increased likelihood of death in the first year. Individuals frequently present with tearfulness, irritability, brooding, obsessive rumination, anxiety, phobias, excessive worry over physical health, and complaints of pain (e.g., headaches; joint, abdominal, or other pains). In children, separation anxiety may occur.

Although an extensive literature exists describing neuroanatomical, neuroendocrinological, and neurophysiological correlates of major depressive disorder, no laboratory test has yielded results of sufficient sensitivity and specificity to be used as a diagnostic tool for this disorder. Until recently, hypothalamic-pituitary-adrenal axis hyperactivity had been the most extensively investigated abnormality associated with major depressive episodes, and it appears to be associated with melancholia (a particularly severe type of depression), psychotic features, and risks for eventual suicide. Molecular studies have also implicated peripheral factors, including genetic variants in neurotrophic factors and pro-inflammatory cytokines. Additionally, volumetric and functional magnetic resonance imaging studies provide evidence for abnormalities in specific neural systems supporting emotion processing, reward seeking, and emotion regulation in adults with major depression.

Prevalence

Twelve-month prevalence of major depressive disorder in the United States is approximately 7%, with marked differences by age group such that the prevalence in 18- to 29-year-old

individuals is threefold higher than the prevalence in individuals age 60 years or older. The most reproducible finding in the epidemiology of major depressive disorder has been a higher prevalence in females, an effect that peaks in adolescence and then stabilizes. Women experience approximately twofold higher rates than men, especially between menarche and menopause. Women report more atypical symptoms of depression characterized by hypersomnia, increased appetite, and leaden paralysis compared with men.

Systematic reviews show that the 12-month and point prevalence of major depressive disorder vary eight- to ninefold across global geographic regions. In the United States, prevalence increased from 2005 to 2015, with steeper rates of increase for youth compared with older groups. After stratification by ethnoracial groups, non-Hispanic Whites showed a significant increase in prevalence after adjustment for demographic characteristics, whereas no significant change in rate of depression was observed among non-Hispanic Blacks or Hispanics.

Development and Course

Major depressive disorder may first appear at any age, but the likelihood of onset increases markedly with puberty. In the United States, incidence appears to peak in the 20s; however, first onset in late life is not uncommon.

The course of major depressive disorder is quite variable, such that some individuals rarely, if ever, experience remission (a period of 2 or more months with no symptoms, or only one or two symptoms to no more than a mild degree), while others experience many years with few or no symptoms between discrete episodes. The course of depression may reflect social-structural adversity associated with poverty, racism, and marginalization.

It is important to distinguish individuals who present for treatment during an exacerbation of a chronic depressive illness from those whose symptoms developed recently. Chronicity of depressive symptoms substantially increases the likelihood of underlying personality, anxiety, and substance use disorders and decreases the likelihood that treatment will be followed by full symptom resolution. It is therefore useful to ask individuals presenting with depressive symptoms to identify the last period of at least 2 months during which they were entirely free of depressive

symptoms. Cases in which depressive symptoms are present for more days than not might warrant an additional diagnosis of persistent depressive disorder.

Recovery from a major depressive episode begins within 3 months of onset for 40% of individuals with major depression and within 1 year for 80% of individuals. Recency of onset is a strong determinant of the likelihood of near-term recovery, and many individuals who have been depressed for only several months can be expected to recover spontaneously. Features associated with lower recovery rates, other than current episode duration, include psychotic features, prominent anxiety, personality disorders, and symptom severity.

The risk of recurrence becomes progressively lower over time as the duration of remission increases. The risk is higher in individuals whose preceding episode was severe, in younger individuals, and in individuals who have already experienced multiple episodes. The persistence of even mild depressive symptoms during remission is a powerful predictor of recurrence.

Many bipolar illnesses begin with one or more depressive episodes, and a substantial proportion of individuals who initially appear to have major depressive disorder will prove, in time, to instead have a bipolar disorder. This is more likely in individuals with onset of the illness in adolescence, those with psychotic features, and those with a family history of bipolar illness. The presence of a “with mixed features” specifier also increases the risk for future manic or hypomanic diagnosis. Major depressive disorder, particularly with psychotic features, may also transition into schizophrenia, a change that is much more frequent than the reverse.

There are no clear effects of current age on the course or treatment response of major depressive disorder. Some symptom differences exist, though, such that hypersomnia and hyperphagia are more likely in younger individuals, and melancholic symptoms, particularly psychomotor disturbances, are more common in older individuals. Depressions with earlier ages at onset are more familial and more likely to involve personality disturbances. The course of major depressive disorder within

individuals does not generally change with aging. Mean times to recovery do not change over multiple episodes, and the likelihood of being in an episode does not generally increase or decrease with time.

Risk and Prognostic Factors

Temperamental. Negative affectivity (neuroticism) is a well-established risk factor for the onset of major depressive disorder, and high levels appear to render individuals more likely to develop depressive episodes in response to stressful life events.

Environmental. Adverse childhood experiences, particularly when they are multiple and of diverse types, constitute a set of potent risk factors for major depressive disorder. Women may be disproportionately at risk for adverse childhood experiences, including sexual abuse, that may contribute to the increased prevalence of depression in this group. Other social determinants of mental health, such as low income, limited formal education, racism, and other forms of discrimination, are associated with higher risk of major depressive disorder. Stressful life events are well recognized as precipitants of major depressive episodes, but the presence or absence of adverse life events near the onset of episodes does not appear to provide a useful guide to prognosis or treatment selection. Etiologically, women are disproportionately affected by major risk factors for depression across the life span, including interpersonal trauma.

Genetic and physiological. First-degree family members of individuals with major depressive disorder have a risk for major depressive disorder two- to fourfold higher than that of the general population. Relative risks appear to be higher for early-onset and recurrent forms. Heritability is approximately 40%, and the personality trait neuroticism accounts for a substantial portion of this genetic liability.

Women may also be at risk for depressive disorders in relation to specific reproductive life stages, including in the premenstrual period, postpartum, and in perimenopause.

Course modifiers. Essentially all major nonmood disorders (i.e., anxiety, substance use, trauma- and stressor-related, feeding and eating, and obsessive-compulsive and related disorders) increase the risk of an

individual developing depression. Major depressive episodes that develop against the background of another disorder often follow a more refractory course. Substance use, anxiety, and borderline personality disorders are among the most common of these, and the presenting depressive symptoms may obscure and delay their recognition. However, sustained clinical improvement in depressive symptoms may depend on the appropriate treatment of underlying illnesses. Chronic or disabling medical conditions also increase risks for major depressive episodes. Prevalent illnesses such as diabetes, morbid obesity, and cardiovascular disease are often complicated by depressive episodes, and these episodes are more likely to become chronic than are depressive episodes in medically healthy individuals.

Culture-Related Diagnostic Issues

Although there is substantial cross-cultural variation in the prevalence, course, and symptomatology of depression, a syndrome similar to major depressive disorder can be identified across diverse cultural contexts. Symptoms commonly associated with depression across cultural contexts, not listed in the DSM criteria, include social isolation or loneliness, anger, crying, and diffuse pain. A wide range of other somatic complaints are

common and vary by cultural context. Understanding the significance of these symptoms requires exploring their meaning in local social contexts.

Symptoms of major depressive disorder may be underdetected or underreported, potentially leading to misdiagnosis, including overdiagnosis of schizophrenia spectrum disorders in some ethnic and racialized groups facing discrimination. Cross-nationally, higher levels of income inequality in a society are associated with higher prevalence of major depressive disorder. In the United States, the chronicity of major depressive disorder appears to be higher among African Americans and Caribbean Blacks compared with non-Latinx Whites, possibly because of the impact of racism, discrimination, greater sociostructural adversity, and lack of access to quality mental health care.

Sex- and Gender-Related Diagnostic Issues

There are no clear differences between genders in treatment response or functional consequences. There is some evidence for sex and gender differences in phenomenology and course of illness. Women tend to experience more disturbances in appetite and sleep, including atypical features such as hyperphagia and hypersomnia, and are more likely to experience interpersonal sensitivity and gastrointestinal symptoms. Men with depression, however, may be more likely than depressed women to report greater frequencies and intensities of maladaptive self-coping and problem-solving strategies, including alcohol or other drug misuse, risk taking, and poor impulse control.

Association With Suicidal Thoughts or Behavior

Age-adjusted rates of suicide in the United States have increased from 10.5 to 14.0 per 100,000 over the past two decades. An earlier review of the literature indicated that individuals with depressive illness have a 17-fold increased risk for suicide over the age- and sex-adjusted general population rate. The likelihood of suicide attempts lessens in middle and late life, although the risk of death by suicide does not. The possibility of suicidal behavior exists at all times during major depressive episodes. The most consistently described risk factor is a past history of suicide attempts or threats, but it should be remembered that most deaths by suicide are not preceded by nonfatal attempts. Anhedonia has a particularly strong association with suicidal ideation. Other features associated with an increased risk for death by suicide include being single, living alone, social disconnectedness, early life adversity, availability of lethal methods such as a firearm, sleep disturbance, cognitive and decision-making deficits, and having prominent feelings of hopelessness. Women attempt suicide at a higher rate than men, while men are more likely to complete suicide. The difference in suicide rate between men and women with depressive disorders is smaller than in the population as a whole, however. Comorbidities, including aggressive-impulsive traits, borderline personality disorder, substance use disorder, anxiety, other medical conditions, and functional impairment, increase risk for future suicidal behavior.

Functional Consequences of Major Depressive Disorder

Many of the functional consequences of major depressive disorder derive from individual symptoms. Impairment can be very mild, such that many of those who interact with the affected individual are unaware of depressive symptoms. Impairment may, however, range to complete incapacity such that the depressed individual is unable to attend to basic self-care needs or is mute or catatonic. For individuals seen in general medical settings, those with major depressive disorder have more pain and physical illness and greater decreases in physical, social, and role functioning. Depressed women report greater functional impairment in their relationships than men.

Differential Diagnosis

Manic episodes with irritable mood or with mixed features. Major depressive episodes with prominent irritable mood may be difficult to distinguish from manic episodes with irritable mood or with mixed features. This distinction requires a careful clinical evaluation of the presence of sufficient manic symptoms to meet threshold criteria (i.e., three if mood is manic, four if mood is irritable but not manic).

Bipolar I disorder, bipolar II disorder, or other specified bipolar and related disorder.

Major depressive episodes along with a history of a manic or hypomanic episode preclude the diagnosis of major depressive disorder. Major depressive episodes with a history of hypomanic episodes and without a history of manic episodes indicate a diagnosis of bipolar II disorder, whereas major depressive episodes with a history of manic episodes (with or without hypomanic episodes) indicate a diagnosis of bipolar I disorder. On the other hand, presentations of major depressive episodes with a history of periods of hypomania that do not meet criteria for a hypomanic episode may be diagnosed as either other specified bipolar and related disorder or major depressive disorder depending on where the clinician judges the presentation to best fall. For example, the presentation may be best considered other specified bipolar and related disorder because of the clinical significance of the subthreshold hypomanic symptoms, or the

presentation may be best considered a case of major depressive disorder with some subthreshold hypomanic symptoms in between episodes.

Depressive disorder due to another medical condition. A diagnosis of depressive disorder due to another medical condition requires the presence of an etiological medical condition. Major depressive disorder is not diagnosed if the major depressive–like episodes are all attributable to the direct pathophysiological consequence of a specific medical condition (e.g., multiple sclerosis, stroke, hypothyroidism).

Substance/medication-induced depressive disorder. This disorder is distinguished from major depressive disorder by the fact that a substance (e.g., a drug of abuse, a medication, a toxin) appears to be etiologically related to the mood disturbance. For example, depressed mood that occurs only in the context of withdrawal from cocaine would be diagnosed as cocaine-induced depressive disorder.

Persistent depressive disorder. Persistent depressive disorder is characterized by depressed mood, more days than not, for at least 2 years. If criteria are met for both major depressive disorder and persistent depressive disorder, both can be diagnosed.

Premenstrual dysphoric disorder. Premenstrual dysphoric disorder is characterized by dysphoric mood that is present in the final week before the onset of menses, that starts to improve within a few days after the onset of menses, and that becomes minimal or absent in the week postmenses. By contrast, the episodes of major depressive disorder are not temporally connected to the menstrual cycle.

Disruptive mood dysregulation disorder. Disruptive mood dysregulation disorder is characterized by severe, recurrent temper outbursts manifested verbally and/or behaviorally, accompanied by persistent or labile mood, most of the day, nearly every day, in between the outbursts. In contrast, in major depressive disorder, irritability is confined to the major depressive episodes.

Major depressive episodes superimposed on schizophrenia, delusional disorder, schizophreniform disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder. Depressive symptoms may be present during schizophrenia, delusional disorder, schizophreniform disorder, or other specified or unspecified

schizophrenia spectrum and other psychotic disorder. Most commonly, such depressive symptoms can be considered associated features of these disorders and do not merit a

separate diagnosis. However, when the depressive symptoms meet full criteria for a major depressive episode, a diagnosis of other specified depressive disorder may be made in addition to the diagnosis of the psychotic disorder.

Schizoaffective disorder. Schizoaffective disorder differs from major depressive disorder, with psychotic features, by the requirement that in schizoaffective disorder, delusions or hallucinations are present for at least 2 weeks in the absence of a major depressive episode.

Attention-deficit/hyperactivity disorder. Distractibility and low frustration tolerance can occur in both attention-deficit/hyperactivity disorder (ADHD) and a major depressive episode; if the criteria are met for both, ADHD may be diagnosed in addition to the mood disorder. However, the clinician must be cautious not to overdiagnose a major depressive episode in children with ADHD whose disturbance in mood is characterized by irritability rather than by sadness or loss of interest.

Adjustment disorder with depressed mood. A major depressive episode that occurs in response to a psychosocial stressor is distinguished from adjustment disorder, with depressed mood, by the fact that the full criteria for a major depressive episode are not met in adjustment disorder.

Bereavement. Bereavement is the experience of losing a loved one to death. It generally triggers a grief response that may be intense and may involve many features that overlap with symptoms characteristic of a major depressive episode, such as sadness, difficulty sleeping, and poor concentration. Features that help differentiate a bereavement-related grief response from a major depressive episode include the following: the predominant affects in grief are feelings of emptiness and loss, whereas in a major depressive episode they are persistent depressed mood and a diminished ability to experience pleasure. Moreover, the dysphoric mood of grief is likely to decrease in intensity over days to weeks and occurs in

waves that tend to be associated with thoughts or reminders of the deceased, whereas the depressed mood in a major depressive episode is more persistent and not tied to specific thoughts or preoccupations. It is important to note that in a vulnerable individual (e.g., someone with a past history of major depressive disorder), bereavement may trigger not only a grief response but also the development of an episode of depression or the worsening of an existing episode.

Sadness. Finally, periods of sadness are inherent aspects of the human experience. These periods should not be diagnosed as a major depressive episode unless criteria are met for severity (i.e., five out of nine symptoms), duration (i.e., most of the day, nearly every day for at least 2 weeks), and clinically significant distress or impairment. The diagnosis other specified depressive disorder may be appropriate for presentations of depressed mood with clinically significant impairment that do not meet criteria for duration or severity.

Comorbidity

Other disorders with which major depressive disorder frequently co-occurs are substance-related disorders, panic disorder, generalized anxiety disorder, posttraumatic stress disorder, obsessive-compulsive disorder, anorexia nervosa, bulimia nervosa, and borderline personality disorder.

While women are more likely than men to report comorbid anxiety disorders, bulimia nervosa, and somatoform disorder (somatic symptom and related disorders), men are more likely to report comorbid alcohol and substance abuse.

Persistent Depressive Disorder

Diagnostic Criteria F34.1

This disorder represents a consolidation of DSM-IV-defined chronic major depressive disorder and dysthymic disorder.

A. Depressed mood for most of the day, for more days than not, as indicated by either subjective account or observation by others, for at least 2 years.

   Note: In children and adolescents, mood can be irritable and duration must be at least 1 year.

B. Presence, while depressed, of two (or more) of the following:

   1. Poor appetite or overeating.
   2. Insomnia or hypersomnia.
   3. Low energy or fatigue.
   4. Low self-esteem.
   5. Poor concentration or difficulty making decisions.
   6. Feelings of hopelessness.

C. During the 2-year period (1 year for children or adolescents) of the disturbance, the individual has never been without the symptoms in Criteria A and B for more than 2 months at a time.

D. Criteria for a major depressive disorder may be continuously present for 2 years.

E. There has never been a manic episode or a hypomanic episode.

F. The disturbance is not better explained by a persistent schizoaffective disorder, schizophrenia, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.

G. The symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hypothyroidism).

H. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: If criteria are sufficient for a diagnosis of a major depressive episode at any time during the 2-year period of depressed mood, then a separate diagnosis of major depression should be made in addition to the diagnosis of persistent depressive disorder along with the relevant specifier (e.g., with intermittent major depressive episodes, with current episode).

Specify if:

With anxious distress (pp. 210–211)

With atypical features (p. 212)

Specify if:

In partial remission (p. 214)

In full remission (p. 214)

Specify if:

Early onset: If onset is before age 21 years.

Late onset: If onset is at age 21 years or older.

Specify if (for most recent 2 years of persistent depressive disorder):

With pure dysthymic syndrome: Full criteria for a major depressive episode have not been met in at least the preceding 2 years.

With persistent major depressive episode: Full criteria for a major depressive episode have been met throughout the preceding 2-year period.

With intermittent major depressive episodes, with current episode: Full criteria for a major depressive episode are currently met, but there have been periods of at least 8 weeks in at least the preceding 2 years with symptoms below the threshold for a full major depressive episode.

With intermittent major depressive episodes, without current episode: Full criteria for a major depressive episode

are not currently met, but there has been one or more major depressive episodes in at least the preceding 2 years.

Specify current severity:

Mild (p. 214)

Moderate (p. 214)

Severe (p. 214)

Diagnostic Features

The essential feature of persistent depressive disorder is a depressed mood that occurs for most of the day, for more days than not, for at least 2 years, or at least 1 year for children and adolescents (Criterion A). This disorder represents a consolidation of DSM-IV-defined chronic major depressive disorder and dysthymic disorder. Major depression may precede persistent depressive disorder, and major depressive episodes may occur during persistent depressive disorder. Individuals whose symptoms meet major depressive disorder criteria for 2 years should be given a diagnosis of persistent depressive disorder as well as major depressive disorder.

Individuals with persistent depressive disorder describe their mood as sad or "down in the dumps." During periods of depressed mood, at least two of the six symptoms from Criterion B are present. Because these symptoms have become a part of the individual's day-to-day experience, particularly in the case of early onset (e.g., "I've always been this way"), they may not be reported unless the individual is directly prompted. During the 2-year period (1 year for children or adolescents), any symptom-free intervals that have occurred have lasted no longer than 2 months (Criterion C).

Prevalence

Persistent depressive disorder is effectively an amalgam of DSM-IV dysthymic disorder and chronic major depressive episode. The 12-month prevalence in the United States is approximately 0.5% for dysthymic disorder and 1.5% for chronic major depressive disorder, with prevalence among women approximately 1.5 and 2 times higher than prevalence

among men for each of these diagnoses, respectively. Based on studies using comparable ascertainment procedures, the lifetime and 12-month estimates of DSM-IV dysthymia may be higher in high-income than in low- and middle-income countries. However, the disorder is associated with elevated risk of suicidal outcomes and comparable levels of disability wherever it occurs.

Development and Course

Persistent depressive disorder often has an early and insidious onset (i.e., in childhood, adolescence, or early adult life) and, by definition, a chronic course. Borderline personality disorder is a particularly robust risk factor for persistent depressive disorder. When persistent depressive disorder and borderline personality disorder coexist, the covariance of the corresponding features over time suggests the operation of a common mechanism. Early onset (i.e., before age 21 years) is associated with a higher likelihood of comorbid personality disorders and substance use disorders.

When symptoms rise to the level of a major depressive episode, they are likely to subsequently revert to a lower level. However, depressive symptoms are much less likely to resolve fully in a given period of time in the context of persistent depressive disorder than they are in a nonchronic major depressive episode.

Risk and Prognostic Factors

Temperamental. Factors predictive of poorer long-term outcome include higher levels of negative affectivity (neuroticism), greater symptom severity, poorer global functioning, and presence of anxiety disorders or conduct disorder.

Environmental. Childhood risk factors include parental loss or separation and childhood adversity.

Genetic and physiological. There are no clear differences in illness development, course, or family history between DSM-IV dysthymic disorder and chronic major depressive disorder. Earlier findings pertaining

to either disorder are therefore likely to apply to persistent depressive disorder. It is thus likely that individuals with persistent depressive disorder will have a higher proportion of first-degree relatives with persistent depressive disorder than do individuals with nonchronic major depressive disorder, and more depressive disorders in general.

A number of brain regions (e.g., prefrontal cortex, anterior cingulate, amygdala, hippocampus) have been implicated in persistent depressive disorder. Possible polysomnographic abnormalities exist as well.

Culture-Related Diagnostic Issues

The perceived abnormality or tolerance of chronic depressive symptoms may vary across cultures, affecting symptom detection and treatment acceptability. For example, some social groups or age cohorts may consider long-standing depressive symptoms to be normal reactions to adversity.

Association With Suicidal Thoughts or Behavior

Persistent depressive disorder is associated with elevated risk of suicidal outcomes and comparable levels of disability, whether the disorder occurs in high-, middle-, or low-income countries.

Functional Consequences of Persistent Depressive Disorder

The degree to which persistent depressive disorder impacts social and occupational functioning is likely to vary widely, but effects can be as great as or greater than those of major depressive disorder.

Differential Diagnosis

Major depressive disorder. If there is a depressed mood for more days than not plus two or more persistent depressive disorder Criterion B symptoms for 2 years or more, then the diagnosis of persistent depressive disorder is made. If the symptom criteria are sufficient for a diagnosis of a major depressive episode at any time during this period, then the additional diagnosis of major depression should be made. The comorbid presence of major depressive episodes during this period should also be noted by

assigning the appropriate course specifier to the persistent depressive disorder diagnosis as follows: If the individual's symptoms currently meet full criteria for a major depressive episode, and there have been periods of at least 8 weeks in at least the preceding 2 years with symptoms below the threshold for a full major depressive episode, then the specifier "with intermittent major depressive episodes, with current episode" would be assigned. If full criteria for a major depressive episode are not currently met but there has been one or more major depressive episodes in at least the preceding 2 years, then the specifier "with intermittent major depressive episodes, without current episode" is assigned. If a major depressive episode

has persisted for at least a 2-year duration and remains present, then the specifier "with persistent major depressive episode" is used. If the individual has not experienced an episode of major depression in the last 2 years, then the specifier "with pure dysthymic syndrome" is used.

Other specified depressive disorder. Because the criteria for a major depressive episode include symptoms (i.e., markedly diminished interest or pleasure in activities; psychomotor agitation or retardation; recurrent thoughts of death, suicidal ideation, suicide attempt or plan) that are absent from the symptom list for persistent depressive disorder (i.e., depressed mood and two out of six Criterion B symptoms), a very limited number of individuals will have depressive symptoms that have persisted longer than 2 years but that do not meet criteria for persistent depressive disorder. If full criteria for a major depressive episode have been met at some point during the current episode of illness, a diagnosis of major depressive disorder would apply. Otherwise, a diagnosis of other specified depressive disorder or unspecified depressive disorder should be given.

Bipolar I and bipolar II disorders. A history of a manic or hypomanic episode precludes the diagnosis of persistent depressive disorder. A history of manic episodes (with or without hypomanic episodes) indicates a diagnosis of bipolar I disorder. A history of hypomanic episodes (without any history of manic episodes in individuals with persistent depressive presentations

during which criteria have been met for a major depressive episode) warrants a diagnosis of bipolar II disorder. Other specified bipolar disorder applies to individuals whose presentations include a history of hypomanic episodes along with persistent depressive presentation that has never met full criteria for a major depressive episode.

Cyclothymic disorder. A diagnosis of cyclothymic disorder precludes the diagnosis of persistent depressive disorder. Thus, if during the period lasting at least 2 years of depressed mood for most of the day, for more days than not, 1) there are numerous periods with hypomanic symptoms that do not meet criteria for a hypomanic episode, 2) there have not been any symptom-free periods of more than 2 months at a time, and 3) criteria have never been met for a major depressive, manic, or hypomanic episode, then the diag-nosis would be cyclothymic disorder instead of persistent depressive disorder.

Psychotic disorders. Depressive symptoms are a common associated feature of chronic psychotic disorders (e.g., schizoaffective disorder, schizophrenia, delusional disorder). A separate diagnosis of persistent depressive disorder is not made if the symptoms occur only during the course of the psychotic disorder (including residual phases).

Depressive or bipolar and related disorder due to another medical condition. Persis tent depressive disorder must be distinguished from a depressive or bipolar and related disorder due to another medical condition. The diagnosis is depressive or bipolar and related disorder due to another medical condition if the mood disturbance is judged, based on history, physical examination, or laboratory findings, to be attributable to the direct pathophysiological effects of a specific, usually chronic, medical condition (e.g., multiple sclerosis). If it is judged that the depressive symptoms are not attributable to the physiological effects of another medical condition, then the primary mental disorder (e.g., persistent depressive disorder) is recorded, and the medical condition is noted as a concomitant medical condition (e.g., diabetes mellitus).

Substance/medication-induced depressive or bipolar and related disorder. A substance/medication-induced depressive or bipolar and related disorder is distinguished from persistent depressive disorder when a substance (e.g., a

drug of abuse, a medication, a toxin) is judged to be etiologically related to the mood disturbance.

Personality disorders. A personality disorder is characterized by an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the

individual's culture, with onset by adolescence or early adulthood. Personality disorders commonly co-occur with persistent depressive disorder. If criteria are met for persistent depressive disorder and a personality disorder, both may be diagnosed.

Comorbidity

In comparison to individuals with major depressive disorder, those with persistent depressive disorder are at higher risk for psychiatric comorbidity in general, and for anxiety disorders, substance use disorders, and personality disorders in particular. Early-onset persistent depressive disorder is strongly associated with DSM-5 Cluster B and C personality disorders.

Premenstrual Dysphoric Disorder

Diagnostic Criteria F32.81

A. In the majority of menstrual cycles, at least five symptoms must be present in the final week before the onset of menses, start to *improve* within a few days after the onset of menses, and become *minimal* or absent in the week postmenses.

B. One (or more) of the following symptoms must be present:

   1. Marked affective lability (e.g., mood swings; feeling suddenly sad or tearful, or increased sensitivity to rejection).

2. Marked irritability or anger or increased interpersonal conflicts.
3. Marked depressed mood, feelings of hopelessness, or self-deprecating thoughts.
4. Marked anxiety, tension, and/or feelings of being keyed up or on edge.

C. One (or more) of the following symptoms must additionally be present, to reach a total of *five* symptoms when combined with symptoms from Criterion B above.

1. Decreased interest in usual activities (e.g., work, school, friends, hobbies).
2. Subjective difficulty in concentration.
3. Lethargy, easy fatigability, or marked lack of energy.
4. Marked change in appetite; overeating; or specific food cravings.
5. Hypersomnia or insomnia.
6. A sense of being overwhelmed or out of control.
7. Physical symptoms such as breast tenderness or swelling, joint or muscle pain, a sensation of “bloating,” or weight gain.

Note: The symptoms in Criteria A–C must have been met for most menstrual cycles that occurred in the preceding year.

D. The symptoms cause clinically significant distress or interference with work, school, usual social activities, or relationships with others (e.g., avoidance of social activities; decreased productivity and efficiency at work, school, or home).

E. The disturbance is not merely an exacerbation of the symptoms of another disorder, such as major depressive disorder, panic disorder, persistent depressive disorder, or a personality disorder (although it may co-occur with any of these disorders).

F. Criterion A should be confirmed by prospective daily ratings during at least two symptomatic cycles. (**Note:** The diagnosis may be made provisionally prior to this confirmation.)

G. The symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, other treatment) or another medical condition (e.g., hyperthyroidism).

Recording Procedures

If symptoms have not been confirmed by prospective daily ratings of at least two symptomatic cycles, “provisional” should be noted after the name of the diagnosis (i.e., “premenstrual dysphoric disorder, provisional”).

Diagnostic Features

The essential features of premenstrual dysphoric disorder are the expression of mood lability, irritability, dysphoria, and anxiety symptoms that occur repeatedly during the premenstrual phase of the cycle and remit around the onset of menses or shortly thereafter. These symptoms may be accompanied by behavioral and physical symptoms. Symptoms must have occurred in most of the menstrual cycles during the past year and must have an adverse effect on work or social functioning. The intensity and/or expressivity of the accompanying symptoms may be closely related to social and cultural background characteristics as well as religious beliefs, social tolerance, attitude toward the female reproductive cycle, and female gender role issues more generally.

Typically, symptoms peak around the time of the onset of menses. Although it is not uncommon for symptoms to linger into the first few days of menses, the individual must have a symptom-free period in the follicular phase after the menstrual period begins. While the core symptoms include mood and anxiety symptoms, behavioral and somatic symptoms commonly also occur. However, the presence of somatic and/or behavioral symptoms in the absence of mood and/or anxious symptoms is not sufficient for a

diagnosis. Symptoms are of comparable severity (but not duration) to those of other mental disorders, such as a major depressive episode or generalized anxiety disorder. In order to confirm a provisional diagnosis, daily prospective symptom ratings are required for at least two symptomatic cycles.

Symptoms must cause clinically significant distress and/or an obvious and marked impairment in the ability to function socially or occupationally in the week prior to menses.

Associated Features

Delusions and hallucinations have been described in the late luteal phase of the menstrual cycle but are rare.

Prevalence

The 12-month prevalence of premenstrual dysphoric disorder in the community has been estimated at 5.8% based on a large study from Germany. Another study that looked at prevalence over two menstrual cycles found 1.3% of menstruating women with the disorder in the United States. Estimates based on retrospective reports are often higher than those based on prospective daily ratings. Yet, estimates based on a daily record of symptoms for 1–2 months may not be fully representative, because those with the most severe symptoms may be unable to sustain the rating process. The most rigorous estimate of premenstrual dysphoric disorder prevalence in the United States using prospective ratings of two consecutive menstrual cycles was 1.3% for women whose symptoms met diagnostic criteria, who experienced functional impairment, and had no co-occurring mental disorder. The prevalence of premenstrual dysphoric disorder symptoms in adolescent girls may be higher than that observed in adult women.

Development and Course

Onset of premenstrual dysphoric disorder can occur at any point after menarche. Incidence of new cases over a 40-month follow-up period in Germany is 2.5% (95% confidence interval = 1.7–3.7). Symptoms cease

after menopause, although cyclical hormone replacement can trigger the re-expression of symptoms.

Risk and Prognostic Factors

Environmental. Environmental factors associated with the expression of premenstrual dysphoric disorder include stress, history of interpersonal trauma, seasonal changes, and sociocultural aspects of female sexual behavior in general, and female gender roles in particular.

Genetic and physiological. No studies have examined heritability in premenstrual dysphoric disorder specifically. Estimates for heritability of premenstrual dysphoric symptoms range between 30% and 80%, although it remains unclear whether the symptoms themselves are heritable or whether they are simply associated with other heritable factors or traits.

Culture-Related Diagnostic Issues

Premenstrual dysphoric disorder has been observed in individuals in the United States, Europe, India, Nigeria, Brazil, and Asia, with a broad prevalence range. Nevertheless, as with most mental disorders, frequency, intensity, and expressivity of symptoms; perceived consequences; help-seeking patterns; and management may be significantly influenced by social and cultural factors, such as a history of sexual abuse or domestic violence, limited social support, and cultural variations in attitudes toward menstruation.

Diagnostic Markers

As indicated earlier, the diagnosis of premenstrual dysphoric disorder is appropriately confirmed by 2 months of prospective symptom ratings. A number of scales, including the Daily Rating of Severity of Problems and the Visual Analogue Scales for Premenstrual Mood Symptoms, have undergone validation and are commonly used in clinical trials for premenstrual dysphoric disorder. The Premenstrual Tension Syndrome Rating Scale has a self-report and an observer version, both of which have

been validated and used widely to measure illness severity in women who have premenstrual dysphoric disorder.

Association With Suicidal Thoughts or Behavior

The premenstrual phase has been considered by some to be a risk period for suicide.

Functional Consequences of Premenstrual Dysphoric Disorder

Impairment in social functioning may be manifested by discord in the intimate partner relationship and problems with children, other family members, or friends that occur only in association with the premenstrual dysphoric disorder (i.e., as opposed to chronic interpersonal problems). Impairments in work and health-related quality of life are also prominent. There is evidence that premenstrual dysphoric disorder can be associated with impairments in function and health-related quality of life that are on par with those observed in major depressive disorder and persistent depressive disorder.

Differential Diagnosis

Premenstrual syndrome. Premenstrual syndrome differs from premenstrual dysphoric disorder in that premenstrual syndrome does not require a minimum of five symptoms nor mood-related symptomatology, and it is generally considered to be less severe than premenstrual dysphoric disorder. Premenstrual syndrome may be more common than premenstrual dysphoric disorder; its estimated prevalence varies with numbers that hover at about 20%. While premenstrual syndrome shares the feature of symptom expression during the premenstrual phase of the menstrual cycle, the presence of somatic or

behavioral symptoms, without the required affective symptoms, likely meets criteria for premenstrual syndrome and not for premenstrual dysphoric disorder.

Dysmenorrhea. Dysmenorrhea is a syndrome of painful menses, but this is distinct from a syndrome characterized by affective changes. Moreover, symptoms of dysmenorrhea begin with the onset of menses, whereas symptoms of premenstrual dysphoric disorder, by definition, begin before the onset of menses, even if they linger into the first few days of menses.

Bipolar disorder, major depressive disorder, and persistent depressive disorder. M any women with (either naturally occurring or substance/medication-induced) bipolar or major depressive disorder or persistent depressive disorder believe that they have premenstrual dysphoric disorder. However, when they chart symptoms, they realize that the symptoms do not follow a premenstrual pattern. Because the onset of menses constitutes a memorable event, they may report that symptoms occur only during the premenstruum or that symptoms worsen premenstrually. This is one of the rationales for the requirement that symptoms be confirmed by daily prospective ratings. The process of differential diagnosis, particularly if the clinician relies on retrospective symptoms only, is made more difficult because of the overlap between symptoms of premenstrual dysphoric disorder and some other diagnoses. The overlap of symptoms is particularly salient for differentiating premenstrual dysphoric disorder from major depressive episodes, persistent depressive disorder, bipolar disorders, and borderline personality disorder.

Use of hormonal treatments. Some women who present with moderate to severe premenstrual symptoms may be using hormonal treatments, including hormonal contraceptives. If such symptoms occur after initiation of exogenous hormone use, the symptoms may be attributable to the use of hormones rather than to the underlying condition of premenstrual dysphoric disorder. If the woman stops hormones and the symptoms disappear, then this is consistent with substance/medication-induced depressive disorder.

Other medical conditions. Women with chronic medical conditions may experience symptoms of premenstrual dysphoria. As with any depressive disorder, medical conditions that could better account for the symptoms should be ruled out, such as thyroid deficiency and anemia.

Comorbidity

A major depressive episode is the most frequently reported previous disorder in individuals presenting with premenstrual dysphoric disorder. A wide range of medical conditions (e.g., migraine, asthma, allergies, seizure disorders) or other mental disorders (e.g., depressive and bipolar disorders, anxiety disorders, bulimia nervosa, substance use disorders) may worsen in the premenstrual phase; however, the absence of a symptom-free period during the postmenstrual interval obviates a diagnosis of premenstrual dysphoric disorder. These conditions are better considered premenstrual exacerbation of a current mental disorder or medical condition. Although the diagnosis of premenstrual dysphoric disorder should not be assigned in situations in which an individual experiences only a premenstrual exacerbation of another mental or physical disorder, it can be considered in addition to the diagnosis of another mental disorder or medical condition if the individual experiences symptoms and changes in level of functioning that are characteristic of premenstrual dysphoric disorder and markedly different from the symptoms experienced as part of the ongoing disorder.

Substance/Medication-Induced Depressive Disorder

Diagnostic Criteria

A. A prominent and persistent disturbance in mood that predominates in the clinical picture and is characterized by depressed mood or markedly diminished interest or pleasure in all, or almost all, activities.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication.

2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a depressive disorder that is not substance/medication-induced. Such evidence of an independent depressive disorder could include the following:

   The symptoms preceded the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced depressive disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance/medication]-induced depressive disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given. If a mild substance use disorder is comorbid with the substance-induced depressive disorder, the 4th position character is “1,” and the clinician should record “mild [substance] use disorder” before the substance-induced depressive disorder (e.g., “mild cocaine use disorder with cocaine-induced depressive

disorder"). If a moderate or severe substance use disorder is comorbid with the substance-induced depressive disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance-induced depressive disorder.

	ICD-10-CM		
	With mild use disorder	**With moderate or severe use disorder**	**Without use disorder**
Alcohol	F10.14	F10.24	F10.94
Phencyclidine	F16.14	F16.24	F16.94
Other hallucinogen	F16.14	F16.24	F16.94
Inhalant	F18.14	F18.24	F18.94
Opioid	F11.14	F11.24	F11.94
Sedative, hypnotic, or anxiolytic	F13.14	F13.24	F13.94
Amphetamine-type substance (or other stimulant)	F15.14	F15.24	F15.94
Cocaine	F14.14	F14.24	F14.94
Other (or unknown) substance	F19.14	F19.24	F19.94

Specify (see Table 1 in the chapter "Substance-Related and Addictive Disorders," which indicates whether "with onset during intoxication" and/or "with onset during withdrawal" applies to a given substance class; or *specify* "with onset after medication use"):

With onset during intoxication: If criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: If symptoms developed at initiation of medication, with a change in use of medication, or during withdrawal of medication.

Recording Procedures

The name of the substance/medication-induced depressive disorder begins with the specific substance (e.g., cocaine, dexamethasone) that is presumed to be causing the depressive symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., dexamethasone), the code for "other (or unknown) substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the same code should also be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance-induced depressive disorder, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal). For example, in the case of depressive symptoms occurring during withdrawal in a man with a severe cocaine use disorder, the diagnosis is F14.24 severe cocaine use disorder with cocaine-induced depressive disorder, with onset during withdrawal. A separate diagnosis of the comorbid severe cocaine use disorder is not given. If the substance-induced depressive disorder occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F16.94 phencyclidine-induced depressive disorder, with onset during intoxication). When more than one substance is judged to play a significant role in the development of depressive

mood symptoms, each should be listed separately (e.g., F15.24 severe methylphenidate use disorder with methylphenidate-induced depressive disorder, with onset during withdrawal; F19.94 dexamethasone-induced depressive disorder, with onset during intoxication).

Diagnostic Features

The essential feature of substance/medication-induced depressive disorder is a prominent and persistent disturbance in mood that predominates in the clinical picture and is characterized by depressed mood or markedly diminished interest or pleasure in all, or almost all, activities (Criterion A) that is due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication, or a toxin exposure) (Criterion B). In order to meet criteria for the diagnosis, the depressive symptoms must have developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication, as evidenced by clinical history, physical examination, or laboratory findings (Criterion B1), and the involved substance/medication must be capable of producing the depressive symptoms (Criterion B2). In addition, the depressed symptoms are not better explained by a non-substance/medication-induced depressive disorder.

Evidence of an independent depressive disorder includes the observation that the depressive symptoms preceded the onset of substance/medication use, the depressive symptoms persist beyond a substantial period of time after the cessation of acute withdrawal or severe intoxication, or there is other evidence that suggests the existence of an independent non-substance/medication-induced depressive disorder (Criterion C), such as a history of recurrent non-substance-induced depressive episodes. This diagnosis should not be made when symptoms occur exclusively during the course of a delirium (Criterion D). Finally, the diagnosis requires that the substance/medication-induced depressive symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E). The substance-induced depressive disorder diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when

the symptoms in Criterion A predominate in the clinical picture and are sufficiently severe to warrant independent clinical attention.

The two categories of drugs of abuse most likely to cause a substance/medication-induced depressive disorder are depressants (e.g., intoxication with alcohol, benzodiazepines and other sedative, hypnotic, or anxiolytic drugs) and stimulants (e.g., withdrawal from amphetamine-type substances and cocaine). Some medications (e.g., steroids; antihypertensive medications such as clonidine, guanethidine, methyldopoa, and reserpine; interferon; L-dopa) are especially likely to cause substance/medication-induced depressive syndromes. Substances implicated in medication-induced depressive disorder, with varying degrees of evidence, include antibiotics, antiviral agents (efavirenz), cardiovascular agents (beta-blockers and calcium channel blockers, retinoic acid derivatives (isotretinoin), antidepressants, anticonvulsants, antimigraine agents (triptans), antipsychotics, hormonal agents (corticosteroids, oral contraceptives, gonadotropin-releasing hormone agonists, tamoxifen), chemotherapeutic drugs, and smoking cessation agents (varenicline). This list is likely to grow as new compounds are synthesized.

Clear clinical histories and careful judgment are essential in determining whether the substance of abuse or medication is truly associated with induced depressive symptoms or whether the symptoms are better understood as constituting an independent depressive disorder. A diagnosis of a substance/medication-induced depressive disorder is most likely if the individual was taking high doses of a relevant drug of abuse or medication and there is no past history of independent depressive episodes. For example, a depressive episode that developed in the context of heavy use of a relevant substance of abuse or within the first several weeks of beginning alpha-methyldopa (an antihypertensive agent) in an individual with no history of major depressive disorder would qualify for the diagnosis of a substance- or medication-induced depressive disorder. In some cases, a previously established condition (e.g., major depressive disorder, recurrent) can recur while the individual is

coincidentally taking a drug or medication that has the capacity to cause depressive symptoms (e.g., alcohol and/or stimulants in context of heavy use, L-dopa, oral contraceptives). In all of these cases, the clinician must make a judgment as to whether the medication is causative in this particular situation.

A substance/medication-induced depressive disorder is distinguished from an independent depressive disorder by the onset or course, or by other factors associated with the substance or medication use. There must be evidence from the history, physical examination, or laboratory findings of use of a drug of abuse or a medication that is capable of producing depressive symptoms after exposure to, withdrawal from, or intoxication with that substance prior to the onset of the depressive disorder. The neurochemical changes associated with intoxication and withdrawal states for some substances can be relatively protracted, and thus intense depressive symptoms can last for a longer period after the cessation of substance use and still be consistent with a diagnosis of a substance/medication-induced depressive disorder.

Prevalence

The lifetime rate of alcohol- and stimulant-induced depressive episodes has been reported to be 40% or higher among individuals with relevant substance use disorders. However, in a nationally representative U.S. adult population, the lifetime prevalence of substance/medication-induced depressive disorder in the absence of a lifetime history of non-substance-induced depressive disorder was only 0.26%. These data indicate that special care must be taken to search for and address substance-induced conditions in individuals with alcohol and stimulant use disorders.

Development and Course

A depressive disorder associated with the use of substances (e.g., alcohol, amphetamine-type substances and/or cocaine, or a prescribed treatment for medical conditions) must have its onset while the individual is using the substance or during withdrawal, if there is a withdrawal syndrome associated with the substance. Most often, the depressive disorder has its onset within the first few weeks or 1 month of heavy use of the substance.

Once the substance is discontinued, the depressive symptoms usually remit within days to several weeks, depending on the half-life of the substance/medication and the presence of a withdrawal syndrome. If symptoms persist 4 weeks beyond the expected time course of withdrawal of a particular substance/medication, other causes for the depressive mood symptoms should be considered.

There are several prospective controlled trials examining the association of depressive symptoms with use of a prescribed medication, but most reports on this topic involve retrospective series of individuals entering treatment, or participants in large cross-sectional studies. More studies exist regarding the clinical course of alcohol- and illicit drug–induced depressions, and most support the contention that the substance-induced conditions are very likely to fade away within a relatively short time after abstinence. Equally important are indications that individuals with significant residual depressive symptoms following treatment for substance use disorders have a greater likelihood of relapse into their substance use.

Risk and Prognostic Factors

Risk factors for substance-induced depressive disorder include a history of antisocial personality disorder, schizophrenia, and bipolar disorder; a history of stressful life events in the past 12 months; a history of prior drug-induced depressions; and a family history of substance use disorders. In addition, neurochemical changes associated with alcohol and other drugs of abuse often contribute to depressive and anxiety symptoms during withdrawal that subsequently influence ongoing substance use and reduce the likelihood of remission of substance use disorders. The course of substance-induced depressive disorder may be worsened by social-structural adversity associated with poverty, racism, and marginalization.

Sex- and Gender-Related Diagnostic Issues

Among individuals with a substance use disorder, the risk for developing a substance-induced depressive disorder appears to be similar in men and women.

Diagnostic Markers

Laboratory assays of the suspected substance in the blood or urine are of limited value in identifying substance-induced depressive disorder because blood and urine levels are often negative when an individual comes for evaluation, reflecting the fact that substance-induced depressions can last for up to 4 weeks after use of the drug of abuse or medication has ceased. Therefore, a positive test value only means that an individual has had recent experience with a substance but by itself does not establish a time course or other characteristics that are likely to be associated with substance-induced depressive disorder. However, as is true of most mental disorders, the most important data in diagnosing these conditions come from a detailed clinical history and the mental status examination.

Association With Suicidal Thoughts or Behavior

The risk of suicide attempts is higher among individuals with possible alcohol use disorder experiencing depressive episodes, whether substance induced or independent of substances, as compared with control subjects.

Differential Diagnosis

Substance intoxication and withdrawal. Depressive symptoms occur commonly in substance intoxication and substance withdrawal. A diagnosis of substance-induced depressive disorder should be made instead of a diagnosis of substance intoxication or substance withdrawal when the mood symptoms are sufficiently severe to warrant independent clinical attention. For example, dysphoric mood is a characteristic feature of cocaine withdrawal. Substance-induced depressive disorder with onset during withdrawal should be diagnosed instead of cocaine withdrawal only if the mood disturbance in Criterion A predominates in the clinical picture and is sufficiently severe to be a separate focus of attention and treatment.

Independent depressive disorder. A substance/medication-induced depressive disorder is distinguished from an independent depressive disorder by the fact that even though a substance is taken in high enough amounts to be possibly etiologically related to the symptoms, if the depressive syndrome

is observed at times other than when the substance or medication is being used, it should be diagnosed as an independent depressive disorder.

Depressive disorder due to another medical condition. Because individuals with medical conditions often take medications for those conditions, the clinician must consider the possibility that the mood symptoms are caused by the physiological consequences of the medical condition rather than the medication, in which case depressive disorder due to another medical condition is diagnosed. The history often provides the primary basis for such a judgment. At times, a change in the treatment for the medical condition (e.g., medication substitution or discontinuation) may be needed to determine empirically whether the medication is the causative agent. If the clinician has ascertained that the disturbance is a function of both another medical condition and substance use or withdrawal, then both diagnoses (i.e., depressive disorder due to another medical condition and substance/medication-induced depressive disorder) may be given. When there is insufficient evidence to determine whether the depressive symptoms are associated with substance (including a medication) ingestion or withdrawal or with another medical condition or are independent (i.e., not a function of either a substance or another medical condition), a diagnosis of other specified depressive disorder or unspecified depressive disorder is indicated.

Comorbidity

In one study using DSM-IV, comparing individuals with independent major depressive disorder and no comorbid substance use disorder and individuals with substance/medication-induced depressive disorder, those with substance/medication-induced depressive disorder had higher rates of comorbidity with any DSM-IV mental disorder; were more likely to have specific disorders of tobacco use disorder, gambling disorder, and antisocial personality disorder; and were less likely to have persistent depressive disorder. Compared with individuals with major depressive disorder and a comorbid substance use disorder, individuals with substance/medication-induced depressive disorder are more likely to have alcohol or other

substance use disorder; however, they are less likely to have persistent depressive disorder.

Depressive Disorder Due to Another Medical Condition

Diagnostic Criteria

A. A prominent and persistent disturbance in mood that predominates in the clinical picture and is characterized by depressed mood or markedly diminished interest or pleasure in all, or almost all, activities.

B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.

C. The disturbance is not better explained by another mental disorder (e.g., adjustment disorder, with depressed mood, in which the stressor is a serious medical condition).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Coding note: The ICD-10-CM code depends on the specifier (see below).

Specify if:

F06.31 With depressive features: Full criteria are not met for a major depressive episode.

F06.32 With major depressive–like episode: Full criteria are met (except Criterion C) for a major depressive episode.

F06.34 With mixed features: Symptoms of mania or hypomania are also present but do not predominate in the clinical picture.

Coding note: Include the name of the other medical condition in the name of the mental disorder (e.g., F06.31 depressive disorder due to hypothyroidism, with depressive features). The other medical condition should also be coded and listed separately immediately before the depressive disorder due to the medical condition (e.g., E03.9 hypothyroidism; F06.31 depressive disorder due to hypothyroidism, with depressive features).

Diagnostic Features

The essential feature of depressive disorder due to another medical condition is a prominent and persistent period of depressed mood or markedly diminished interest or pleasure in all, or almost all, activities that predominates in the clinical picture (Criterion A) and that is thought to be due to the physiological effects of another medical condition (Criterion B). In determining whether the mood disturbance is due to another medical condition, the clinician must first establish the presence of another medical condition. Further, the clinician must establish that the mood disturbance is etiologically related to another medical condition through a physiological

mechanism. A careful and comprehensive assessment of multiple factors is necessary to make this judgment. Although there are no infallible guidelines for determining whether the relationship between the mood disturbance and another medical condition is etiological, several considerations provide some guidance in this area. One consideration is the presence of a temporal association between the onset, exacerbation, or remission of another medical condition and that of the mood disturbance. A second consideration is the presence of features that are atypical of independent depressive disorders (e.g., atypical age at onset or course or absence of family history). Evidence from the literature that suggests that there can be a direct

association between another medical condition in question and the development of mood symptoms can provide a useful context in the assessment of a particular situation.

Associated Features

Etiology (i.e., a causal relationship to another medical condition based on best clinical evidence) is the key variable in depressive disorder due to another medical condition. The listing of the medical conditions that are said to be able to induce major depression is never complete, and the clinician's best judgment is the essence of this diagnosis.

There are clear associations, as well as some neuroanatomical correlates, of depression with cerebrovascular accident (CVA), Huntington's disease, Parkinson's disease, and traumatic brain injury (TBI). Among the neuroendocrine conditions most closely associated with depression are Cushing's syndrome and hypothyroidism. Autoimmune disorders, such systemic lupus erythematosus, and deficiencies of certain vitamins, such as vitamin B_{12}, have also been linked to depression. There are numerous other conditions thought to be associated with depression, such as multiple sclerosis. However, the literature's support for a causal association is greater with some conditions than with others. Currently, there is support for a direct pathophysiological mechanism for depressive symptoms in focal lesions (CVA, TBI, neoplasm) affecting certain brain regions, Parkinson's disease, Huntington's disease, hypothyroidism, Cushing's syndrome, and pancreatic cancer.

Prevalence

Sex differences in prevalence depend somewhat on the sex difference associated with the medical condition (e.g., systemic lupus erythematosus is more common in women; stroke is somewhat more common in middle-age men compared with women).

Development and Course

Following stroke, the onset of depression appears to be acute, occurring within a few days of the CVA in the largest case series. However, in some

cases, onset of the depression is weeks to months following the CVA. In the largest series, the duration of the major depressive episode following stroke was 9–11 months on average. With Parkinson's disease and Huntington's disease, depression often precedes the major motor impairments and cognitive impairments associated with each condition. This is more prominently the case for Huntington's disease, in which depression is considered to be the first neuropsychiatric symptom. There is some observational evidence that depression is less common as the neurocognitive disorder due to Huntington's disease progresses. In some individuals with static brain injuries and other central nervous system diseases, mood symptoms may be episodic (i.e., recurring) over the course of the disorder. In Cushing's syndrome and hypothyroidism, depression can be an early manifestation of the disease. In pancreatic cancer, depression often precedes other features.

Risk and Prognostic Factors

The risk of acute onset of a major depressive disorder following a CVA (within 1 day to a week of the event) appears to be strongly correlated with lesion location, with greatest risk associated with left frontal strokes and least risk apparently associated with right frontal lesions

in those individuals who present within days of the stroke. The association with frontal regions and laterality is not observed in depressive states that occur in the 2–6 months following stroke, perhaps indicative of later depressive symptoms representing major depressive disorder, adjustment disorder, or demoralization. In individuals with Parkinson's disease, early age at onset, greater burden of motor symptoms, and longer duration of the disease have been associated with depression. Risk of depression after TBI has been associated with female gender, prior depressive disorder, early psychiatric symptoms following injury, lower brain volume, and unemployment.

Sex- and Gender-Related Diagnostic Issues

Women may be at differentially higher risk for developing depression in the setting of cardiovascular disease, particularly poststroke.

Diagnostic Markers

Diagnostic markers pertain to those associated with the medical condition (e.g., steroid levels in blood or urine to help corroborate the diagnosis of Cushing's disease, which can be associated with manic or depressive syndromes).

Association With Suicidal Thoughts or Behavior

There are no epidemiological studies that provide evidence to differentiate the risk of suicide from a major depressive episode due to another medical condition compared with the risk from a major depressive episode in general. There are case reports of suicides in association with major depressive episodes associated with another medical condition. There is a clear association between serious medical illnesses and suicide, particularly shortly after onset or diagnosis of the illness. Thus, it would be prudent to assume that the risk of suicide for major depressive episodes associated with medical conditions is not less than that for other forms of major depressive episode, and might even be greater.

Differential Diagnosis

Depressive disorders not due to another medical condition. Determination of whether a medical condition accompanying a depressive disorder is causing the disorder depends on a) the absence of an episode(s) of depressive episodes prior to the onset of the medical condition, b) the probability that the associated medical condition has a potential to promote or cause a depressive disorder, and c) a course of the depressive symptoms shortly after the onset or worsening of the medical condition, especially if the depressive symptoms remit near the time that the medical disorder is effectively treated or remits.

Medication-induced depressive disorder. An important caveat is that some medical conditions are treated with medications (e.g., steroids or alpha-interferon) that can induce depressive or manic symptoms. In these cases,

clinical judgment, based on all the evidence in hand, is the best way to try to separate the most likely and/or the most important of two etiological factors (i.e., association with the medical condition vs. a substance-induced syndrome).

Delirium and major or mild neurocognitive disorder. A separate diagnosis of depressive disorder due to another medical condition is not given if the depressive disturbance occurs exclusively during the course of a delirium. However, a diagnosis of depressive disorder due to another medical condition may be given in addition to a diagnosis of major or mild neurocognitive disorder if the depressive disturbance is judged to be a physiological consequence of the pathological process causing the neurocognitive disorder and if symptoms of depression are a prominent part of the clinical presentation.

Adjustment disorders. It is important to differentiate a depressive episode from an adjustment disorder, as the onset of the medical condition is in itself a life stressor that could

bring on either an adjustment disorder or an episode of major depression. The major differentiating elements are the pervasiveness of the depressive picture and the number and quality of the depressive symptoms that the individual reports or demonstrates on the mental status examination. The differential diagnosis of the associated medical conditions is relevant but largely beyond the scope of the present manual.

Demoralization. Demoralization is a common reaction to chronic medical illness. It is marked by a sense of subjective incompetence, helplessness, and hopelessness, and a desire to give up. It is often accompanied by depressive symptoms such as low mood and fatigue. Demoralization typically lacks the anhedonia associated with depressive disorder due to another medical condition, and individuals will generally find pleasure in previously meaningful activities and be able to experience moments of happiness.

Comorbidity

Conditions comorbid with depressive disorder due to another medical condition are those associated with the medical conditions of etiological relevance. It has been noted that delirium can occur before or along with depressive symptoms in individuals with a variety of medical conditions, such as Cushing's disease. The association of anxiety symptoms, usually generalized symptoms, is common in depressive disorders, regardless of cause.

Other Specified Depressive Disorder

F32.89

This category applies to presentations in which symptoms characteristic of a depressive disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the depressive disorders diagnostic class and do not meet criteria for adjustment disorder with depressed mood or adjustment disorder with mixed anxiety and depressed mood. The other specified depressive disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific depressive disorder. This is done by recording "other specified depressive disorder" followed by the specific reason (e.g., "short-duration depressive episode").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Recurrent brief depression:** Concurrent presence of depressed mood and at least four other symptoms of depression for 2–13 days at least once per month (not associated with the menstrual cycle) for at least 12 consecutive months in an individual whose presentation has never met criteria for any other depressive or bipolar disorder

and does not currently meet active or residual criteria for any psychotic disorder.

2. **Short-duration depressive episode (4–13 days):** Depressed affect and at least four of the other eight symptoms of a major depressive episode associated with clinically significant distress or impairment that persists for more than 4 days, but less than 14 days, in an individual whose presentation has never met criteria for any other depressive or bipolar disorder, does not currently meet active or residual criteria for any psychotic disorder, and does not meet criteria for recurrent brief depression.
3. **Depressive episode with insufficient symptoms:** Depressed affect and at least one of the other eight symptoms of a major depressive episode associated with clinically significant distress or impairment that persist for at least 2 weeks in an individual whose presentation has never met criteria for any other depressive or bipolar disorder, does not currently meet active or residual criteria for any psychotic disorder, and does not meet criteria for mixed anxiety and depressive disorder symptoms.

4. **Major depressive episode superimposed** on schizophrenia, schizophreniform disorder, delusional disorder, or other specified and unspecified schizophrenia spectrum and other psychotic disorder. **Note:** Major depressive episodes that are part of schizoaffective disorder do not merit an additional diagnosis of other specified depressive disorder.

Unspecified Depressive Disorder

F32.A

This category applies to presentations in which symptoms characteristic of a depressive disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the depressive disorders diagnostic class and do not meet criteria for adjustment disorder with depressed mood or adjustment disorder with mixed anxiety and depressed mood. The unspecified depressive disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific depressive disorder, and includes presentations for which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Unspecified Mood Disorder

F39

This category applies to presentations in which symptoms characteristic of a mood disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not at the time of the evaluation meet the full criteria for any of the disorders in either the bipolar or the depressive disorders diagnostic classes and in which it is difficult to choose between unspecified bipolar and related disorder and unspecified depressive disorder (e.g., acute agitation).

Specifiers for Depressive Disorders

Specify if:

With anxious distress: Anxious distress is defined as the presence of at least two of the following symptoms during the majority of days of the current major depressive episode (or the most recent major depressive episode if major depressive disorder is currently in partial or full remission) or current persistent depressive disorder:

1. Feeling keyed up or tense.
2. Feeling unusually restless.
3. Difficulty concentrating because of worry.
4. Fear that something awful may happen.
5. Feeling that the individual might lose control of himself or herself.

Specify current severity:

Mild: Two symptoms.

Moderate: Three symptoms.

Moderate-severe: Four or five symptoms.

Severe: Four or five symptoms and with motor agitation.

Note: Anxious distress has been noted as a prominent feature of both bipolar and major depressive disorder in both primary care and specialty mental health

settings. High levels of anxiety have been associated with higher suicide risk, longer duration of illness, and greater likelihood of treatment nonresponse. As a result, it is clinically useful to specify accurately the presence and severity levels of anxious distress for treatment planning and monitoring of response to treatment.

With mixed features:

A. At least three of the following manic/hypomanic symptoms are present during the majority of days of the current major depressive episode (or the most recent major depressive

episode if major depressive disorder is currently in partial or full remission):

1. Elevated, expansive mood.
2. Inflated self-esteem or grandiosity.
3. More talkative than usual or pressure to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Increase in energy or goal-directed activity (either socially, at work or school, or sexually).
6. Increased or excessive involvement in activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments).
7. Decreased need for sleep (feeling rested despite sleeping less than usual; to be contrasted with insomnia).

B. Mixed symptoms are observable by others and represent a change from the person's usual behavior.

C. For individuals whose symptoms meet full criteria for either mania or hypomania, the diagnosis should be bipolar I or bipolar II disorder.

D. The mixed symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication or other treatment).

Note: Mixed features associated with a major depressive episode have been found to be a significant risk factor for the development of bipolar I or bipolar II disorder. As a result, it is clinically useful to note the presence of this specifier for treatment planning and monitoring of response to treatment.

With melancholic features:

A. One of the following is present during the most severe period of the current major depressive episode (or the most

recent major depressive episode if major depressive disorder is currently in partial or full remission):

1. Loss of pleasure in all, or almost all, activities.
2. Lack of reactivity to usually pleasurable stimuli (does not feel much better, even temporarily, when something good happens).

B. Three (or more) of the following:

1. A distinct quality of depressed mood characterized by profound despondency, despair, and/or moroseness or by so-called empty mood.
2. Depression that is regularly worse in the morning.
3. Early-morning awakening (i.e., at least 2 hours before usual awakening).
4. Marked psychomotor agitation or retardation.
5. Significant anorexia or weight loss.
6. Excessive or inappropriate guilt.

Note: The specifier “with melancholic features” is applied if these features are present at the most severe stage of the episode. There is a near-complete absence of the

capacity for pleasure, not merely a diminution. A guideline for evaluating the lack of reactivity of mood is that even highly desired events are not associated with marked brightening of mood. Either mood does not brighten at all, or it brightens only partially (e.g., up to 20%–40% of normal for only minutes at a time). The “distinct quality” of mood that is characteristic of the “with melancholic features” specifier is experienced as qualitatively different from that during a nonmelancholic depressive episode. A depressed mood that is described as merely more severe, longer lasting, or present without a reason is not considered distinct in quality. Psychomotor changes are nearly always present and are observable by

others.

Melancholic features exhibit only a modest tendency to repeat across episodes in the same individual. They are more frequent in inpatients, as opposed to outpatients; are less likely to occur in milder than in more severe major depressive episodes; and are more likely to occur in individuals with psychotic features.

With atypical features: This specifier is applied when these features predominate during the majority of days of the current major depressive episode (or the most recent major depressive episode if major depressive disorder is currently in partial or full remission) or current persistent depressive disorder.

A. Mood reactivity (i.e., mood brightens in response to actual or potential positive events).
B. Two (or more) of the following:
    1. Significant weight gain or increase in appetite.
    2. Hypersomnia.
    3. Leaden paralysis (i.e., heavy, leaden feelings in arms or legs).
    4. A long-standing pattern of interpersonal rejection sensitivity (not limited to episodes of mood disturbance) that results in significant social or occupational impairment.
C. Criteria are not met for “with melancholic features” or “with catatonia” during the same episode.

Note: “Atypical depression” has historical significance (i.e., atypical in contradistinction to the more classical agitated, “endogenous” presentations of depression that were the norm when depression was rarely diagnosed in outpatients and almost never in adolescents or younger adults) and today does not connote an uncommon or unusual clinical presentation as the term might imply.

Mood reactivity is the capacity to be cheered up when presented with positive events (e.g., a visit from children, compliments from others). Mood may become euthymic (not sad) even for extended periods of time if the external circumstances remain favorable. Increased appetite may be manifested by an obvious increase in food intake or by weight gain. Hypersomnia may include either an extended period of nighttime sleep or daytime napping that totals at least 10 hours of sleep per day (or at least 2 hours more than when not depressed). Leaden paralysis is defined as feeling heavy, leaden, or weighted down, usually in the arms or legs. This sensation is generally present for at least an hour a day but often lasts for many hours at a time. Unlike the other atypical features, pathological sensitivity to perceived interpersonal rejection is a trait that has an early onset and persists throughout most of adult life. Rejection sensitivity occurs when the person is and is not depressed, though it may be exacerbated during depressive periods.

With psychotic features: Delusions and/or hallucinations are present at any time in the current major depressive episode (or the most recent major depressive episode if major depressive disorder is currently in partial or full remission). If psychotic features are present, *specify* if mood-congruent or mood-incongruent:

With mood-congruent psychotic features: The content of all delusions and hallucinations is consistent with the typical depressive themes of personal inadequacy, guilt, disease, death, nihilism, or deserved punishment.

With mood-incongruent psychotic features: The content of the delusions or hallucinations does not involve typical depressive themes of personal inadequacy, guilt, disease,

death, nihilism, or deserved punishment, or the content is a mixture of mood-incongruent and mood-congruent themes.

With catatonia: This specifier is applied to the current major depressive episode (or the most recent major depressive episode if major depressive disorder is currently in partial or full remission) if catatonic features are present during most of the episode. See criteria for catatonia associated with a mental disorder in the chapter “Schizophrenia Spectrum and Other Psychotic Disorders.”

With peripartum onset: This specifier is applied to the current major depressive episode (or the most recent major depressive episode if major depressive disorder is currently in partial or full remission) if onset of mood symptoms occurs during pregnancy or in the 4 weeks following delivery.

Note: Mood episodes can have their onset either during pregnancy or postpartum. About 50% of postpartum major depressive episodes begin prior to delivery. Thus, these episodes are referred to collectively as *peripartum* episodes.

Between conception and birth, about 9% of women will experience a major depressive episode. The best estimate for prevalence of a major depressive episode between birth and 12 months postpartum is just below 7%.

Peripartum-onset mood episodes can present either with or without psychotic features. Infanticide (a rare occurrence) is most often associated with postpartum psychotic episodes that are characterized by command hallucinations to kill the infant or delusions that the infant is possessed, but psychotic symptoms can also occur in severe postpartum mood episodes without such specific delusions or hallucinations.

Postpartum mood (major depressive or manic) episodes with psychotic features appear to occur in from 1 in 500 to 1 in 1,000 deliveries and may be more common in

primiparous women. The risk of postpartum episodes with psychotic features is particularly increased for women with prior postpartum psychotic mood episodes but is also elevated for those with a prior history of a depressive or bipolar disorder (especially bipolar I disorder) and those with a family history of bipolar disorders.

Once a woman has had a postpartum episode with psychotic features, the risk of recurrence with each subsequent delivery is between 30% and 50%. Postpartum episodes must be differentiated from delirium occurring in the postpartum period, which is distinguished by a fluctuating level of awareness or attention.

Peripartum-onset depressive disorders must be distinguished from the much more common “maternity blues,” or what is known in lay terms as “baby blues.” Maternity blues is not considered to be a mental disorder and is characterized by sudden changes in mood (e.g., the sudden onset of tearfulness in the absence of depression) that do not cause functional impairment and that are likely caused by physiological changes occurring after delivery. It is temporary and self-limited, typically improving quickly (within a week) without the need for treatment. Other symptoms of maternity blues include sleep disturbance and even confusion that can occur shortly after delivery.

Perinatal women may be at higher risk for depressive disorders due to thyroid abnormalities as well as other medical conditions that can cause depressive symptoms. If the depressive symptoms are judged to be due to another medical condition related to the perinatal period, depressive disorder due to another medical condition should be diagnosed instead of a major depressive episode, with peripartum onset.

With seasonal pattern: This specifier applies to recurrent major depressive disorder.

A. There has been a regular temporal relationship between the onset of major depressive episodes in major depressive disorder and a particular time of the year (e.g., in the fall or winter).

   Note: Do not include cases in which there is an obvious effect of seasonally related psychosocial stressors (e.g., regularly being unemployed every winter).

B. Full remissions also occur at a characteristic time of the year (e.g., depression disappears in the spring).

C. In the last 2 years, two major depressive episodes have occurred that demonstrate the temporal seasonal relationships defined above and no nonseasonal major depressive episodes have occurred during that same period.

D. Seasonal major depressive episodes (as described above) substantially outnumber the nonseasonal major depressive episodes that may have occurred over the individual's lifetime.

Note: The specifier "with seasonal pattern" can apply to the pattern of major depressive episodes in major depressive disorder, recurrent. The essential feature is the onset and remission of major depressive episodes at characteristic times of the year. In most cases, the episodes begin in fall or winter and remit in spring. Less commonly, there may be recurrent summer depressive episodes. This pattern of onset and remission of episodes must have occurred during at least a 2-year period, without any nonseasonal episodes occurring during this period. In addition, the seasonal depressive episodes must substantially outnumber any nonseasonal depressive episodes over the individual's lifetime.

This specifier does not apply to those situations in which the pattern is better explained by seasonally linked

psychosocial stressors (e.g., seasonal unemployment or school schedule). Major depressive episodes that occur in a seasonal pattern are often characterized by loss of energy, hypersomnia, overeating, weight gain, and a craving for carbohydrates.

The prevalence of winter-type seasonal pattern appears to vary with latitude, age, and sex. Prevalence increases with higher latitudes. Age is also a strong predictor of seasonality, with younger persons at higher risk for winter depressive episodes.

Specify if:

In partial remission: Symptoms of the immediately previous major depressive episode are present but full criteria are not met, or there is a period lasting less than 2 months without any significant symptoms of a major depressive episode following the end of such an episode.

In full remission: During the past 2 months, no significant signs or symptoms of the disturbance were present.

Specify current severity:

Severity is based on the number of criterion symptoms, the severity of those symptoms, and the degree of functional disability.

Mild: Few, if any, symptoms in excess of those required to make the diagnosis are present, the intensity of the symptoms is distressing but manageable, and the symptoms result in minor impairment in social or occupational functioning.

Moderate: The number of symptoms, intensity of symptoms, and/or functional impairment are between those specified for “mild” and “severe.”

Severe: The number of symptoms is substantially in excess of that required to make the diagnosis, the intensity of the symptoms is seriously distressing and unmanageable, and

the symptoms markedly interfere with social and occupational functioning.

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[1]In distinguishing grief from a major depressive episode (MDE), it is useful to consider that in grief the predominant affect is feelings of emptiness and loss, while in an MDE it is persistent depressed mood and the inability to anticipate happiness or pleasure. The dysphoria in grief is likely to decrease in intensity over days to weeks and occurs in waves, the so-called pangs of grief. These waves tend to be associated with thoughts or reminders of the deceased. The depressed mood of an MDE is more persistent and not tied to specific thoughts or preoccupations. The pain of grief may be accompanied by positive emotions and humor that are uncharacteristic of the pervasive unhappiness and misery characteristic of an MDE. The thought content associated with grief generally features a preoccupation with thoughts and memories of the deceased, rather than the self-critical or pessimistic ruminations seen in an MDE. In grief, self-esteem is generally preserved, whereas in an MDE feelings of worthlessness and self-loathing are common. If self-derogatory ideation is present in grief, it typically involves perceived failings vis-à-vis the deceased (e.g., not visiting frequently enough, not telling the deceased how much he or she was loved). If a bereaved individual thinks about death and dying, such thoughts are generally focused on the deceased and possibly about "joining" the deceased, whereas in an MDE such thoughts are focused on ending one's own life because of feeling worthless, undeserving of life, or unable to cope with the pain of depression.

Anxiety Disorders

Anxiety disorders include disorders that share features of excessive fear and anxiety and related behavioral disturbances. *Fear* is the emotional response to real or perceived imminent threat, whereas *anxiety* is anticipation of future threat. Obviously, these two states overlap, but they also differ, with fear more often associated with surges of autonomic arousal necessary for fight or flight, thoughts of immediate danger, and escape behaviors, and anxiety more often associated with muscle tension and vigilance in preparation for future danger and cautious or avoidant behaviors. Sometimes the level of fear or anxiety is reduced by pervasive avoidance behaviors. *Panic attacks* feature prominently within the anxiety disorders as a particular type of fear response. Panic attacks are not limited to anxiety disorders but rather can be seen in other mental disorders as well.

The anxiety disorders differ from one another in the types of objects or situations that induce fear, anxiety, or avoidance behavior, and the associated cognition. Thus, while the anxiety disorders tend to be highly comorbid with each other, they can be differentiated by close examination of the types of situations that are feared or avoided and the content of the associated thoughts or beliefs.

Anxiety disorders differ from developmentally normative fear or anxiety by being excessive or persisting beyond developmentally appropriate periods. They differ from transient fear or anxiety, often stress-induced, by being persistent (e.g., typically lasting 6 months or more), although the criterion for duration is intended as a general guide with allowance for some degree of flexibility and is sometimes of shorter duration in children (as in separation anxiety disorder and selective mutism). Since individuals with anxiety disorders typically overestimate the danger in situations they fear or avoid, the primary determination of whether the fear or anxiety is excessive or out of proportion is made by the

clinician, taking cultural contextual factors into account. Many of the anxiety disorders develop in childhood and tend to persist if not treated. Most occur more frequently in girls than in boys (approximately 2:1 ratio). Each anxiety disorder is diagnosed only when the symptoms are not attributable to the physiological effects of a substance/medication or to another medical condition or are not better explained by another mental disorder.

The chapter is arranged developmentally, with disorders sequenced according to the typical age at onset. The individual with separation anxiety disorder is fearful or anxious about separation from attachment figures to a degree that is developmentally inappropriate. There is persistent fear or anxiety about harm coming to attachment figures and events that could lead to loss of or separation from attachment figures and reluctance to go away from attachment figures, as well as nightmares and physical symptoms of distress. Although the symptoms often develop in childhood, they can be expressed throughout adulthood as well in the absence of a history of childhood separation anxiety disorder.

Selective mutism is characterized by a consistent failure to speak in social situations in which there is an expectation to speak (e.g., school) even though the individual speaks in other situations. The failure to speak has significant consequences on achievement in academic or occupational settings or otherwise interferes with normal social communication.

Individuals with specific phobia are fearful or anxious about or avoidant of circumscribed objects or situations. A specific cognition is not featured in this disorder, as it is in other anxiety disorders. The fear, anxiety, or avoidance is almost always immediately induced by the phobic situation, to a degree that is persistent and out of proportion to the actual risk posed. There are various types of specific phobias: animal; natural environment; blood-injection-injury; situational; and other situations.

In social anxiety disorder, the individual is fearful or anxious about or avoidant of social interactions and situations that involve the possibility of being scrutinized. These include social interactions such as meeting unfamiliar people, situations in which the individual may be observed

eating or drinking, and situations in which the individual performs in front of others. The cognition is of being negatively evaluated by others, by being embarrassed, humiliated, or rejected, or offending others.

In panic disorder, the individual experiences recurrent unexpected panic attacks and is persistently concerned or worried about having more panic attacks or changes his or her behavior in maladaptive ways because of the panic attacks (e.g., avoidance of exercise or of unfamiliar locations). Panic attacks are abrupt surges of intense fear or intense discomfort that reach a peak within minutes, accompanied by physical and/or cognitive symptoms. Limited-symptom panic attacks include fewer than four symptoms. Panic attacks may be *expected*, such as in response to a typically feared object or situation, or *unexpected*, meaning that the panic attack occurs for no apparent reason. Panic attacks function as a marker and prognostic factor for severity of diagnosis, course, and comorbidity across an array of disorders, including, but not limited to, anxiety, substance use, depressive, and psychotic disorders. The specifier “with panic attacks” may therefore be used for panic attacks that occur in the context of any anxiety disorder, as well as other mental disorders (e.g., depressive disorders, posttraumatic stress disorder).

Individuals with agoraphobia are fearful and anxious in many different situations, and the diagnostic criteria require symptoms in two or more of the following: using public transportation, being in open spaces, being in enclosed places, standing in line or being in a crowd, or being outside of the home alone in other situations. The individual fears these situations because of thoughts that escape might be difficult or help might not be available in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms. These situations consistently induce fear or anxiety and are often avoided or require the presence of a companion.

The key features of generalized anxiety disorder are persistent and excessive anxiety and worry about various domains, including work and school performance, that the individual finds difficult to control. In addition, the individual experiences physical symptoms, including restlessness or feeling keyed up or on edge; being easily fatigued; difficulty concentrating or mind going blank; irritability; muscle tension; and sleep disturbance.

Substance/medication-induced anxiety disorder involves anxiety due to substance intoxication or withdrawal or to a medication treatment. In anxiety disorder due to another medical condition, anxiety symptoms are the physiological consequence of another medical condition.

Disorder-specific scales are available to better characterize the severity of each anxiety disorder and to capture change in severity over time. For ease of use, particularly for individuals with more than one anxiety disorder, these scales have been developed to have the same format (but different focus) across the anxiety disorders, with ratings of behavioral symptoms, cognitive symptoms, and physical symptoms relevant to each disorder.

Individuals with anxiety may be more likely to have suicidal thoughts, attempt suicide, and die by suicide than those without anxiety. Panic disorder, generalized anxiety disorder, and specific phobia have been identified as the anxiety disorders most strongly associated with a transition from suicidal thoughts to suicide attempt.

Separation Anxiety Disorder

Diagnostic Criteria F93.0

A. Developmentally inappropriate and excessive fear or anxiety concerning separation from those to whom the individual is attached, as evidenced by at least three of the following:

   1. Recurrent excessive distress when anticipating or experiencing separation from home or from major attachment figures.
   2. Persistent and excessive worry about losing major attachment figures or about possible harm to them, such as illness, injury, disasters, or death.
   3. Persistent and excessive worry about experiencing an untoward event (e.g., getting lost, being kidnapped, having

an accident, becoming ill) that causes separation from a major attachment figure.

4. Persistent reluctance or refusal to go out, away from home, to school, to work, or elsewhere because of fear of separation.
5. Persistent and excessive fear of or reluctance about being alone or without major attachment figures at home or in other settings.
6. Persistent reluctance or refusal to sleep away from home or to go to sleep without being near a major attachment figure.
7. Repeated nightmares involving the theme of separation.
8. Repeated complaints of physical symptoms (e.g., headaches, stomachaches, nausea, vomiting) when separation from major attachment figures occurs or is anticipated.

B. The fear, anxiety, or avoidance is persistent, lasting at least 4 weeks in children and adolescents and typically 6 months or more in adults.

C. The disturbance causes clinically significant distress or impairment in social, academic, occupational, or other important areas of functioning.

D. The disturbance is not better explained by another mental disorder, such as refusing to leave home because of excessive resistance to change in autism spectrum disorder; delusions or hallucinations concerning separation in psychotic disorders; refusal to go outside without a trusted companion in agoraphobia; worries about ill health or other harm befalling significant others in generalized anxiety disorder; or concerns about having an illness in illness anxiety disorder.

Diagnostic Features

The essential feature of separation anxiety disorder is excessive fear or anxiety concerning separation from home or attachment figures. The anxiety exceeds what may be expected given the individual's developmental level (Criterion A). Individuals with separation anxiety disorder have symptoms that meet at least three of the following criteria: They experience recurrent excessive distress when separation from home or major attachment figures is anticipated or occurs (Criterion A1). They worry about the well-being or death of attachment figures, particularly when separated from them, and they need to know the whereabouts of their attachment figures and want to stay in touch with them (Criterion A2). They also worry about untoward events to themselves, such as getting lost, being kidnapped, or having an accident, that would keep them from ever being reunited with their major attachment figure (Criterion A3). Individuals with separation anxiety disorder are reluctant or refuse to go out by themselves because of separation fears (Criterion A4). They have

persistent and excessive fear or reluctance about being alone or without major attachment figures at home or in other settings. Children with separation anxiety disorder may be unable to stay or go in a room by themselves and may display "clinging" behavior, staying close to or "shadowing" the parent around the house, or requiring someone to be with them when going to another room in the house (Criterion A5). They have persistent reluctance or refusal to go to sleep without being near a major attachment figure or to sleep away from home (Criterion A6). Children with this disorder often have difficulty at bedtime and may insist that someone stay with them until they fall asleep. During the night, they may make their way to their parents' bed (or that of a significant other, such as a sibling). Children may be reluctant or refuse to attend camp, to sleep at friends' homes, or to go on errands. Adults may be uncomfortable when traveling independently (e.g., sleeping in a hotel room away from home or attachment figures). There may be repeated nightmares in which the content expresses the individual's separation anxiety (e.g., destruction of the family through fire, murder, or other catastrophe) (Criterion A7). Physical symptoms (e.g., headaches, abdominal complaints, nausea, vomiting) are

common in children when separation from major attachment figures occurs or is anticipated (Criterion A8). Cardiovascular symptoms such as palpitations, dizziness, and feeling faint are rare in younger children but may occur in adolescents and adults.

The disturbance must last for a period of at least 4 weeks in children and adolescents younger than 18 years and is typically 6 months or longer in adults (Criterion B). However, the duration criterion for adults should be used as a general guide, with allowance for some degree of flexibility. The disturbance must cause clinically significant distress or impairment in social, academic, occupational, or other important areas of functioning (Criterion C).

Associated Features

When separated from major attachment figures, children and adults with separation anxiety disorder may exhibit social withdrawal, apathy, sadness, or difficulty concentrating on work or play. Depending on their age, individuals may have fears of animals, monsters, the dark, muggers, burglars, kidnappers, car accidents, plane travel, and other situations that are perceived as presenting danger to the family or themselves. Some individuals become homesick and extremely uncomfortable when away from home. Separation anxiety disorder in children may lead to school refusal, which in turn may lead to academic difficulties and social isolation. When extremely upset at the prospect of separation, children may show anger or occasionally aggression toward someone who is forcing separation. When alone, especially in the evening or the dark, young children may report unusual perceptual experiences (e.g., seeing people peering into their room, frightening creatures reaching for them, feeling eyes staring at them). Children with this disorder may be described as demanding, intrusive, and in need of constant attention, and, as adults, may appear dependent and overprotective as parents. Adults with the disorder are likely to text or phone their major attachment figures throughout the day and repeatedly check on their whereabouts. The individual’s excessive demands often become a source of frustration for family members, leading to resentment and conflict within the family.

Prevalence

The 6- to 12-month prevalence of separation anxiety disorder in children is estimated to be approximately 4%. In a community sample of toddlers, separation anxiety disorder appears to be equally represented among girls and boys; however, school-age girls appear to have higher prevalence rates than school-age boys. In adolescents in the United States, the 12-month prevalence is 1.6%. Separation anxiety disorder decreases in prevalence from childhood through adolescence and adulthood. In clinical samples of children, the disorder is equally common in boys and girls in contrast to community samples, where the

disorder is more frequent in girls. Reports from children tend to yield higher rates of separation anxiety disorder than parent reports of the child's symptoms.

For adults, the 12-month prevalence of separation anxiety disorder in the United States ranges from 0.9% to 1.9%. Among adults with separation anxiety disorder, women tend to have higher prevalence rates of the disorder in both clinical and community studies. Across 18 countries, the mean 12-month prevalence in adults is 1.0%, with a range of < 0.1% to 2.7% (e.g., 0.3% in Romania, 2.7% in Colombia). A higher prevalence was observed in women compared with men in this total sample (1.3% compared with 0.8%).

Development and Course

Periods of heightened separation anxiety from attachment figures are part of normal early development and may indicate the development of secure attachment relationships (e.g., around age 1 year, when infants may experience stranger anxiety). Onset of separation anxiety disorder may be as early as preschool age and may occur at any time during childhood and in adolescence. Across 18 countries, median age at onset reported by adults (age 18 years and older) with the disorder is in late adolescence in high- and upper-middle-income countries and in the mid-20's in low- and lower-middle-income countries. Most adults report a fluctuating course of the

disorder over their lifetime, and they may report some symptoms in childhood.

Typically there are periods of exacerbation and remission. In some cases, both the anxiety about possible separation and the avoidance of situations involving separation from the home or nuclear family (e.g., going away to college, moving away from attachment figures) may persist through adulthood. However, the majority of children with separation anxiety disorder are free of impairing anxiety disorders over their lifetimes.

The manifestations of separation anxiety disorder vary with age. Younger children are more reluctant to go to school or may avoid school altogether. Younger children may not express worries or specific fears of definite threats to parents, home, or themselves, and the anxiety is manifested only when separation is experienced. As children age, worries emerge; these are often worries about specific dangers (e.g., accidents, kidnapping, mugging, death) or vague concerns about not being reunited with attachment figures. In adults, separation anxiety disorder may limit their ability to cope with changes in circumstances (e.g., moving, getting married). Adults with the disorder are typically overconcerned about their offspring, spouses, parents, and pets and experience marked discomfort when separated from them. They may also experience significant disruption in work or social experiences because of needing to continuously check on the whereabouts of a significant other.

Risk and Prognostic Factors

Environmental. Separation anxiety disorder often develops after life stress, especially a loss (e.g., the death of a relative or pet; an illness of the individual or a relative; a change of schools; parental divorce; a move to a new neighborhood; immigration; a disaster that involved periods of separation from attachment figures). Being bullied during childhood has been shown to be a risk factor for the development of separation anxiety disorder. In young adults, other examples of life stress include leaving the parental home, entering into a romantic relationship, and becoming a parent. A history of parental overprotection and intrusiveness may be associated with separation anxiety disorder in both childhood and adulthood.

Genetic and physiological. There is evidence that separation anxiety disorder may be heritable. Heritability was estimated at 73% in a community sample of 6-year-old twins, with higher rates found in girls. Children with separation anxiety disorder display particularly enhanced sensitivity to respiratory stimulation using CO_2-enriched air. Separation anxiety disorder also appears to aggregate in families.

Culture-Related Diagnostic Issues

There are cultural variations in the degree to which it is considered desirable to tolerate separation, so that demands and opportunities for separation between parents and children are avoided in some cultural contexts. For example, there is wide variation across countries and cultural contexts with respect to the age at which it is expected that offspring should leave the parental home. Youth vary in their self-reports of separation anxiety symptoms; for instance, Taiwanese youth endorse higher symptoms of separation anxiety compared with U.S. youth. It is important to differentiate separation anxiety disorder from the high value some cultural communities place on strong interdependence among family members.

Association With Suicidal Thoughts or Behavior

Separation anxiety disorder in children and adolescents may be associated with an increased risk for suicide, although this association is not specific to separation anxiety disorder and is found in other anxiety disorders where there is significant comorbidity. A large twin study showed that being bullied during childhood was a risk factor for suicidal thoughts during young adulthood.

Functional Consequences of Separation Anxiety Disorder

Individuals with separation anxiety disorder often limit independent activities away from home or attachment figures (e.g., in children, avoiding school, not going to camp, having difficulty sleeping alone; in adolescents,

not going away to college; in adults, not leaving the parental home, not traveling long distances without their close attachments, not working outside the home). Symptoms in adults are often debilitating and affect multiple areas of their lives. For example, adults with separation anxiety disorder may deliberately reorganize their work schedules and other activities because of their anxieties about possible separations from close attachment figures; they may often express frustration with the limitations on their lives because of their need to maintain proximity to, or at least virtual contact with, their key attachment figures (e.g., by texting or phoning them repeatedly throughout the day). Separation anxiety disorder is associated with greater reported impairment in individuals from high- and upper-middle-income countries compared with those from low- and lower-middle-income countries.

Differential Diagnosis

Generalized anxiety disorder. Separation anxiety disorder is distinguished from generalized anxiety disorder in that the anxiety in separation anxiety disorder predominantly concerns real or imagined separation from attachment figures. Furthermore, if other worries occur, they are not excessive.

Panic disorder. In separation anxiety disorder, threats of separation from close attachments may lead to extreme anxiety and panic attacks. In contrast to panic disorder, where panic attacks occur unexpectedly and are usually accompanied by fears of dying or going "crazy," the panic attacks in separation anxiety disorder occur in anticipation of real or imagined separations from attachment figures or places of safety and security, or from worries that untoward events will befall the individual's close attachments.

Agoraphobia. Unlike individuals with agoraphobia, those with separation anxiety disorder are not anxious about being trapped or incapacitated in situations from which escape is perceived as difficult in the event of panic-like symptoms or other incapacitating symptoms. Instead, they fear being away from places of safety associated with their major attachment figures.

Conduct disorder. School avoidance (truancy) is common in conduct disorder, but anxiety about separation is not responsible for school absences, and the child or adolescent usually stays away from, rather than returns to, the home.

Social anxiety disorder. School refusal may be attributable to social anxiety disorder. In such instances, the school avoidance is due to fear of being judged negatively by others rather than due to worries about being separated from attachment figures.

Posttraumatic stress disorder. Fear of separation from loved ones is common after a traumatic event such as a major disaster, particularly when periods of separation from loved ones are experienced during the traumatic event. In posttraumatic stress disorder (PTSD), the central symptoms concern intrusions about, and avoidance of, memories associated with the traumatic event itself, whereas in separation anxiety disorder, the worries and avoidance concern the well-being of attachment figures and separation from them.

Illness anxiety disorder. Separation anxiety disorder concerns worries about the health and well-being of close attachments. In contrast, individuals with illness anxiety disorder worry about specific medical illnesses they themselves may have, not about them being separated from their close attachments.

Prolonged grief disorder. Intense yearning or longing for the deceased, intense sorrow and emotional pain, and preoccupation with the deceased or the circumstances of the death are expected responses occurring in prolonged grief disorder, whereas fear of possible separation from key attachment figures is central in separation anxiety disorder.

Depressive and bipolar disorders. These disorders may be associated with reluctance to leave home, but the main concern is not worry or fear of untoward events befalling attachment figures, but rather low motivation for engaging with the outside world. However, individuals with separation anxiety disorder may become depressed while being separated or in anticipation of separation.

Oppositional defiant disorder. Children and adolescents with separation anxiety disorder may be oppositional in the context of being forced to

separate from attachment figures. Oppositional defiant disorder should be considered only when there is persistent oppositional behavior unrelated to the anticipation or occurrence of separation from attachment figures.

Psychotic disorders. Unlike the hallucinations in psychotic disorders, the unusual perceptual experiences that may occur in separation anxiety disorder are usually based on a misperception of an actual stimulus, occur only in certain situations (e.g., nighttime), and are reversed by the presence of an attachment figure.

Personality disorders. Dependent personality disorder is characterized by an indiscriminate tendency to rely on others, whereas separation anxiety disorder involves concern about the proximity and safety of key attachment figures. Borderline personality disorder is characterized by fear of abandonment by loved ones, but problems in identity, self-direction, interpersonal functioning, and impulsivity are additionally central to that disorder, whereas they are not central to separation anxiety disorder.

Comorbidity

In children, separation anxiety disorder is highly comorbid with generalized anxiety disorder and specific phobia. In adults, common comorbidities include specific phobia, PTSD, panic disorder, generalized anxiety disorder, social anxiety disorder, agoraphobia, obsessive-compulsive disorder, prolonged grief disorder, and personality disorders. Among the personality disorders, dependent, avoidant, and obsessive-compulsive (Cluster C) personality disorders may be comorbid with separation anxiety disorder. Depressive and bipolar disorders are also comorbid with separation anxiety disorder in adults.

Selective Mutism

Diagnostic Criteria	F94.0

A. Consistent failure to speak in specific social situations in which there is an expectation for speaking (e.g., at school) despite speaking in other situations.
B. The disturbance interferes with educational or occupational achievement or with social communication.
C. The duration of the disturbance is at least 1 month (not limited to the first month of school).
D. The failure to speak is not attributable to a lack of knowledge of, or comfort with, the spoken language required in the social situation.
E. The disturbance is not better explained by a communication disorder (e.g., childhood-onset fluency disorder) and does not occur exclusively during the course of autism spectrum disorder, schizophrenia, or another psychotic disorder.

Diagnostic Features

When encountering other individuals in social interactions, children with selective mutism do not initiate speech or reciprocally respond when spoken to by others. Lack of speech occurs in social interactions with children or adults. Children with selective mutism will speak in their home in the presence of immediate family members but often not even in front of close friends or second-degree relatives, such as grandparents or cousins. The disturbance is most often marked by high social anxiety. Children with selective mutism often refuse to speak at school, leading to academic or educational impairment, as teachers often find it difficult to assess skills such as reading. The lack of speech may interfere with social communication, although children with this disorder sometimes use nonspoken or nonverbal means (e.g., grunting, pointing, writing) to communicate and may be willing or eager to perform or engage in social encounters when speech is not required (e.g., nonverbal parts in school plays).

Associated Features

Associated features of selective mutism may include excessive shyness, fear of social embarrassment, social isolation and withdrawal, clinging, compulsive traits, negativism, temper tantrums, or mild oppositional behavior. Although children with this disorder generally have normal language skills, there may occasionally be an associated communication disorder, although no particular association with a specific communication disorder has been identified. Even when these disorders are present, anxiety is present as well. In clinical settings, children with selective mutism are almost always given an additional diagnosis of another anxiety disorder—most commonly, social anxiety disorder.

Prevalence

Selective mutism is a relatively rare disorder and has not been included as a diagnostic category in epidemiological studies of prevalence of childhood disorders. Point prevalence using various clinic or school samples in the United States, Europe, and Israel ranges between 0.03% and 1.9% depending on the setting and ages of the sample. Studies in community-based and treatment-seeking samples suggest an equal gender distribution for selective mutism, although there is also evidence that selective mutism is more common among girls than boys. Prevalence does not seem to vary by race/ethnicity, but individuals who need to speak in a non-native language (e.g., children of immigrant families) run greater risk for developing the disorder. The disorder is more likely to manifest in young children than in adolescents and adults.

Development and Course

The onset of selective mutism is usually before age 5 years, but the disturbance may not come to clinical attention until entry into school, where there is an increase in social interaction and performance tasks, such as reading aloud. The persistence of the disorder is variable. Although clinical reports suggest that many individuals “outgrow” selective mutism, the longitudinal course of the disorder is largely unknown. In most cases,

selective mutism may fade, but symptoms of social anxiety disorder often remain.

Risk and Prognostic Factors

Temperamental. Temperamental risk factors for selective mutism are not well identified. Negative affectivity (neuroticism) or behavioral inhibition may play a role, as may parental history of shyness, social isolation, and social anxiety. Children with selective mutism may have subtle receptive language difficulties compared with their peers, although receptive language is still within the normal range.

Environmental. Social inhibition on the part of parents may serve as a model for social reticence and selective mutism in children. Furthermore, parents of children with selective mutism have been described as overprotective or more controlling than parents of children with other anxiety disorders or no disorder.

Genetic and physiological. Because of the significant overlap between selective mutism and social anxiety disorder, there may be shared genetic factors between these conditions. There is also evidence for increased abnormalities in the auditory efferent neural activity during vocalization in individuals with selective mutism, which could lead to peculiarities in the perception of their own voice and hence a reticence to speak.

Culture-Related Diagnostic Issues

Children in families who have migrated to a country where a different language is spoken may appear to have selective mutism because they may refuse to speak the new language because of lack of knowledge of the language. Such children would not qualify for the diagnosis because such cases are explicitly excluded from the diagnosis.

Functional Consequences of Selective Mutism

Selective mutism may result in social impairment, as children may be too anxious to engage in reciprocal social interaction with other children. As children with selective mutism mature, they may face increasing social isolation. In school settings, these children may suffer academic

impairment, because often they do not communicate with teachers regarding their academic or personal needs (e.g., not understanding a class assignment, not asking to use the restroom). Severe impairment in school and social functioning, including that resulting from teasing by peers, is common. In certain instances, selective mutism may serve as a compensatory strategy to decrease anxious arousal in social encounters.

Differential Diagnosis

Silent period in immigrant children learning a second language. Selective mutism must be distinguished from the typical "silent period" associated with the acquisition of a new language in young children. If comprehension of the new language is adequate but refusal to speak persists in both languages, in several unfamiliar settings, and for a prolonged period, a diagnosis of selective mutism may be warranted.

Communication disorders. Selective mutism should be distinguished from speech disturbances that are better explained by a communication disorder, such as language disorder, speech sound disorder (previously phonological disorder), childhood-onset

fluency disorder (stuttering), or social (pragmatic) communication disorder. Unlike selective mutism, the speech disturbance in these conditions is not restricted to a specific social situation.

Neurodevelopmental disorders and schizophrenia and other psychotic disorders. I ndividuals with an autism spectrum disorder, schizophrenia or another psychotic disorder, or severe intellectual developmental disorder (intellectual disability) may have problems in social communication and be unable to speak appropriately in social situations. In contrast, selective mutism should be diagnosed only when a child has an established capacity to speak in some social situations (e.g., typically at home).

Social anxiety disorder. The social anxiety and social avoidance in social anxiety disorder may be associated with selective mutism. In such cases, both diagnoses may be given.

Comorbidity

The most common comorbid conditions are other anxiety disorders, most often social anxiety disorder, followed by separation anxiety disorder and specific phobia. In clinical settings, selective mutism and autism spectrum disorder have also been noted as frequently co-occurring conditions. Oppositional behaviors can be observed in a substantial minority of children with selective mutism, although this oppositional behavior may be limited to situations requiring speech. Communication delays or disorders also may appear in some children with selective mutism.

Specific Phobia

Diagnostic Criteria

A. Marked fear or anxiety about a specific object or situation (e.g., flying, heights, animals, receiving an injection, seeing blood).

   Note: In children, the fear or anxiety may be expressed by crying, tantrums, freezing, or clinging.

B. The phobic object or situation almost always provokes immediate fear or anxiety.

C. The phobic object or situation is actively avoided or endured with intense fear or anxiety.

D. The fear or anxiety is out of proportion to the actual danger posed by the specific object or situation and to the sociocultural context.

E. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.

F. The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

G. The disturbance is not better explained by the symptoms of another mental disorder, including fear, anxiety, and avoidance of situations associated with panic-like symptoms or other incapacitating symptoms (as in agoraphobia); objects or situations related to obsessions (as in obsessive-compulsive disorder); reminders of traumatic events (as in posttraumatic stress disorder); separation from home or attachment figures (as in separation anxiety disorder); or social situations (as in social anxiety disorder).

Specify if:

Code based on the phobic stimulus:

F40.218 Animal (e.g., spiders, insects, dogs).

F40.228 Natural environment (e.g., heights, storms, water).

F40.23x Blood-injection-injury (e.g., needles, invasive medical procedures).

Coding note: Select specific ICD-10-CM code as follows: **F40.230** fear of blood; **F40.231** fear of injections and transfusions; **F40.232** fear of other medical care; or **F40.233** fear of injury.

F40.248 Situational (e.g., airplanes, elevators, enclosed places).

F40.298 Other (e.g., situations that may lead to choking or vomiting; in children, e.g., loud sounds or costumed characters).

Coding note: When more than one phobic stimulus is present, code all ICD-10-CM codes that apply (e.g., for fear of snakes and flying, F40.218 specific phobia, animal, and F40.248 specific phobia, situational).

Specifiers

It is common for individuals to have multiple specific phobias. The average individual with specific phobia fears three objects or situations, and approximately 75% of individuals with specific phobia fear more than one situation or object. In such cases, multiple specific phobia diagnoses, each with its own diagnostic code reflecting the phobic stimulus, would need to be given. For example, if an individual fears thunderstorms and flying, then two diagnoses would be given: specific phobia, natural environment, and specific phobia, situational.

Diagnostic Features

A key feature of this disorder is that the fear or anxiety is circumscribed to the presence of a particular situation or object (Criterion A), which may be termed the *phobic stimulus.* The categories of feared situations or objects are provided as specifiers. Many individuals fear objects or situations from more than one category, or phobic stimulus. For the diagnosis of specific phobia, the response must differ from normal, transient fears that commonly occur in the population. To meet the criteria for a diagnosis, the fear or anxiety must be intense or severe (i.e., "marked") (Criterion A). The amount of fear experienced may vary with proximity to the feared object or situation and may occur in anticipation of or in the actual presence of the object or situation. Also, the fear or anxiety may take the form of a full or limited symptom panic attack (i.e., expected panic attack). Another characteristic of specific phobias is that fear or anxiety is evoked nearly every time the individual comes into contact with the phobic stimulus (Criterion B). Thus, an individual who becomes anxious only occasionally upon being confronted with the situation or object (e.g., becomes anxious when flying only on one out of every five airplane flights) would not be diagnosed with specific phobia. However, the degree of fear or anxiety expressed may vary (from anticipatory anxiety to a full panic attack) across different occasions of encountering the phobic object or situation because of various contextual factors such as the presence of others, duration of exposure, and other threatening elements such as turbulence on a flight for individuals who fear flying. Fear and anxiety are often expressed differently between children and adults. Also, the fear or anxiety occurs as soon as the

phobic object or situation is encountered (i.e., immediately rather than being delayed).

The individual actively avoids the situation, or if he or she either is unable or decides not to avoid it, the situation or object evokes intense fear or anxiety (Criterion C). *Active avoidance* means the individual intentionally behaves in ways that are designed to prevent or minimize contact with phobic objects or situations (e.g., takes tunnels instead of bridges on daily commute to work for fear of heights; avoids entering a dark room for fear of spiders; avoids accepting a job in a locale where a phobic stimulus is more common). Avoidance behaviors are often obvious (e.g., an individual who fears blood refusing to go to the doctor) but are sometimes less obvious (e.g., an individual who fears snakes refusing to look at pictures that resemble the form or shape of snakes). Many individuals with specific phobias have suffered over many years and have changed their living circumstances in

ways designed to avoid the phobic object or situation as much as possible (e.g., an individual diagnosed with specific phobia, animal, who moves to reside in an area devoid of the particular feared animal). Therefore, they no longer experience fear or anxiety in their daily life. In such instances, avoidance behaviors or ongoing refusal to engage in activities that would involve exposure to the phobic object or situation (e.g., repeated refusal to accept offers for work-related travel because of fear of flying) may be helpful in confirming the diagnosis in the absence of overt anxiety or panic.

The fear or anxiety is out of proportion to the actual danger that the object or situation poses, or more intense than is deemed necessary (Criterion D). Although individuals with specific phobia often recognize their reactions as disproportionate, they tend to overestimate the danger in their feared situations, and thus the judgment of being out of proportion is made by the clinician. The individual’s sociocultural context should also be considered. For example, fears of the dark may be reasonable in a context of ongoing violence, and the degree of fear of insects considered to be disproportionate would be higher in settings where insects are consumed in the diet. The fear, anxiety, or avoidance is persistent, typically lasting for 6

months or more (Criterion E), which helps distinguish the disorder from transient fears that are common in the population, particularly among children. The specific phobia must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning in order for the disorder to be diagnosed (Criterion F).

Associated Features

Individuals with specific phobia typically experience an increase in physiological arousal in anticipation of or during exposure to a phobic object or situation. However, the physiological response to the feared situation or object varies. Whereas individuals with situational, natural environment, and animal specific phobias are likely to show sympathetic nervous system arousal, individuals with blood-injection-injury specific phobia often demonstrate a vasovagal fainting or near-fainting response that is marked by initial brief acceleration of heart rate and elevation of blood pressure followed by a deceleration of heart rate and a drop in blood pressure. Additionally, specific phobia is most consistently associated with abnormal activity in the amygdala, anterior cingulate cortex, thalamus, and insula in response to the phobic object/situation.

Prevalence

In the United States, the 12-month community prevalence estimate for specific phobia is approximately 8%–12%. Prevalence rates in European countries are largely similar to those in the United States (e.g., about 6%), but rates are generally lower in Asian, African, and Latin American countries (2%–4%). Prevalence estimates in children average approximately 5% across various countries, with a range of 3%–9%, and are approximately 16% in adolescents ages 13–17 years in the United States. Prevalence estimates are lower in older individuals (about 3%–5%), possibly reflecting diminishing severity to subclinical levels. Women are more frequently affected than men across subtypes, at a rate of approximately 2:1.

Development and Course

Specific phobia sometimes develops following a traumatic event (e.g., being attacked by an animal or stuck in an elevator), observation of others going through a traumatic event (e.g., watching someone drown), an unexpected panic attack in the to be feared situation (e.g., an unexpected panic attack while on the subway), or informational transmission (e.g., extensive media coverage of a plane crash). However, many individuals with specific phobia are unable to recall the specific reason for the onset of their phobias. Specific phobia usually develops in early childhood, with the majority of cases developing prior to age 10 years. Median age at onset is between 7 and 11 years, with the mean at about 10 years.

Situational specific phobias tend to have a later age at onset than natural environment, animal, or blood-injection-injury specific phobias. Specific phobias that develop in childhood and adolescence are likely to wax and wane during that period. However, phobias that do persist into adulthood are unlikely to remit for the majority of individuals.

When specific phobia is being diagnosed in children, two issues should be considered. First, young children may express their fear and anxiety by crying, tantrums, freezing, or clinging. Second, young children typically are not able to understand the concept of avoidance. Therefore, the clinician should assemble additional information from parents, teachers, or others who know the child well. Excessive fears are quite common in young children but are usually transitory and only mildly impairing and thus considered developmentally appropriate. In such cases a diagnosis of specific phobia would not be made. When the diagnosis of specific phobia is being considered in a child, it is important to assess the degree of impairment and the duration of the fear, anxiety, or avoidance, and whether it is typical for the child’s particular developmental stage.

Although the prevalence of specific phobia is lower in older populations, it remains one of the more commonly experienced disorders in late life. Several issues should be considered when diagnosing specific phobia in older populations. First, older individuals may be more likely to endorse natural environment specific phobias, as well as phobias of falling. Second, specific phobia (like all anxiety disorders) tends to co-occur with

medical concerns in older individuals, including coronary heart disease, chronic obstructive pulmonary disease, and Parkinson's disease. Third, older individuals may be more likely to attribute the symptoms of anxiety to medical conditions. Fourth, older individuals may be more likely to manifest anxiety in an atypical manner (e.g., involving symptoms of both anxiety and depression) and thus be more likely to warrant a diagnosis of unspecified anxiety disorder. Additionally, the presence of specific phobia in older adults is associated with decreased quality of life and may serve as a risk factor for major neurocognitive disorder.

Although most specific phobias develop in childhood and adolescence, it is possible for a specific phobia to develop at any age, often as the result of experiences that are traumatic. For example, phobias of choking almost always follow a near-choking event at any age.

Risk and Prognostic Factors

Temperamental. Temperamental risk factors for specific phobia, such as negative affectivity (neuroticism) or behavioral inhibition, are risk factors for other anxiety disorders as well.

Environmental. Environmental risk factors for specific phobias, such as parental overprotectiveness, parental loss and separation, and physical and sexual abuse, tend to predict other anxiety disorders as well. As noted earlier, negative or traumatic encounters with the feared object or situation sometimes (but not always) precede the development of specific phobia.

Genetic and physiological. There may be a genetic susceptibility to a certain category of specific phobia (e.g., an individual with a first-degree relative with a specific phobia of animals is significantly more likely to have the same type of specific phobia than any other category of phobia). Twin studies have examined the heritability of individual subtypes of fears and phobias, suggesting that animal phobia has approximately 32% heritability, blood-injury-injection phobia has 33%, and situational phobia has 25%.

Culture-Related Diagnostic Issues

In the United States, individuals of Asian and Latinx descent report lower prevalence of specific phobia than non-Latinx Whites and African

Americans. The prevalence of specific phobia subtypes varies cross-nationally.

Sex- and Gender-Related Diagnostic Issues

Animal, natural environment, and situational specific phobias are predominantly experienced by women, whereas blood-injection-injury phobia is experienced nearly equally among women and men. The average age at onset of specific phobia during childhood does not differ between girls/women and boys/men.

Association With Suicidal Thoughts or Behavior

Specific phobia is associated with both suicidal thoughts and suicide attempts based on national U.S. survey data. Specific phobia is also associated with a transition from ideation to attempt. For individuals in the community ages 14–24 years, a large prospective study over a 10-year period in Germany found that 30% of first suicide attempts could be attributable to specific phobia.

Functional Consequences of Specific Phobia

Individuals with specific phobia show similar patterns of impairment in psychosocial functioning and decreased quality of life as individuals with other anxiety disorders and alcohol and substance use disorders, including impairments in occupational and interpersonal functioning. In older adults, impairment may be seen in caregiving duties and volunteer activities. Also, fear of falling in older adults can lead to reduced mobility and reduced physical and social functioning, and may lead to receiving formal or informal home support. The distress and impairment caused by specific phobias tend to increase with the number of feared objects and situations. Thus, an individual who fears four objects or situations is likely to have more impairment in his or her occupational and social roles and a lower quality of life than an individual who fears only one object or situation. Individuals with blood-injection-injury specific phobia are often reluctant to

obtain medical care even when a medical concern is present. Additionally, fear of vomiting and choking may substantially reduce dietary intake.

Differential Diagnosis

Agoraphobia. Situational specific phobia may resemble agoraphobia in its clinical presentation, given the overlap in feared situations (e.g., flying, enclosed places, elevators). If an individual fears only one of the agoraphobic situations, then specific phobia, situational, may be diagnosed. If two or more agoraphobic situations are feared, a diagnosis of agoraphobia is likely warranted. For example, an individual who fears airplanes and elevators (which overlap with the "public transportation" agoraphobic situation) but does not fear other agoraphobic situations would be diagnosed with specific phobia, situational, whereas an individual who fears airplanes, elevators, and crowds (which overlap with two agoraphobic situations, "using public transportation" and "standing in line or being in a crowd") would be diagnosed with agoraphobia. Criterion B of agoraphobia (the situations are feared or avoided "because of thoughts that escape might be difficult or help might not be available in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms") can also be useful in differentiating agoraphobia from specific phobia. If the situations are feared for reasons other than not being able to escape or get help, such as fear of being harmed directly by the object or situation (e.g., fear of the plane crashing, fear of the animal biting), a specific phobia diagnosis may be more appropriate.

Social anxiety disorder. If the situations are feared because of negative evaluation, social anxiety disorder should be diagnosed instead of specific phobia.

Separation anxiety disorder. If the situations are feared because of separation from a primary caregiver or attachment figure, separation anxiety disorder should be diagnosed instead of specific phobia.

Panic disorder. Individuals with specific phobia may experience panic attacks when confronted with their feared situation or object. A diagnosis of

specific phobia would be given if the panic attacks only occurred in response to the specific object or situation, whereas a diagnosis of panic disorder would be given if the individual also experienced panic attacks that were unexpected (i.e., not in response to the specific phobia object or situation).

Obsessive-compulsive disorder. If an individual's primary fear or anxiety is of an object or situation as a result of obsessions (e.g., fear of blood due to obsessive thoughts about contamination from blood-borne pathogens [i.e., HIV]; fear of driving due to obsessive images of harming others), and if other diagnostic criteria for obsessive-compulsive disorder are met, then obsessive-compulsive disorder should be diagnosed.

Trauma- and stressor-related disorders. If the phobia develops following a traumatic event, posttraumatic stress disorder (PTSD) should be considered as a diagnosis. However, traumatic events can precede the onset of PTSD and specific phobia. In this case, a diagnosis of specific phobia would be assigned only if all of the criteria for PTSD are not met.

Eating disorders. A diagnosis of specific phobia is not given if the avoidance behavior is exclusively limited to avoidance of food and food-related cues, in which case a diagnosis of anorexia nervosa or bulimia nervosa should be considered.

Schizophrenia spectrum and other psychotic disorders. When the fear and avoidance are attributable to delusional thinking (as in schizophrenia or other schizophrenia spectrum and other psychotic disorders), a diagnosis of specific phobia is not warranted.

Comorbidity

Specific phobia is rarely seen in medical-clinical settings in the absence of other psychopathology and is more frequently seen in nonmedical mental health settings. Specific phobia is frequently associated with a range of other disorders. Because of early onset, specific phobia is typically the temporally primary disorder. Individuals with specific phobia are at increased risk for the development of other disorders, including other anxiety disorders, depressive and bipolar disorders, substance-related

disorders, somatic symptom and related disorders, and personality disorders (particularly dependent personality disorder).

Social Anxiety Disorder

Diagnostic Criteria F40.10

A. Marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others. Examples include social interactions (e.g., having a conversation, meeting unfamiliar people), being observed (e.g., eating or drinking), and performing in front of others (e.g., giving a speech).

   Note: In children, the anxiety must occur in peer settings and not just during interactions with adults.

B. The individual fears that he or she will act in a way or show anxiety symptoms that will be negatively evaluated (i.e., will be humiliating or embarrassing; will lead to rejection or offend others).

C. The social situations almost always provoke fear or anxiety.

   Note: In children, the fear or anxiety may be expressed by crying, tantrums, freezing, clinging, shrinking, or failing to speak in social situations.

D. The social situations are avoided or endured with intense fear or anxiety.

E. The fear or anxiety is out of proportion to the actual threat posed by the social situation and to the sociocultural context.

F. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.

G. The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

H. The fear, anxiety, or avoidance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

I. The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder, such as panic disorder, body dysmorphic disorder, or autism spectrum disorder.

J. If another medical condition (e.g., Parkinson's disease, obesity, disfigurement from burns or injury) is present, the fear, anxiety, or avoidance is clearly unrelated or is excessive.

Specify if:

Performance only: If the fear is restricted to speaking or performing in public.

Specifiers

Individuals with the performance only type of social anxiety disorder have performance fears that are typically most impairing in their professional lives (e.g., musicians, dancers, performers, athletes) or in roles that require regular public speaking. Performance fears may also manifest in work, school, or academic settings in which regular public presentations are required. Individuals with performance only social anxiety disorder do not fear or avoid nonperformance social situations.

Diagnostic Features

The essential feature of social anxiety disorder is a marked, or intense, fear or anxiety of social situations in which the individual may be scrutinized by others. In children the fear or anxiety must occur in peer settings and not just during interactions with adults (Criterion A). When exposed to such social situations, the individual fears that he or she will be negatively evaluated. The individual is concerned that he or she will be judged as anxious, weak, crazy, stupid, boring, intimidating, dirty, or unlikable. The

individual fears that he or she will act or appear in a certain way or show anxiety symptoms, such as blushing, trembling, sweating, stumbling over one's words, or staring, that will be negatively evaluated by others (Criterion B). Some individuals fear offending others or being rejected as a result. Fear of offending others—for example, by a gaze or by showing anxiety symptoms—may be the predominant fear in individuals from cultures with strong collectivistic orientations. An individual with fear of trembling of the hands may avoid drinking, eating, writing, or pointing in public; an individual with fear of sweating may avoid shaking hands or eating spicy foods; and an individual with fear of blushing may avoid public performance, bright lights, or discussion about intimate topics. Some individuals fear and avoid urinating in public restrooms when other individuals are present (i.e., paruresis, or "shy bladder syndrome").

The social situations almost always provoke fear or anxiety (Criterion C). Thus, an individual who becomes anxious only occasionally in the social situation(s) would not be diagnosed with social anxiety disorder. However, the degree and type of fear and anxiety may vary (e.g., anticipatory anxiety, a panic attack) across different occasions. The anticipatory anxiety may occur sometimes far in advance of upcoming situations (e.g., worrying every day for weeks before attending a social event, repeating a speech for days in advance). In children, the fear or anxiety may be expressed by crying, tantrums, freezing, clinging, or shrinking in social situations. The individual will often avoid the feared social situations. Alternatively, the situations are endured with intense fear or anxiety (Criterion D). Avoidance can be extensive (e.g., not going to parties, refusing school) or subtle (e.g., overpreparing the text of a speech, diverting attention to others, limiting eye contact).

The fear or anxiety is judged to be out of proportion to the actual risk of being negatively evaluated or to the consequences of such negative evaluation (Criterion E).

Sometimes, the anxiety may not be judged to be excessive, because it is related to an actual danger (e.g., being bullied or tormented by others). However, individuals with social anxiety disorder often overestimate the

negative consequences of social situations, and thus the judgment of being out of proportion is made by the clinician. The individual's sociocultural context needs to be taken into account when this judgment is being made. For example, in certain cultures, behavior that might otherwise appear socially anxious may be considered appropriate in social situations (e.g., might be seen as a sign of respect).

The duration of the disturbance is typically at least 6 months (Criterion F). This duration threshold helps distinguish the disorder from transient social fears that are common, particularly among children and in the community. The fear, anxiety, and avoidance must interfere significantly with the individual's normal routine, occupational or academic functioning, or social activities or relationships, or must cause clinically significant distress (Criterion G). For example, an individual who is afraid to speak in public would not receive a diagnosis of social anxiety disorder if this activity is not routinely encountered on the job or in classroom work, and if the individual is not significantly distressed about it. However, if the individual avoids, or is passed over for, the job or education he or she really wants because of social anxiety symptoms, Criterion G is met.

Associated Features

Individuals with social anxiety disorder may be inadequately assertive or excessively submissive or, less commonly, highly controlling of the conversation. They may show overly rigid body posture or inadequate eye contact, or speak with an overly soft voice. These individuals may be shy or withdrawn, and they may be less open in conversations and disclose little about themselves. They may seek employment in jobs that do not require social contact, although this is not the case for individuals with social anxiety disorder, performance only. They may live at home longer. Men may be delayed in marrying and having a family, whereas women who would want to work outside the home may live a life without ever doing so. Self-medication with substances is common (e.g., drinking before going to a party). Social anxiety among older adults may also include exacerbation of symptoms of medical illnesses, such as increased tremor or tachycardia. Blushing is a hallmark physical response of social anxiety disorder.

Prevalence

The 12-month prevalence estimate of social anxiety disorder for the United States is approximately 7%. Lower 12-month prevalence estimates are seen in much of the world using the same diagnostic instrument, clustering around 0.5%–2.0%; median prevalence in Europe is 2.3%. Prevalence appears to be increasing in the United States and East Asian countries. Twelve-month prevalence rates in young adolescents (ages 13–17 years) are roughly half those in adults. Twelve-month prevalence rates decrease after age 65. The 12-month prevalence for older adults in North America, Europe, and Australia ranges from 2% to 5%. In general, higher rates of social anxiety disorder are found in women than in men in the general population (with odds ratios ranging from 1.5 to 2.2), and the gender difference in prevalence is more pronounced in adolescents and young adults. Gender rates are equivalent or slightly higher for men in clinical samples, and it is assumed that gender roles and social expectations play a significant role in explaining the heightened help-seeking behavior in men. Prevalence in the United States has been found to be lower in individuals of Asian, Latinx, African American, and Caribbean Black descent compared with non-Hispanic Whites.

Development and Course

Median age at onset of social anxiety disorder in the United States is 13 years, and 75% of individuals have an age at onset between 8 and 15 years. The disorder sometimes emerges

out of a childhood history of social inhibition or shyness in U.S. and European studies. Onset can also occur in early childhood. Onset of social anxiety disorder may follow a stressful or humiliating experience (e.g., being bullied, vomiting during a public speech), or it may be insidious, developing slowly. First onset in adulthood is relatively rare and is more likely to occur after a stressful or humiliating event or after life changes that require new social roles (e.g., marrying someone from a different social class, receiving a job promotion). Social anxiety disorder may diminish

after an individual with fear of dating marries and may reemerge after divorce. Among individuals presenting to clinical care, the disorder tends to be particularly persistent.

Adolescents endorse a broader pattern of fear and avoidance, including of dating, compared with younger children. Older adults express social anxiety at lower levels but across a broader range of situations, whereas younger adults express higher levels of social anxiety for specific situations. In older adults, social anxiety may concern disability due to declining sensory functioning (hearing, vision) or embarrassment about one's appearance (e.g., tremor as a symptom of Parkinson's disease) or functioning due to medical conditions, incontinence, or cognitive impairment (e.g., forgetting people's names). Detection of social anxiety disorder in older adults may be challenging because of several factors, including a focus on somatic symptoms, comorbid medical illness, limited insight, changes to social environment or roles that may obscure impairment in social functioning, or reticence about describing psychological distress. There is large variation in rates of remission for social anxiety disorder, suggestive of different trajectories (short, fluctuating, and chronic).

Risk and Prognostic Factors

Temperamental. Underlying traits that predispose individuals to social anxiety disorder include behavioral inhibition and fear of negative evaluation, as well as harm avoidance. Personality traits consistently associated with social anxiety disorder are high negative affectivity (neuroticism) and low extraversion.

Environmental. There is evidence that negative social experiences, particularly peer victimization, are associated with the development of social anxiety disorder, although causal pathways remain unknown. Childhood maltreatment and adversity are risk factors for social anxiety disorder. Among African Americans and Caribbean Blacks in the United States, everyday forms of ethnic discrimination and racism are associated with social anxiety disorder.

Genetic and physiological. Traits predisposing individuals to social anxiety disorder, such as behavioral inhibition, are strongly genetically influenced.

The genetic influence is subject to gene-environment interaction; that is, children with high behavioral inhibition are more susceptible to environmental influences, such as socially anxious modeling by parents. Also, social anxiety disorder is heritable. First-degree relatives have a two to six times greater chance of having social anxiety disorder, and liability to the disorder involves the interplay of disorder-specific (e.g., fear of negative evaluation) and nonspecific (e.g., negative affectivity [neuroticism]) genetic factors. Genetic contribution to social anxiety disorder has been found to be higher for social anxiety disorder in children than social anxiety disorder in adults and higher for social anxiety symptoms than a clinical diagnosis of social anxiety disorder.

Culture-Related Diagnostic Issues

The nature and types of social situations that precipitate symptoms of social anxiety disorder are similar across U.S. ethnoracial groups, including fear of performance/public speaking, social interaction, and being observed. U.S. non-Latinx Whites report an earlier age at onset of social anxiety disorder than U.S. Latinx, yet the latter describe greater impairment across home, work, and relationship domains associated with the disorder.

Immigrant status is associated with lower rates of social anxiety disorder in both Latinx and non-Latinx White groups. The syndrome of *taijin kyofusho* (e.g., in Japan and Korea) is often characterized by social-evaluative concerns, fulfilling criteria for social anxiety disorder, which are associated with the fear that the individual makes *other* people uncomfortable (e.g., “My gaze upsets people so they look away and avoid me”), a fear that is at times experienced with delusional intensity. Other presentations of *taijin kyofusho* may fulfill criteria for body dysmorphic disorder or delusional disorder.

Sex- and Gender-Related Diagnostic Issues

Age at onset of social anxiety disorder does not differ by gender. Women with social anxiety disorder report a greater number of social fears and

comorbid major depressive disorder and other anxiety disorders, whereas men are more likely to fear dating; have oppositional defiant disorder, conduct disorder, or antisocial personality disorder; and use alcohol and illicit drugs to relieve symptoms of the disorder. Paruresis is more common in men.

Association With Suicidal Thoughts or Behavior

Among U.S. adolescents, social anxiety disorder has been reported to increase the risk for active suicidal thoughts and suicide attempts in Latinx but not in non-Latinx Whites, independently of the effect of major depression and family income.

Functional Consequences of Social Anxiety Disorder

Social anxiety disorder is associated with elevated rates of school dropout and with decreased well-being, employment, workplace productivity, socioeconomic status, and quality of life. Social anxiety disorder is also associated with being single, unmarried, or divorced and with not having children, particularly among men, whereas women are more likely to be unemployed. Social anxiety disorder is also negatively associated with friendship quality, such that individuals with social anxiety disorder report having friendships that are less close and less supportive than persons without the disorder. In older adults, there may be impairment in caregiving duties and volunteer activities. Social anxiety disorder also impedes leisure activities. Despite the extent of distress and social impairment associated with social anxiety disorder, only about half of individuals with the disorder in high-income societies ever seek treatment, and they tend to do so only after 15–20 years of experiencing symptoms. Not being employed is a strong predictor for the persistence of social anxiety disorder.

Differential Diagnosis

Normative shyness. Shyness (i.e., social reticence) is a common personality trait and is not by itself pathological. In some societies, shyness is even evaluated positively. However, when there is a significant adverse impact on social, occupational, and other important areas of functioning, a

diagnosis of social anxiety disorder should be considered, and when full diagnostic criteria for social anxiety disorder are met, the disorder should be diagnosed. Only a minority (12%) of self-identified shy individuals in the United States have symptoms that meet diagnostic criteria for social anxiety disorder.

Agoraphobia. Individuals with agoraphobia may fear and avoid social situations (e.g., going to a movie) because escape might be difficult or help might not be available in the event of incapacitation or panic-like symptoms, whereas individuals with social anxiety disorder are most fearful of scrutiny by others. Moreover, individuals with social anxiety disorder are likely to be calm when left entirely alone, which is often not the case in agoraphobia.

Panic disorder. Individuals with social anxiety disorder may have panic attacks, but the panic attacks are always cued by social situations and do not occur “out of the blue.” Additionally, individuals with social anxiety disorder are more likely to be distressed by fear of negative evaluation stemming from a panic attack than by the panic attacks themselves.

Generalized anxiety disorder. Social worries are common in generalized anxiety disorder, but the focus is more on the nature of ongoing relationships rather than on fear of negative evaluation. Individuals with generalized anxiety disorder, particularly children, may have excessive worries about the quality of their social performance, but these worries also pertain to nonsocial performance and when the individual is not being evaluated by others. In social anxiety disorder, the worries focus on social performance and others’ evaluation.

Separation anxiety disorder. Individuals with separation anxiety disorder may avoid social settings (including school refusal) because of concerns about being separated from attachment figures or, in children, about requiring the presence of a parent when it is not developmentally appropriate. Individuals with separation anxiety disorder are usually comfortable in social settings when their attachment figure is present or when they are at home, whereas

those with social anxiety disorder may be uncomfortable when social situations occur at home or in the presence of attachment figures.

Specific phobias. Individuals with specific phobias may fear embarrassment or humiliation (e.g., embarrassment about fainting when they have their blood drawn), but they do not generally fear negative evaluation in other social situations.

Selective mutism. Individuals with selective mutism may fail to speak because of fear of negative evaluation, but they do not fear negative evaluation in social situations where no speaking is required (e.g., nonverbal play).

Major depressive disorder. Individuals with major depressive disorder may be concerned about being negatively evaluated by others because they feel they are bad or not worthy of being liked. In contrast, individuals with social anxiety disorder are worried about being negatively evaluated because of certain social behaviors or physical symptoms.

Body dysmorphic disorder. Individuals with body dysmorphic disorder are preoccupied with one or more perceived defects or flaws in their physical appearance that are not observable or appear slight to others; this preoccupation often causes social anxiety and avoidance. If their social fears and avoidance are caused only by their beliefs about their appearance, a separate diagnosis of social anxiety disorder is not warranted.

Delusional disorder. Individuals with delusional disorder may have nonbizarre delusions and/or hallucinations related to the delusional theme that focus on being rejected by or offending others. Although extent of insight into beliefs about social situations may vary, many individuals with social anxiety disorder have good insight that their beliefs are out of proportion to the actual threat posed by the social situation.

Autism spectrum disorder. Social anxiety and social communication deficits are hallmarks of autism spectrum disorder. Individuals with social anxiety disorder typically have adequate age-appropriate social relationships and social communication capacity, although they may appear to have impairment in these areas when first interacting with unfamiliar peers or adults.

Personality disorders.

Given its frequent onset in childhood and its persistence into and through adulthood, social anxiety disorder may resemble a personality disorder. The most apparent overlap is with avoidant personality disorder. Individuals with avoidant personality disorder have a broader avoidance pattern and higher rates of impairment than those individuals with social anxiety disorder. Moreover, individuals with avoidant personality disorder have a strong and pervasively negative self-concept, a view of rejection as equating to a global evaluation of the self as being of little worth, and a sense of not fitting in

socially that dates from early childhood. Nonetheless, social anxiety disorder is typically more comorbid with avoidant personality disorder than with any other personality disorder, and avoidant personality disorder is more comorbid with social anxiety disorder than with any other anxiety disorder.

Other mental disorders. Social fears and discomfort can occur as part of schizophrenia, but other evidence for psychotic symptoms is usually present. In individuals with an eating disorder, it is important to determine that fear of negative evaluation about eating disorder symptoms or behaviors (e.g., purging and vomiting) is not the sole source of social anxiety before applying a diagnosis of social anxiety disorder. Similarly, obsessive-compulsive disorder may be associated with social anxiety, but the additional diagnosis of social anxiety disorder is used only when social fears and avoidance are independent of the foci of the obsessions and compulsions.

Other medical conditions. Medical conditions may produce symptoms that may be embarrassing (e.g., trembling in Parkinson’s disease). When the fear of negative evaluation due to other medical conditions is judged to be excessive, a diagnosis of social anxiety disorder should be considered.

Oppositional defiant disorder. Refusal to speak because of opposition to authority figures should be differentiated from failure to speak because of fear of negative evaluation.

Comorbidity

Social anxiety disorder is often comorbid with other anxiety disorders, major depressive disorder, and substance use disorders, and the onset of social anxiety disorder generally precedes that of the other disorders, except for specific phobia and separation anxiety disorder. Chronic social isolation in the course of social anxiety disorder may result in major depressive disorder. Comorbidity with depression is high also in older adults. Substances may be used as self-medication for social fears, but the symptoms of substance intoxication or withdrawal, such as trembling, may also be a source of (further) social fear. Social anxiety disorder is frequently comorbid with body dysmorphic disorder, and generalized social anxiety disorder is often comorbid with avoidant personality disorder. In children, comorbidities with high-functioning autism spectrum disorder and selective mutism are common.

Panic Disorder

Diagnostic Criteria F41.0

A. Recurrent unexpected panic attacks. A panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes, and during which time four (or more) of the following symptoms occur:

   Note: The abrupt surge can occur from a calm state or an anxious state.

   1. Palpitations, pounding heart, or accelerated heart rate.
   2. Sweating.
   3. Trembling or shaking.
   4. Sensations of shortness of breath or smothering.
   5. Feelings of choking.
   6. Chest pain or discomfort.
   7. Nausea or abdominal distress.

8. Feeling dizzy, unsteady, light-headed, or faint.
9. Chills or heat sensations.

10. Paresthesias (numbness or tingling sensations).
11. Derealization (feelings of unreality) or depersonalization (being detached from oneself).
12. Fear of losing control or “going crazy.”
13. Fear of dying.

Note: Culture-specific symptoms (e.g., tinnitus, neck soreness, headache, uncontrollable screaming or crying) may be seen. Such symptoms should not count as one of the four required symptoms.

B. At least one of the attacks has been followed by 1 month (or more) of one or both of the following:
   1. Persistent concern or worry about additional panic attacks or their consequences (e.g., losing control, having a heart attack, “going crazy”).
   2. A significant maladaptive change in behavior related to the attacks (e.g., behaviors designed to avoid having panic attacks, such as avoidance of exercise or unfamiliar situations).

C. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism, cardiopulmonary disorders).

D. The disturbance is not better explained by another mental disorder (e.g., the panic attacks do not occur only in response to feared social situations, as in social anxiety disorder; in response to circumscribed phobic objects or situations, as in specific phobia; in response to obsessions, as in obsessive-compulsive disorder; in response to reminders of traumatic

events, as in posttraumatic stress disorder; or in response to separation from attachment figures, as in separation anxiety disorder).

Diagnostic Features

Panic disorder is characterized by recurrent unexpected panic attacks (Criterion A). (For a detailed description of symptoms and course characterizing a panic attack, see Panic Attack Specifier, “Features” section, following this text on panic disorder.) A *panic attack* is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes, and during which time 4 or more of a list of 13 physical and cognitive symptoms occur. The term *recurrent* means more than one unexpected panic attack. The term *unexpected* refers to a panic attack for which there is no obvious cue or trigger at the time of occurrence—that is, the attack appears to occur from out of the blue, such as when the individual is relaxing or emerging from sleep (nocturnal panic attack). In contrast, *expected* panic attacks are those for which there is an obvious cue or trigger, such as a situation in which panic attacks have typically occurred. The determination of whether panic attacks are expected or unexpected is made by the clinician, who makes this judgment based on a combination of careful questioning as to the sequence of events preceding or leading up to the attack and the individual’s own judgment of whether the attack seemed to occur for no apparent reason. Cultural interpretations may influence the assignment of panic attacks as expected or unexpected (see section “Culture-Related Diagnostic Issues” for this disorder). In the United States and Europe, approximately one-half of individuals with panic disorder have expected panic attacks as well as unexpected panic attacks. Thus, the presence of expected panic attacks does not rule out the diagnosis of panic disorder.

The frequency and severity of panic attacks vary widely. In terms of frequency, there may be moderately frequent attacks (e.g., one per week) for months at a time, or short bursts of more frequent attacks (e.g., daily) separated by weeks or months without any attacks or with less frequent attacks (e.g., two per month) over many years. Individuals who have infrequent panic attacks resemble those with more frequent panic attacks in

terms of panic attack symptoms, demographic characteristics, comorbidity with other disorders, family

history, and biological data. In terms of severity, individuals with panic disorder may have both full-symptom (four or more symptoms) and limited-symptom (fewer than four symptoms) attacks, and the number and type of panic attack symptoms frequently differ from one panic attack to the next. However, more than one unexpected full-symptom panic attack is required for the diagnosis of panic disorder.

A *nocturnal* panic attack (i.e., waking from sleep in a state of panic) differs from panicking after fully waking from sleep. In the United States, nocturnal panic attack has been estimated to occur at least one time in roughly one-quarter to one-third of individuals with panic disorder, of whom the majority also have daytime panic attacks. Individuals with both daytime and nocturnal panic attacks tend to have more severe panic disorder overall.

The worries about panic attacks or their consequences usually pertain to physical concerns, such as worry that panic attacks reflect the presence of life-threatening illnesses (e.g., cardiac disease, seizure disorder); social concerns, such as embarrassment or fear of being judged negatively by others because of visible panic symptoms; and concerns about mental functioning, such as “going crazy” or losing control (Criterion B). Individuals who report fears of dying in their panic attacks tend to have more severe presentations of panic disorder (e.g., panic attacks involving more symptoms). The maladaptive changes in behavior represent attempts to minimize or avoid panic attacks or their consequences. Examples include avoiding physical exertion, reorganizing daily life to ensure that help is available in the event of a panic attack, restricting usual daily activities, and avoiding agoraphobia-type situations, such as leaving home, using public transportation, or shopping. If agoraphobia is present, a separate diagnosis of agoraphobia is given.

Associated Features

In addition to worry about panic attacks and their consequences, many individuals with panic disorder report constant or intermittent feelings of anxiety that are more broadly related to health and mental health concerns. For example, individuals with panic disorder often anticipate a catastrophic outcome from a mild physical symptom or medication side effect (e.g., thinking that they may have heart disease or that a headache means presence of a brain tumor). Such individuals often are relatively intolerant of medication side effects. In addition, there may be pervasive concerns about abilities to complete daily tasks or withstand daily stressors, excessive use of drugs (e.g., alcohol, prescribed medications or illicit drugs) to control panic attacks, or extreme behaviors aimed at controlling panic attacks (e.g., severe restrictions on food intake or avoidance of specific foods or medications because of concerns about physical symptoms that provoke panic attacks).

Prevalence

In the general population, the 12-month prevalence estimate for panic disorder across the United States and several European countries is about 2%–3% in adults and adolescents. The global lifetime prevalence is estimated at 1.7%, with a 2.7% projected lifetime risk in the World Mental Health Surveys. In the United States, significantly lower prevalence estimates of panic disorder are reported among Latinx, African Americans, Caribbean Blacks, and Asian Americans, compared with non-Latinx Whites. Prevalence estimates of panic disorder in American Indians range from 2.6% to 4.1%. Lower estimates have been reported for Asian, African, and Latin American countries, ranging from 0.1% to 0.8%. Women are more frequently affected than men, at a rate of approximately 2:1. The gender differentiation occurs in adolescence and is already observable before age 14 years. Although panic attacks occur in children, the overall prevalence of panic disorder is low before age 14 years (<0.4%). The rates of panic disorder show a gradual increase during adolescence and possibly following the onset of puberty, and peak during adulthood. The prevalence declines in older individuals (i.e., 1.2% in adults older than age 55, 0.7% in adults older than age 64), possibly reflecting diminishing severity to subclinical levels.

Development and Course

The median age at onset for panic disorder in the United States is 20–24 years, and cross-nationally is 32 years. The mean age at onset is 34.7 years. A small number of cases begin in childhood, and onset after age 55 years is unusual but can occur. The usual course, if the disorder is untreated, is chronic but waxing and waning. Some individuals may have episodic outbreaks with years of remission in between, and others may have continuous severe symptomatology. According to a longitudinal study in the Netherlands, about one-quarter of the individuals with panic disorder experienced recurrence of symptoms within the initial 2-year follow-up period. Only a minority of individuals have full remission without subsequent relapse within a few years. The course of panic disorder typically is complicated by a range of other disorders, in particular other anxiety disorders, depressive disorders, and substance use disorders (see section “Comorbidity” for this disorder). African American adults have been reported to have a more chronic course of panic disorder compared with non-Latinx White adults, possibly because of the enduring impact of racism and discrimination, stigma due to mental illness, and limited access to adequate care.

Although panic disorder is very rare in childhood, first occurrence of “fearful spells” is often dated retrospectively back to childhood. As in adults, panic disorder in adolescents tends to have a chronic course and is frequently comorbid with other anxiety, depressive, and bipolar disorders. To date, no differences in the clinical presentation between adolescents and adults have been found. However, adolescents may be less worried about additional panic attacks than are young adults. Lower prevalence of panic disorder in older adults appears to be attributable to age-related “dampening” of the autonomic nervous system response. Many older individuals with “panicky feelings” are observed to have a “hybrid” of limited-symptom panic attacks and generalized anxiety. Also, older adults tend to attribute their panic attacks to certain stressful situations, such as a medical procedure or social setting. Older individuals may retrospectively endorse explanations for the panic attack (which would preclude the

diagnosis of panic disorder), even if an attack might actually have been unexpected in the moment (and thus qualify as the basis for a panic disorder diagnosis). This may result in under-endorsement of unexpected panic attacks in older individuals. Thus, careful questioning of older adults is required to assess whether panic attacks were expected before entering the situation, so that unexpected panic attacks and the diagnosis of panic disorder are not overlooked.

While the low rate of panic disorder in children could relate to difficulties in symptom reporting, this seems unlikely given that children are capable of reporting intense fear or panic in relation to separation and to phobic objects or phobic situations. Adolescents might be less willing than adults to openly discuss panic attacks. Therefore, clinicians should be aware that unexpected panic attacks do occur in adolescents, much as they do in adults, and be attuned to this possibility when encountering adolescents presenting with episodes of intense fear or distress.

Risk and Prognostic Factors

Temperamental. Negative affectivity (neuroticism) (i.e., proneness to experiencing negative emotions), anxiety sensitivity (i.e., the disposition to believe that symptoms of anxiety are harmful), behavioral inhibition, and harm avoidance are risk factors for the onset of panic attacks and panic disorder. History of "fearful spells" (i.e., limited-symptom attacks that do not meet full criteria for a panic attack) may be a risk factor for later panic attacks and panic disorder, particularly when the first panic experience is appraised as negative. Although separation anxiety in childhood, especially when severe, may precede the later development of panic disorder, it is not a consistent risk factor.

Environmental. Most individuals report identifiable stressors in the months before their first panic attack (e.g., interpersonal stressors and stressors related to physical well-being,

such as negative experiences with illicit or prescription drugs, disease, or death in the family). Furthermore, more chronic life stress is associated with

greater panic disorder severity. Between 10% and 60% of individuals with panic disorder endorse a history of trauma, and stressful life experiences and childhood adversities are associated with more severe panic pathology. Parental overprotection and low emotional warmth are also risk factors for panic disorder. Individuals with few economic resources are more likely to have symptoms that meet criteria for panic disorder. Smoking is a risk factor for panic attacks and panic disorder.

Genetic and physiological. Multiple genes likely confer vulnerability to panic disorder; however, the exact genes, gene products, or functions related to the genetic regions implicated remain unknown. There is an increased risk for panic disorder among offspring of parents with anxiety, depressive, and bipolar disorders.

Individuals with panic disorder display particularly enhanced sensitivity to respiratory stimulation using CO_2-enriched air. Respiratory disturbance, such as asthma, may be associated with panic disorder, in terms of past history, comorbidity, and family history.

Culture-Related Diagnostic Issues

The rate of fears about mental and somatic symptoms of anxiety appears to vary across cultural contexts and may influence the rate of panic attacks and panic disorder. Also, cultural expectations may influence the classification of panic attacks as expected or unexpected. For example, a Vietnamese individual who has a panic attack after walking out into a windy environment (*trúng gió;* "hit by the wind") may attribute the panic attack to exposure to wind as a result of the cultural syndrome that links these two experiences, resulting in classification of the panic attack as expected. Various other cultural concepts of distress are associated with panic disorder, including *ataque de nervios* ("attack of nerves") among Latin Americans and *khyâl* attacks and "soul loss" among Cambodians. *Ataque de nervios* may involve trembling, uncontrollable screaming or crying, aggressive or suicidal behavior, and depersonalization or derealization, which may be experienced longer than the few minutes typical of panic attacks. Some clinical presentations of *ataque de nervios* fulfill criteria for conditions other than panic attack (e.g., functional neurological symptom disorder). These concepts of distress have an impact on the symptoms and

frequency of panic disorder, including the individual's attribution of unexpectedness, as cultural concepts of distress may create fear of certain situations, ranging from interpersonal arguments (associated with *ataque de nervios*), to types of exertion (associated with *khyâl* attacks), to atmospheric wind (associated with *trúng gió* attacks). Clarification of the details of cultural attributions may aid in distinguishing expected and unexpected panic attacks. For more information regarding cultural concepts of distress, refer to the "Culture and Psychiatric Diagnosis" chapter in Section III.

The specific worries about panic attacks or their consequences are likely to vary across ethnoracial groups and cultural contexts (and across different age groups and gender). Among Asian Americans, Hispanic Americans, and African Americans in the United States, panic disorder is associated with reports of ethnic discrimination and racism, after the effect of demographic factors is taken into account. For panic disorder, U.S. community samples of non-Latinx Whites have significantly less functional impairment than African Americans. There are also higher rates of objectively defined severity in non-Latinx Caribbean Blacks with panic disorder, and lower reported rates of panic disorder overall in both African Americans and Caribbean Blacks, suggesting that among U.S. community samples of African descent, panic disorder criteria may be endorsed only when there is substantial severity and impairment. The rate of mental health service use for panic disorder varies across ethnoracial groups.

Sex- and Gender-Related Diagnostic Issues

The rate of panic disorder is nearly twofold higher in women compared with men. Relapse from panic disorder also occurs more frequently in adult women compared with men,

suggesting that women have a more unstable illness course. Gender differences in clinical course are also found among adolescents. Panic disorder has a larger impact on health-related quality of life in women than in men, which may be attributable to greater anxiety sensitivity among some women or greater comorbidity with agoraphobia and depression.

There is some evidence for sexual dimorphism, with high expression of *MAOA*-uVNTR alleles potentially acting as a female-specific risk factor for panic disorder.

Diagnostic Markers

Individuals with panic disorder exhibit an attentional bias to threatening stimuli. Panic attacks may be provoked by agents with disparate mechanisms of action, such as sodium lactate, caffeine, isoproterenol, yohimbine, CO_2, and cholecystokinin, to a much greater extent in individuals with panic disorder than in those without it. There is considerable interest in the relationship between panic disorder and sensitivity to these panic-provoking agents. While none of the data suggest diagnostic utility, data for sensitivity to respiratory stimulation reflect some level of specificity for panic disorder and related conditions, such as separation anxiety disorder. Chronically higher baseline hyperventilation and rate of sighing may occur among individuals with panic disorder. However, none of these laboratory findings are considered diagnostic of panic disorder.

Association With Suicidal Thoughts or Behavior

Panic attacks and a diagnosis of panic disorder in the past 12 months are related to a higher rate of suicidal behavior and suicidal thoughts in the past 12 months even when comorbidity and a history of childhood abuse and other suicide risk factors are taken into account. Approximately 25% of primary care patients with panic disorder report suicidal thoughts. Panic disorder may increase risk for future suicidal behaviors but not deaths.

Epidemiological survey data of panic attack symptoms show that the cognitive symptoms of panic (e.g., derealization) may be associated with suicidal thoughts, whereas physical symptoms (e.g., dizziness, nausea) may be associated with suicidal behaviors.

Functional Consequences of Panic Disorder

Panic disorder is associated with high levels of social, occupational, and physical disability; considerable economic costs; and the highest number of

medical visits among the anxiety disorders, although the effects are strongest with the presence of agoraphobia. Individuals with panic disorder may be frequently absent from work or school for doctor and emergency room visits, which can lead to unemployment or dropping out of school. In older adults, impairment may be seen in caregiving duties or volunteer activities, and panic disorder is related to lower health-related quality of life and greater receipt of emergency department services. Full-symptom panic attacks typically are associated with greater morbidity (e.g., greater health care utilization, more disability, poorer quality of life) than limited-symptom attacks.

Differential Diagnosis

Only limited-symptom panic attacks. Panic disorder should not be diagnosed if full-symptom (unexpected) panic attacks have never been experienced. In the case of only limited-symptom unexpected panic attacks, an other specified anxiety disorder or unspecified anxiety disorder diagnosis should be considered.

Anxiety disorder due to another medical condition. Panic disorder is not diagnosed if the panic attacks are judged to be a direct physiological consequence of another medical condition. Examples of medical conditions that can cause panic attacks include hyperthyroidism, hyperparathyroidism, pheochromocytoma, vestibular dysfunctions, seizure

disorders, and cardiopulmonary conditions (e.g., arrhythmias, supraventricular tachycardia, asthma, chronic obstructive pulmonary disease [COPD]). Appropriate laboratory tests (e.g., serum calcium levels for hyperparathyroidism; Holter monitor for arrhythmias) or physical examinations (e.g., for cardiac conditions) may be helpful in determining the etiological role of another medical condition. Features such as onset after age 45 years or the presence of atypical symptoms during a panic attack (e.g., vertigo, loss of consciousness, loss of bladder or bowel control, slurred speech, amnesia) suggest the possibility that another medical condition or a substance may be causing the panic attack symptoms.

Substance/medication-induced anxiety disorder. Panic disorder is not diagnosed if the panic attacks are judged to be a direct physiological consequence of a substance. Intoxication with central nervous system stimulants (e.g., cocaine, amphetamine-type substances, caffeine) or cannabis and withdrawal from central nervous system depressants (e.g., alcohol, barbiturates) can precipitate a panic attack.

However, if panic attacks continue to occur outside of the context of substance use (e.g., long after the effects of intoxication or withdrawal have ended), a diagnosis of panic disorder should be considered. In addition, because panic disorder may precede substance use in some individuals and may be associated with increased substance use, especially for purposes of self-medication, a detailed history should be taken to determine if the individual had panic attacks prior to excessive substance use. If this is the case, a diagnosis of panic disorder should be considered in addition to a diagnosis of substance use disorder. Features such as onset after age 45 years or the presence of atypical symptoms during a panic attack (e.g., vertigo, loss of consciousness, loss of bladder or bowel control, slurred speech, amnesia) suggest the possibility that another medical condition or a substance may be causing the panic attack symptoms.

Other mental disorders with panic attacks as an associated feature (e.g., other anxiety disorders and psychotic disorders). Panic attacks that occur as a symptom of other anxiety disorders are expected (e.g., triggered by social situations in social anxiety disorder, by phobic objects or situations in specific phobia or agoraphobia, by worry in generalized anxiety disorder, by separation from home or attachment figures in separation anxiety disorder) and thus would not meet criteria for panic disorder. (**Note:** Sometimes an unexpected panic attack is associated with the onset of another anxiety disorder, but then the attacks become expected, whereas panic disorder is characterized by recurrent unexpected panic attacks.) If the panic attacks occur only in response to specific triggers, then only the relevant anxiety disorder is assigned. However, if the individual experiences unexpected panic attacks as well and shows persistent concern and worry or behavioral change because of the attacks, then an additional diagnosis of panic disorder should be considered.

Comorbidity

Panic disorder infrequently occurs in clinical settings in the absence of other psychopathology. In the general population, 80% of individuals with panic disorder had a lifetime comorbid mental diagnosis. The prevalence of panic disorder is elevated in individuals with other disorders, particularly other anxiety disorders (and especially agoraphobia), major depressive disorder, bipolar I and bipolar II disorder, and possibly mild alcohol use disorder. While panic disorder occasionally has an earlier age at onset than the comorbid disorder(s), onset often occurs after the comorbid disorder and may be seen as a severity marker of the comorbid illness.

Reported lifetime rates of comorbidity between major depressive disorder and panic disorder vary widely, ranging from 10% to 65% in individuals with panic disorder. In approximately one-third of individuals with both disorders, the depression precedes the onset of panic disorder. In the remaining two-thirds, depression occurs coincident with or following the onset of panic disorder. A subset of individuals with panic disorder develop a

substance-related disorder, which for some represents an attempt to treat their anxiety with alcohol or medications. Comorbidity with other anxiety disorders and illness anxiety disorder is also common.

Panic disorder is significantly comorbid with numerous general medical symptoms and conditions, including, but not limited to, dizziness, cardiac arrhythmias, hyperthyroidism, asthma, COPD, and irritable bowel syndrome. However, the nature of the association (e.g., cause and effect) between panic disorder and these conditions remains unclear. Although mitral valve prolapse and thyroid disease are more common among individuals with panic disorder than in the general population, the increases in prevalence are not consistent.

Panic Attack Specifier

Note: Symptoms are presented for the purpose of identifying a panic attack; however, panic attack is not a mental disorder and

cannot be coded. Panic attacks can occur in the context of any anxiety disorder as well as other mental disorders (e.g., depressive disorders, posttraumatic stress disorder, substance use disorders) and some medical conditions (e.g., cardiac, respiratory, vestibular, gastrointestinal). When the presence of a panic attack is identified, it should be noted as a specifier (e.g., "posttraumatic stress disorder with panic attacks"). For panic disorder, the presence of panic attack is contained within the criteria for the disorder and panic attack is not used as a specifier.

An abrupt surge of intense fear or intense discomfort that reaches a peak within minutes, and during which time four (or more) of the following symptoms occur:

Note: The abrupt surge can occur from a calm state or an anxious state.

1. 1. Palpitations, pounding heart, or accelerated heart rate.
   2. Sweating.
   3. Trembling or shaking.
   4. Sensations of shortness of breath or smothering.
   5. Feelings of choking.
   6. Chest pain or discomfort.
   7. Nausea or abdominal distress.
   8. Feeling dizzy, unsteady, light-headed, or faint.
   9. Chills or heat sensations.
   10. Paresthesias (numbness or tingling sensations).
   11. Derealization (feelings of unreality) or depersonalization (being detached from oneself).
   12. Fear of losing control or "going crazy."
   13. Fear of dying.

Note: Culture-specific symptoms (e.g., tinnitus, neck soreness, headache, uncontrollable screaming or crying) may be seen. Such symptoms should not count as one of the four required symptoms.

Features

The essential feature of a panic attack is an abrupt surge of intense fear or intense discomfort that reaches a peak within minutes and during which time 4 or more of 13 physical and cognitive symptoms occur. Eleven of these 13 symptoms are physical (e.g., palpitations, sweating), while 2 are cognitive (i.e., fear of losing control or going crazy, fear of dying). “Fear of going crazy” is a colloquialism often used by individuals with panic attacks and is not intended as a pejorative or diagnostic term. The term *within minutes* means that the time to peak intensity is literally only a few minutes. A panic attack can arise from either a calm state or an

anxious state, and time to peak intensity should be assessed independently of any preceding anxiety. That is, the start of the panic attack is the point at which there is an abrupt increase in discomfort rather than the point at which anxiety first developed. Likewise, a panic attack can return to either an anxious state or a calm state and possibly peak again. A panic attack is distinguished from ongoing anxiety by its time to peak intensity, which occurs within minutes; its discrete nature; and its typically greater severity. Attacks that meet all other criteria but have fewer than four physical and/or cognitive symptoms are referred to as *limited-symptom attacks.*

There are two characteristic types of panic attacks: expected and unexpected. *Expected panic attacks* are attacks for which there is an obvious cue or trigger, such as situations in which panic attacks have typically occurred. *Unexpected panic attacks* are those for which there is no obvious cue or trigger at the time of occurrence (e.g., when relaxing or out of sleep [nocturnal panic attack]). The determination of whether panic attacks are expected or unexpected is made by the clinician, who makes this judgment based on a combination of careful questioning as to the sequence of events preceding or leading up to the attack and the individual’s own judgment of whether the attack seemed to occur for no apparent reason. Panic attacks can occur in the context of any mental disorder (e.g., anxiety disorders, depressive disorders, bipolar disorders, eating disorders, obsessive-compulsive and related disorders, personality disorders,

psychotic disorders, substance use disorders) and some medical conditions (e.g., cardiac, respiratory, vestibular, gastrointestinal), with the majority of presentations never meeting criteria for panic disorder. Recurrent unexpected panic attacks are required for a diagnosis of panic disorder.

Associated Features

One type of unexpected panic attack is a *nocturnal panic attack* (i.e., waking from sleep in a state of panic), which differs from panicking after fully waking from sleep.

Prevalence

In the general population, 12-month prevalence estimates for panic attacks in Spain and in the United States range from 9.5% to 11.2% in adults. Twelve-month prevalence estimates do not appear to differ significantly among African Americans, Asian Americans, and Latinx. Approximately 8.5% of American Indians report a lifetime history of panic attacks. Lifetime prevalence rates of panic attacks cross-nationally are 13.2%. Women are more frequently affected than men, although this gender difference is more pronounced for panic disorder. Panic attacks can occur in children but are relatively rare until the age of puberty, when the prevalence rates increase. The prevalence declines in older individuals, possibly reflecting diminishing severity to subclinical levels.

Development and Course

The mean age at onset for panic attacks in the United States is approximately 22–23 years among adults. However, the course of panic attacks is likely influenced by the course of any co-occurring mental disorder(s) and stressful life events. Panic attacks are uncommon, and unexpected panic attacks are rare, in preadolescent children. Adolescents might be less willing than adults to openly discuss panic attacks, even though they present with episodes of intense fear or discomfort. Lower prevalence of panic attacks in older individuals may be related to a weaker autonomic response to emotional states relative to younger individuals. Older individuals may be less inclined to use the word “fear” and more

inclined to use the word “discomfort” to describe panic attacks. Older individuals with “panicky feelings” may have a hybrid of limited-symptom attacks and generalized anxiety. In addition, older individuals tend to attribute panic attacks to certain situations that are stressful (e.g., medical procedures, social settings) and may retrospectively endorse explanations for the panic attack even if it was unexpected in the moment. This may result in under-endorsement of unexpected panic attacks in older individuals.

Risk and Prognostic Factors

Temperamental. Negative affectivity (neuroticism) (i.e., proneness to experiencing negative emotions), anxiety sensitivity (i.e., the disposition to believe that symptoms of anxiety are harmful), behavioral inhibition, and harm avoidance are risk factors for the onset of panic attacks. History of “fearful spells” (i.e., limited-symptom attacks that do not meet full criteria for a panic attack) may be a risk factor for later panic attacks.

Environmental. Smoking is a risk factor for panic attacks. Most individuals report identifiable stressors in the months before their first panic attack (e.g., interpersonal stressors and stressors related to physical well-being, such as negative experiences with illicit or prescription drugs, disease, or death in the family). Separation from parents, overprotective parenting, and parental rejection are risk factors for panic attacks.

Genetic and physiological. Individuals with chronic obstructive pulmonary disease who report low perceptions of control over the disease and negative beliefs about the consequences of unpredictable breathless attacks are more likely to have panic symptoms.

Culture-Related Diagnostic Issues

Cultural interpretations may influence the determination of panic attacks as expected or unexpected. Culture-specific symptoms (e.g., tinnitus, neck soreness, headache, and uncontrollable screaming or crying) may be seen; however, such symptoms should not count as one of the four required

symptoms. Frequency of each of the 13 symptoms varies cross-culturally (e.g., higher rates of paresthesias in African Americans, of dizziness in several Asian groups, and of trembling in non-Latinx Whites). Cultural concepts of distress also influence the cross-cultural presentation of panic attacks, resulting in different symptom profiles across different cultural groups. Examples include *khyâl* (wind) attacks, a Cambodian cultural syndrome involving dizziness, tinnitus, and neck soreness; and *trúng gió* (wind-related) attacks, a Vietnamese cultural syndrome associated with headaches. Cultural explanatory models can heighten the salience of specific panic symptoms. For example, traditional Cambodian views about the abnormal circulation of *khyâl* in the body are associated with the perceived dangerousness of some symptoms (e.g., neck soreness), which can trigger catastrophic cognitions and panic attacks. *Ataque de nervios* (attack of nerves) is a cultural syndrome among Latin Americans that may involve trembling, uncontrollable screaming or crying, aggressive or suicidal behavior, and depersonalization or derealization, and which may be experienced for longer than only a few minutes. Some clinical presentations of *ataque de nervios* fulfill criteria for conditions other than panic attack (e.g., other specified dissociative disorder). Also, cultural expectations may influence the classification of panic attacks as expected or unexpected, as cultural syndromes may create fear of certain situations, ranging from interpersonal arguments (associated with *ataque de nervios*), to types of exertion (associated with *khyâl* attacks), to atmospheric wind (associated with *trúng gió* attacks). Clarification of the details of cultural attributions may aid in distinguishing expected and unexpected panic attacks. For more information about cultural concepts of distress, see the “Culture and Psychiatric Diagnosis” chapter in Section III.

Sex- and Gender-Related Diagnostic Issues

Panic attacks are more common in women than in men. Among those who report panic attacks, women are more likely to endorse symptoms of shortness of breath and nausea but less likely to endorse sweating than are men.

Diagnostic Markers

Physiological recordings of naturally occurring panic attacks in individuals with panic disorder indicate abrupt surges of arousal, usually of heart rate, that reach a peak within

minutes and subside within minutes, and for a proportion of these individuals the panic attack may be preceded by cardiorespiratory instabilities. Panic attacks are characterized by heart rate and tidal volume increases and a drop in pCO_2.

Association With Suicidal Thoughts or Behavior

Panic attacks are related to a higher rate of suicide attempts and suicidal thoughts even when comorbidity and other suicide risk factors are taken into account.

Functional Consequences of Panic Attacks

In the context of co-occurring mental disorders, including anxiety disorders, depressive disorders, bipolar disorder, substance use disorders, psychotic disorders, and personality disorders, panic attacks are associated with increased symptom severity, higher rates of comorbidity, and poorer treatment response. Recurrent panic attacks in particular are associated with increased odds of many mental health diagnoses. Furthermore, more severe panic attacks are associated with a greater likelihood of developing panic disorder and a variety of other mental health conditions, as well as greater persistence of mental illness and impaired functioning. Also, full-symptom panic attacks typically are associated with greater morbidity (e.g., greater health care utilization, more disability, poorer quality of life) than limited-symptom attacks.

Differential Diagnosis

Other paroxysmal episodes (e.g., “anger attacks”). Panic attacks should not be diagnosed if the episodes do not involve the essential feature of an abrupt surge of intense fear or intense discomfort, but rather other emotional states (e.g., anger, grief).

Anxiety disorder due to another medical condition. Medical conditions that can cause or be misdiagnosed as panic attacks include hyperthyroidism, hyperparathyroidism, pheochromocytoma, vestibular dysfunctions, seizure disorders, and cardiopulmonary conditions (e.g., arrhythmias, supraventricular tachycardia, asthma, chronic obstructive pulmonary disease). Appropriate laboratory tests (e.g., serum calcium levels for hyperparathyroidism; Holter monitor for arrhythmias) or physical examinations (e.g., for cardiac conditions) may be helpful in determining the etiological role of another medical condition.

Substance/medication-induced anxiety disorder. Intoxication with central nervous system stimulants (e.g., cocaine, amphetamine-type substances, caffeine) or cannabis and withdrawal from central nervous system depressants (e.g., alcohol, barbiturates) can precipitate a panic attack. A detailed history should be taken to determine if the individual had panic attacks prior to excessive substance use. Features such as onset after age 45 years or the presence of atypical symptoms during a panic attack (e.g., vertigo, loss of consciousness, loss of bladder or bowel control, slurred speech, or amnesia) suggest the possibility that a medical condition or a substance may be causing the panic attack symptoms.

Panic disorder. Repeated unexpected panic attacks are required but are not sufficient for the diagnosis of panic disorder (i.e., full diagnostic criteria for panic disorder must be met).

Comorbidity

Panic attacks are associated with increased likelihood of various comorbid mental disorders, including anxiety disorders, depressive disorders, bipolar disorders, impulse-control disorders, and substance use disorders. Panic attacks are associated with increased likelihood of later developing anxiety disorders, depressive disorders, bipolar disorders, alcohol use disorder, and possibly other disorders.

Diagnostic Criteria F40.00

A. Marked fear or anxiety about two (or more) of the following five situations:
   1. Using public transportation (e.g., automobiles, buses, trains, ships, planes).
   2. Being in open spaces (e.g., parking lots, marketplaces, bridges).
   3. Being in enclosed places (e.g., shops, theaters, cinemas).
   4. Standing in line or being in a crowd.
   5. Being outside of the home alone.

B. The individual fears or avoids these situations because of thoughts that escape might be difficult or help might not be available in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms (e.g., fear of falling in the elderly; fear of incontinence).

C. The agoraphobic situations almost always provoke fear or anxiety.

D. The agoraphobic situations are actively avoided, require the presence of a companion, or are endured with intense fear or anxiety.

E. The fear or anxiety is out of proportion to the actual danger posed by the agoraphobic situations and to the sociocultural context.

F. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.

G. The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

H. If another medical condition (e.g., inflammatory bowel disease, Parkinson’s disease) is present, the fear, anxiety, or avoidance is clearly excessive.

I. The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder—for example, the symptoms are not confined to specific phobia, situational type; do not involve only social situations (as in social anxiety disorder); and are not related exclusively to obsessions (as in obsessive-compulsive disorder), perceived defects or flaws in physical appearance (as in body dysmorphic disorder), reminders of traumatic events (as in posttraumatic stress disorder), or fear of separation (as in separation anxiety disorder).

Note: Agoraphobia is diagnosed irrespective of the presence of panic disorder. If an individual's presentation meets criteria for panic disorder and agoraphobia, both diagnoses should be assigned.

Diagnostic Features

The essential feature of agoraphobia is marked fear or anxiety triggered by the real or anticipated exposure to a wide range of situations (Criterion A). The diagnosis requires endorsement of symptoms occurring in at least two of the following five situations: 1) using public transportation, such as automobiles, buses, trains, ships, or planes; 2) being in open spaces, such as parking lots, marketplaces, or bridges; 3) being in enclosed spaces, such as shops, theaters, or cinemas; 4) standing in line or being in a crowd; or 5) being outside of the home alone. The examples for each situation are not exhaustive; other situations may be feared. When experiencing fear and anxiety cued by such situations, individuals typically experience thoughts that something terrible might happen (Criterion B). Individuals frequently believe that escape from such situations might be difficult (e.g., "can't get out of here") or that help might be unavailable (e.g., "there is nobody to help me") when panic-like symptoms or other incapacitating or embarrassing symptoms occur. "Panic-like symptoms" refer to any of the 13 symptoms included in the criteria for panic attack, such as dizziness, faintness, and fear of dying. "Other incapacitating or embarrassing symptoms" include

symptoms such as vomiting and inflammatory bowel symptoms, as well as, in older adults, a fear of falling or, in children, a sense of disorientation and getting lost.

The amount of fear experienced may vary with proximity to the feared situation and may occur in anticipation of or in the actual presence of the agoraphobic situation. Also, the fear or anxiety may take the form of a full- or limited-symptom panic attack (i.e., an expected panic attack). Fear or anxiety is evoked nearly every time the individual comes into contact with the feared situation (Criterion C). Thus, an individual who becomes anxious only occasionally in an agoraphobic situation (e.g., becomes anxious when standing in line on only one out of every five occasions) would not be diagnosed with agoraphobia. The individual actively avoids the situation, requires the presence of a companion, or, if he or she either is unable or decides not to avoid it, the situation evokes intense fear or anxiety (Criterion D). *Active avoidance* means the individual is currently behaving in ways that are intentionally designed to prevent or minimize contact with agoraphobic situations. Avoidance can be behavioral (e.g., changing daily routines, choosing a job nearby to avoid using public transportation, arranging for food delivery to avoid entering shops and supermarkets) as well as cognitive (e.g., using distraction to get through agoraphobic situations) in nature. The avoidance can become so severe that the individual is completely homebound. Often, an individual is better able to confront a feared situation when accompanied by a companion, such as a partner, friend, or health professional. Also, the individual may employ safety behaviors (e.g., sitting near exits when taking public transportation or at the movies) to better endure such situations.

The fear, anxiety, or avoidance must be out of proportion to the actual danger posed by the agoraphobic situations and to the sociocultural context (Criterion E). Differentiating disproportionate, clinically significant agoraphobic fears from reasonable fears (e.g., not wanting to leave the house during a bad storm) or from situations that are deemed dangerous (e.g., walking in a parking lot or using public transportation in a high-crime area) is important for a number of reasons. First, what constitutes avoidance may be difficult to judge across cultures and sociocultural contexts (e.g., it

is socioculturally appropriate for orthodox Muslim women in certain parts of the world to avoid leaving the house alone, and thus such avoidance would not be considered indicative of agoraphobia). Second, older adults are likely to overattribute their fears to age-related constraints and are less likely to judge their fears as being out of proportion to the actual risk. Third, individuals with agoraphobia are likely to overestimate danger in relation to panic-like or other bodily symptoms. Agoraphobia should be diagnosed only if the fear, anxiety, or avoidance is persistent (Criterion F) and if it causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion G). The duration of "typically lasting for 6 months or more" is meant to exclude individuals with short-lived, transient problems.

Associated Features

In its most severe forms, agoraphobia can cause individuals to become completely homebound, unable to leave their home and dependent on others for services or assistance to provide even for basic needs. Demoralization and depressive symptoms, as well as abuse of alcohol and sedative medication as inappropriate self-medication strategies, are common.

Prevalence

Every year approximately 1%–1.7% of adolescents and adults worldwide have symptoms that meet diagnostic criteria for agoraphobia. Women are twice as likely as men to experience agoraphobia. Agoraphobia may occur in childhood, but incidence peaks in late adolescence and early adulthood. Studies have shown the 12-month prevalence in individuals living in the United States who are older than 65 years is 0.4% and 0.5% among individuals in Europe and North America older than 55 years. Approximately 0.2%–0.8% of

the adult population in various countries has a past 12-month diagnosis of agoraphobia without panic disorder.

Development and Course

The percentage of individuals with agoraphobia reporting panic attacks or panic disorder preceding the onset of agoraphobia ranges from 30% in community samples to more than 50% in clinical samples.

In two-thirds of all cases of agoraphobia, initial onset is before age 35 years, with 21 years the mean age at onset, although age at onset of agoraphobia without preceding panic attacks or panic disorder is 25–29 years. First onset in childhood is rare. There is a substantial incidence risk in adolescence and early adulthood, with indications for a second high incidence risk phase after age 40 years. Approximately 10% of older adults with agoraphobia reported their first episode of agoraphobia occurring after age 65.

The course of agoraphobia is typically persistent and chronic. Complete remission is rare (10%), unless the agoraphobia is treated. Individuals with comorbid panic disorder and agoraphobia are more likely to experience recurrence of symptoms after a period of remission if they had an earlier age at onset (< 20 years old). With more severe agoraphobia, rates of full remission decrease, whereas rates of relapse and chronicity increase. Approximately 36% of individuals with agoraphobia who achieve remission eventually experience relapse. A range of other disorders, in particular other anxiety disorders, depressive disorders, substance use disorders, and personality disorders, may complicate the course of agoraphobia. The long-term course and outcome of agoraphobia are associated with substantially elevated risk of secondary major depressive disorder, persistent depressive disorder, and substance use disorders.

The clinical features of agoraphobia are relatively consistent across the life span, although the type of agoraphobic situations triggering fear, anxiety, or avoidance, as well as the type of cognitions, may vary. For example, in children, being outside of the home alone is the most frequent situation feared, whereas in older adults, being in shops, standing in line, and being in open spaces are most often feared. Also, cognitions often pertain to becoming lost (in children), to experiencing panic-like symptoms (in adults), to falling (in older adults).

The apparent low prevalence of agoraphobia in children could reflect difficulties in symptom reporting, and thus assessments in young children

may require solicitation of information from multiple sources, including parents or teachers. Adolescents, particularly boys, may be less willing than adults to openly discuss agoraphobic fears and avoidance; however, agoraphobia can occur before adulthood and should be assessed in children and adolescents. In older adults, comorbid somatic symptom disorder, having medical complications, and motor disturbances (e.g., a sense of falling) are frequently mentioned by individuals as the reason for their fear and avoidance. In these instances, care is to be taken in evaluating whether the fear and avoidance are out of proportion to the real danger involved.

Risk and Prognostic Factors

Temperamental. Behavioral inhibition, negative affectivity (neuroticism), anxiety sensitivity, and trait anxiety are closely associated with agoraphobia but are relevant to most anxiety disorders (specific phobia, social anxiety disorder, panic disorder, generalized anxiety disorder). Anxiety sensitivity (the disposition to believe that symptoms of anxiety are harmful) is also characteristic of individuals with agoraphobia.

Environmental. Negative events in childhood (e.g., separation, death of parent) and other stressful events, such as being attacked or mugged, are associated with the onset of agoraphobia. Furthermore, individuals with agoraphobia describe the family climate and child-rearing behavior as being characterized by reduced warmth and increased overprotection.

Genetic and physiological. Heritability for agoraphobia is 61%. Of the various phobias, agoraphobia has the strongest and most specific association with the genetic factor that represents proneness to phobias. Family history of anxiety disorders is associated with an earlier age at onset of agoraphobia (< 27 years old), and family history of panic disorder in particular is associated with agoraphobia.

Sex- and Gender-Related Diagnostic Issues

Women have different patterns of comorbid disorders than men. Consistent with gender differences in the prevalence of mental disorders, men have

higher rates of comorbid substance use disorders.

Association With Suicidal Thoughts or Behavior

Approximately 15% of individuals with agoraphobia report suicidal thoughts or behavior. For individuals with panic disorder, symptoms of agoraphobia may be a risk factor for suicidal thoughts.

Functional Consequences of Agoraphobia

Like most other anxiety disorders, agoraphobia is associated with considerable impairment and disability in terms of role functioning, work productivity, and disability days. Agoraphobia severity is a strong determinant of the degree of disability, irrespective of the presence of comorbid panic disorder, panic attacks, and other comorbid conditions. Individuals with agoraphobia can be completely homebound or unable to work. Individuals with panic disorder with agoraphobia who have an early course of onset (< age 20 years) are less likely to be married.

Differential Diagnosis

Specific phobia, situational type. Differentiating agoraphobia from situational specific phobia can be challenging in some cases, because these conditions share several symptom characteristics and criteria. Specific phobia, situational type, should be diagnosed versus agoraphobia if the fear, anxiety, or avoidance is limited to one of the agoraphobic situations. Requiring fears from two or more of the agoraphobic situations is a robust means for differentiating agoraphobia from specific phobias, particularly the situational subtype. Additional differentiating features include the content of the individual's cognitions. Thus, if the situation is feared for reasons other than panic-like symptoms or other incapacitating or embarrassing symptoms (e.g., fears of being directly harmed by the situation itself, such as fear of the plane crashing for individuals who fear flying), then a diagnosis of specific phobia may be more appropriate.

Separation anxiety disorder. Separation anxiety disorder can be best differentiated from agoraphobia by examining the individual's cognitions. In separation anxiety disorder, the thoughts are about detachment from

significant others and the home environment (i.e., parents or other attachment figures), whereas in agoraphobia the focus is on panic-like symptoms or other incapacitating or embarrassing symptoms in the feared situations.

Social anxiety disorder. Agoraphobia should be differentiated from social anxiety disorder based primarily on the situational clusters that trigger fear, anxiety, or avoidance and the individual's cognitions. In social anxiety disorder, the focus is on fear of being negatively evaluated.

Panic disorder. When criteria for panic disorder are met, agoraphobia should not be diagnosed if the avoidance behaviors associated with the panic attacks do not extend to avoidance of two or more agoraphobic situations.

Acute stress disorder and posttraumatic stress disorder. Acute stress disorder and posttraumatic stress disorder (PTSD) can be differentiated from agoraphobia by examining whether the fear, anxiety, or avoidance is related only to situations that remind the individual of a traumatic event. If the fear, anxiety, or avoidance is restricted to trauma reminders, and if the avoidance behavior does not extend to two or more agoraphobic situations, then a diagnosis of agoraphobia is not warranted.

Major depressive disorder. In major depressive disorder, the individual may avoid leaving home because of apathy, loss of energy, low self-esteem, and anhedonia. If the avoidance is unrelated to fears of panic-like or other incapacitating or embarrassing symptoms, then agoraphobia should not be diagnosed.

Avoidance related to other medical conditions. Individuals with certain medical conditions may avoid situations because of realistic concerns about being incapacitated (e.g., fainting in an individual with transient ischemic attacks) or being embarrassed (e.g., diarrhea in an individual with Crohn's disease). The diagnosis of agoraphobia should be given only when the fear or avoidance is clearly in excess of that usually associated with these medical conditions.

Comorbidity

About 90% of individuals with agoraphobia also have other mental disorders. The most frequent additional diagnoses are other anxiety disorders (e.g., specific phobias, panic disorder, social anxiety disorder), depressive disorders (major depressive disorder), PTSD, and alcohol use disorder. Whereas other anxiety disorders (e.g., separation anxiety disorder, specific phobias, panic disorder) frequently precede onset of agoraphobia, depressive disorders and substance use disorders typically occur secondary to agoraphobia. In some individuals, a substance use disorder precedes agoraphobia. Individuals with comorbid agoraphobia and major depressive disorder tend to have a more treatment-resistant course of agoraphobia relative to individuals with agoraphobia alone.

Generalized Anxiety Disorder

Diagnostic Criteria F41.1

A. Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance).
B. The individual finds it difficult to control the worry.
C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms having been present for more days than not for the past 6 months):

   Note: Only one item is required in children.

   1. Restlessness or feeling keyed up or on edge.
   2. Being easily fatigued.
   3. Difficulty concentrating or mind going blank.
   4. Irritability.
   5. Muscle tension.

6. Sleep disturbance (difficulty falling or staying asleep, or restless, unsatisfying sleep).

D. The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism).

F. The disturbance is not better explained by another mental disorder (e.g., anxiety or worry about having panic attacks in panic disorder, negative evaluation in social anxiety disorder, contamination or other obsessions in obsessive-compulsive disorder, separation from attachment figures in separation anxiety disorder, reminders of traumatic events in posttraumatic stress disorder, gaining weight in anorexia nervosa, physical complaints in somatic symptom disorder, perceived appearance flaws in body dysmorphic disorder, having a serious illness in illness anxiety disorder, or the content of delusional beliefs in schizophrenia or delusional disorder).

Diagnostic Features

The essential feature of generalized anxiety disorder is excessive anxiety and worry (apprehensive expectation) about a number of events or activities. The intensity, duration, or frequency of the anxiety and worry is out of proportion to the actual likelihood or impact of the anticipated event. The individual finds it difficult to control the worry and to keep worrisome thoughts from interfering with attention to tasks at hand. Adults with generalized anxiety disorder often worry about everyday, routine life circumstances, such as possible job responsibilities, health and finances, the health of family members, misfortune to their children, or minor matters

(e.g., doing household chores or being late for appointments). Children with generalized anxiety disorder tend to worry excessively about their competence or the quality of their performance. During the course of the disorder, the focus of worry may shift from one concern to another.

Several features distinguish generalized anxiety disorder from nonpathological anxiety. First, the worries associated with generalized anxiety disorder are excessive and typically interfere significantly with psychosocial functioning, whereas the worries of everyday life are not excessive and are perceived as more manageable and may be put off when more pressing matters arise. Second, the worries associated with generalized anxiety disorder are more pervasive, pronounced, and distressing; have longer duration; and frequently occur without precipitants. The greater the range of life circumstances about which a person worries (e.g., finances, children's safety, job performance), the more likely his or her symptoms are to meet criteria for generalized anxiety disorder. Third, everyday worries are much less likely to be accompanied by physical symptoms (e.g., restlessness or feeling keyed up or on edge). Individuals with generalized anxiety disorder report subjective distress as a result of constant worry and related impairment in social, occupational, or other important areas of functioning.

The anxiety and worry are accompanied by at least three of the following additional symptoms: restlessness or feeling keyed up or on edge, being easily fatigued, difficulty concentrating or mind going blank, irritability, muscle tension, and disturbed sleep, although only one additional symptom is required in children.

Associated Features

Associated with muscle tension, there may be trembling, twitching, feeling shaky, and muscle aches or soreness. Many individuals with generalized anxiety disorder also experience somatic symptoms (e.g., sweating, nausea, diarrhea) and an exaggerated startle response. Symptoms of autonomic hyperarousal (e.g., accelerated heart rate, shortness of breath, dizziness) are less prominent in generalized anxiety disorder than in other anxiety disorders, such as panic disorder. Other conditions that may be associated

with stress (e.g., irritable bowel syndrome, headaches) frequently accompany generalized anxiety disorder.

Prevalence

The 12-month prevalence of generalized anxiety disorder is 0.9% among adolescents and 2.9% among adults in the general community of the United States. The mean 12-month

prevalence for the disorder around the world is 1.3%, with a range of 0.2% to 4.3%. The lifetime morbid risk in the United States is 9.0%. Women and adolescent girls are at least twice as likely as men and adolescent boys to experience generalized anxiety disorder. The 12-month prevalence in older adults including individuals age 75 years and older ranges from 2.8% to 3.1% in the United States, Israel, and European countries.

Individuals of European descent tend to have symptoms that meet criteria for generalized anxiety disorder more frequently than do individuals of Asian and African descent. Furthermore, individuals from high-income countries are more likely than individuals from low- and middle-income countries to report that they have experienced symptoms that meet criteria for generalized anxiety disorder in their lifetime.

Development and Course

Many individuals with generalized anxiety disorder report that they have felt anxious and nervous all their lives. The mean age at onset for generalized anxiety disorder in North America is 35 years, later than that for the other anxiety disorders; the disorder rarely occurs prior to adolescence. However, age at onset is spread over a very broad range and tends to be older in lower-income countries worldwide. The symptoms of excessive worry and anxiety may occur early in life but are then manifested as an anxious temperament. Generalized anxiety disorder symptoms tend to be chronic and wax and wane across the life span, fluctuating between syndromal and subsyndromal forms of the disorder. Course is more

persistent in lower-income countries, but impairment tends to be higher in high-income countries. Rates of full remission are very low.

The earlier in life individuals have symptoms that meet criteria for generalized anxiety disorder, the more comorbidity and impairment they tend to have. Younger adults experience greater severity of symptoms than do older adults.

The clinical expression of generalized anxiety disorder is relatively consistent across the life span. The primary difference across age groups is in the content of the individual's worry; thus, the content of an individual's worry tends to be age appropriate.

In children and adolescents with generalized anxiety disorder, the anxieties and worries often concern the quality of their performance or competence at school or in sporting events, even when their performance is not being evaluated by others. There may be excessive concerns about punctuality. They may also worry about catastrophic events, such as earthquakes or nuclear war. Children with the disorder may be overly conforming, perfectionistic, and unsure of themselves and may tend to redo tasks because of excessive dissatisfaction with less-than-perfect performance. They may be overzealous in seeking reassurance and approval and require excessive reassurance about their performance and other things they are worried about.

In the elderly, the advent of chronic physical disease can be a potent issue for excessive worry. In the frail elderly, worries about safety—and especially about falling—may limit activities.

Risk and Prognostic Factors

Temperamental. Behavioral inhibition, negative affectivity (neuroticism), harm avoidance, reward dependence, and attentional bias to threat have been associated with generalized anxiety disorder.

Environmental. Childhood adversities and parenting practices (e.g., overprotection, overcontrol, reinforcement of avoidance) have been associated with generalized anxiety disorder.

Genetic and physiological. One-third of the risk of experiencing generalized anxiety disorder is genetic, and these genetic factors overlap with the risk of

negative affectivity (neuroticism) and are shared with other anxiety and mood disorders, particularly major depressive disorder.

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Culture-Related Diagnostic Issues

There is considerable cultural variation in the expression of generalized anxiety disorder. For example, in some cultural contexts, somatic symptoms predominate in the expression of the disorder, whereas in other cultural contexts cognitive symptoms tend to predominate. This difference may be more evident on initial presentation than subsequently, as more symptoms are reported over time. There is no information as to whether the propensity for excessive worrying is related to cultural background, although the topic being worried about can be culturally specific. It is important to consider the social and cultural context when evaluating whether worries about certain situations are excessive. In the United States, higher prevalence is associated with exposure to racism and ethnic discrimination and, for some ethnoracial groups, with being born in the United States.

Sex- and Gender-Related Diagnostic Issues

In clinical settings, generalized anxiety disorder is diagnosed somewhat more frequently in women than in men (about 55%–60% of those presenting with the disorder are women). In epidemiological studies, approximately two-thirds are women. Women and men who experience generalized anxiety disorder appear to have similar symptoms but demonstrate different patterns of comorbidity consistent with gender differences in the prevalence of disorders. In women, comorbidity is largely confined to the anxiety disorders and unipolar depression, whereas in men, comorbidity is more likely to extend to the substance use disorders as well.

Association With Suicidal Thoughts or Behavior

Generalized anxiety disorder is associated with increased suicidal thoughts and behavior, even after adjustment for comorbid disorders and stressful life events. Psychological autopsy studies show that generalized anxiety disorder is the most frequent anxiety disorder diagnosed in suicides. Both

subthreshold and threshold generalized anxiety disorder occurring in the past year may be associated with suicidal thoughts.

Functional Consequences of Generalized Anxiety Disorder

Excessive worrying impairs the individual's capacity to do things quickly and efficiently, whether at home or at work. The worrying takes time and energy; the associated symptoms of muscle tension and feeling keyed up or on edge, tiredness, difficulty concentrating, and disturbed sleep contribute to the impairment. Importantly the excessive worrying may impair the ability of individuals with generalized anxiety disorder to encourage confidence in their children.

Generalized anxiety disorder is associated with significant disability and distress that is independent of comorbid disorders, and most non-institutionalized adults with the disorder are moderately to seriously disabled. Generalized anxiety disorder accounts for 110 million disability days per annum in the U.S. population. Generalized anxiety disorder is also linked to decreased work performance, increased medical resource use, and increased risk for coronary morbidity.

Differential Diagnosis

Anxiety disorder due to another medical condition. The diagnosis of anxiety disorder due to another medical condition should be assigned if the individual's anxiety and worry are judged, based on history, laboratory findings, or physical examination, to be a physiological effect of another specific medical condition (e.g., pheochromocytoma, hyperthyroidism).

Substance/medication-induced anxiety disorder. A substance/medication-induced anxiety disorder is distinguished from generalized anxiety disorder by the fact that a substance or medication (e.g., a drug of abuse, exposure to a toxin) is judged to be etiologically related to the anxiety. For example, severe anxiety that occurs only in the context of heavy coffee consumption would be diagnosed as caffeine-induced anxiety disorder.

Social anxiety disorder. Individuals with social anxiety disorder often have anticipatory anxiety that is focused on upcoming social situations in which they must perform or be evaluated by others, whereas individuals with generalized anxiety disorder worry, whether or not they are being evaluated.

Separation anxiety disorder. Individuals with separation anxiety disorder worry excessively only about separation from attachment figures, whereas individuals with generalized anxiety disorder may worry about separation but present other excessive worry concerns as well.

Panic disorder. Panic attacks that are triggered by worry in generalized anxiety disorder would not qualify for panic disorder. However, if the individual experiences unexpected panic attacks as well and shows persistent concern and worry or behavioral change because of the attacks, then an additional diagnosis of panic disorder should be considered.

Illness anxiety disorder and somatic symptom disorder. Individuals with generalized anxiety disorder worry about multiple events, situations, or activities, only one of which may involve their health. If the individual's only fear is his or her own illness, then illness anxiety disorder should be diagnosed. Worry focusing on somatic symptoms is characteristic for somatic symptom disorder.

Obsessive-compulsive disorder. Several features distinguish the excessive worry of generalized anxiety disorder from the obsessional thoughts of obsessive-compulsive disorder. In generalized anxiety disorder the focus of the worry is about forthcoming problems, and it is the excessiveness of the worry about future events that is abnormal. In obsessive-compulsive disorder, the obsessions are inappropriate ideas that take the form of intrusive and unwanted thoughts, urges, or images.

Posttraumatic stress disorder and adjustment disorders. Anxiety is invariably present in posttraumatic stress disorder. Generalized anxiety disorder is not diagnosed if the anxiety and worry are better explained by symptoms of posttraumatic stress disorder. Although anxiety may manifest in adjustment disorder, this residual category should be used only when the criteria are not met for any other mental disorder (including generalized anxiety disorder). Moreover, in adjustment disorders, the anxiety occurs in response to an identifiable stressor within 3 months of the onset of the stressor and does

not persist for more than 6 months after the termination of the stressor or its consequences.

Depressive, bipolar, and psychotic disorders. Although generalized anxiety/worry is a common associated feature of depressive, bipolar, and psychotic disorders, generalized anxiety disorder may be diagnosed comorbidly if the anxiety/worry is sufficiently severe to warrant clinical attention.

Comorbidity

Individuals whose presentation meets criteria for generalized anxiety disorder are likely to have met, or currently meet, criteria for other anxiety and unipolar depressive disorders. The negative affectivity (neuroticism) or emotional liability that underpins this pattern of comorbidity is associated with temperamental antecedents and genetic and environmental risk factors shared between these disorders, although independent pathways are also possible. Comorbidity with substance use, conduct, psychotic, neurodevelopmental, and neurocognitive disorders is less common.

Substance/Medication-Induced Anxiety Disorder

Diagnostic Criteria

A. Panic attacks or anxiety is predominant in the clinical picture.
B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):
    1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication.

2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by an anxiety disorder that is not substance/medication-induced. Such evidence of an independent anxiety disorder could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced anxiety disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance/medication]-induced anxiety disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given. If a mild substance use disorder is comorbid with the substance-induced anxiety disorder, the 4th position character is “1,” and the clinician should record “mild [substance] use disorder” before the substance-induced anxiety disorder (e.g., “mild cocaine use disorder with cocaine-induced anxiety disorder”). If a moderate or severe substance use disorder is

comorbid with the substance-induced anxiety disorder, the 4th position character is “2,” and the clinician should record “moderate [substance] use disorder” or “severe [substance] use disorder,” depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is “9,” and the clinician should record only the substance-induced anxiety disorder.

	ICD-10-CM		
	With mild use disorder	**With moderate or severe use disorder**	**Without use disorder**
Alcohol	F10.180	F10.280	F10.980
Caffeine	NA	NA	F15.980
Cannabis	F12.180	F12.280	F12.980
Phencyclidine	F16.180	F16.280	F16.980
Other hallucinogen	F16.180	F16.280	F16.980
Inhalant	F18.180	F18.280	F18.980
Opioid	F11.188	F11.288	F11.988
Sedative, hypnotic, or anxiolytic	F13.180	F13.280	F13.980
Amphetamine-type substance (or other stimulant)	F15.180	F15.280	F15.980
Cocaine	F14.180	F14.280	F14.980
Other (or unknown) substance	F19.180	F19.280	F19.980

Specify (see Table 1 in the chapter “Substance-Related and Addictive Disorders,” which indicates whether “with onset during intoxication” and/or “with onset during withdrawal” applies to a given substance class; or *specify* “with onset after medication use”):

With onset during intoxication: If criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: If symptoms developed at initiation of medication, with a change in use of medication, or during withdrawal of medication.

Recording Procedures

The name of the substance/medication-induced anxiety disorder begins with the specific substance (e.g., cocaine, salbutamol) that is presumed to be causing the anxiety symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., salbutamol), the code for "other (or unknown) substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the same code should also be used.

To record the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by "with substance/medication-induced anxiety disorder" (incorporating the name of the specific etiological substance/medication), followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal, with onset after medication use). For example, in the case of anxiety symptoms occurring during withdrawal in a man with a severe lorazepam use disorder, the diagnosis is F13.280 severe lorazepam use disorder with lorazepam-induced anxiety disorder, with onset during withdrawal. A separate diagnosis of the comorbid severe lorazepam use disorder is not given. If the substance-induced anxiety disorder occurs without a comorbid substance use disorder (e.g., after a one-time

heavy use of the substance), no accompanying substance use disorder is noted (e.g., F16.980 psilocybin-induced anxiety disorder, with onset during intoxication). When more than one substance is judged to play a significant

role in the development of anxiety symptoms, each should be listed separately (e.g., F15.280 severe methylphenidate use disorder with methylphenidate-induced anxiety disorder, with onset during intoxication; F19.980 salbutamol-induced anxiety disorder, with onset after medication use).

Diagnostic Features

The essential features of substance/medication-induced anxiety disorder are prominent symptoms of panic or anxiety (Criterion A) that are judged to be due to the effects of a substance (e.g., a drug of abuse, a medication, or a toxin exposure). The panic or anxiety symptoms must have developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication, and the substances or medications must be capable of producing the symptoms (Criterion B2). Substance/medication-induced anxiety disorder due to a prescribed treatment for a mental disorder or another medical condition must have its onset while the individual is receiving the medication (or during withdrawal, if a withdrawal is associated with the medication). Once the treatment is discontinued, the panic or anxiety symptoms will usually improve or remit within days to several weeks to a month (depending on the half-life of the substance/medication and the presence of withdrawal). The diagnosis of substance/medication-induced anxiety disorder should not be given if the onset of the panic or anxiety symptoms precedes the substance/medication intoxication or withdrawal, or if the symptoms persist for a substantial period of time (i.e., usually longer than 1 month) from the time of severe intoxication or withdrawal. If the panic or anxiety symptoms persist for substantial periods of time, other causes for the symptoms should be considered.

The substance/medication-induced anxiety disorder diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A are predominant in the clinical picture and are sufficiently severe to warrant independent clinical attention.

Associated Features

Panic or anxiety can occur in association with intoxication with the following classes of substances: alcohol, caffeine, cannabis, phencyclidine, other hallucinogens, inhalants, stimulants (including cocaine), and other (or unknown) substances. Panic or anxiety can occur in association with withdrawal from the following classes of substances: alcohol; opioids; sedatives, hypnotics, and anxiolytics; stimulants (including cocaine); and other (or unknown) substances. Some medications that evoke anxiety symptoms include anesthetics and analgesics, sympathomimetics or other bronchodilators, anticholinergics, insulin, thyroid preparations, oral contraceptives, antihistamines, antiparkinsonian medications, corticosteroids, antihypertensive and cardiovascular medications, anticonvulsants, lithium carbonate, antipsychotic medications, and antidepressant medications. Heavy metals and toxins (e.g., organophosphate insecticide, nerve gases, carbon monoxide, CO_2, volatile substances such as gasoline and paint) may also cause panic or anxiety symptoms.

Prevalence

The prevalence of substance/medication-induced anxiety disorder is not clear. General population data suggest that it may be rare, with a 12-month prevalence in the United States of approximately 0.002%. However, in clinical populations, the prevalence is likely to be higher.

Diagnostic Markers

Laboratory assessments (e.g., urine toxicology) may be useful to measure substance intoxication as part of an assessment for substance/medication-induced anxiety disorder.

Differential Diagnosis

Substance intoxication and substance withdrawal. Anxiety symptoms commonly occur in substance intoxication and substance withdrawal. The diagnosis of the substance-specific intoxication or substance-specific withdrawal will usually suffice to categorize the symptom presentation. A

diagnosis of substance/medication-induced anxiety disorder either with onset during intoxication or with onset during withdrawal should be made instead of a diagnosis of substance intoxication or substance withdrawal when the panic or anxiety symptoms are predominant in the clinical picture and are sufficiently severe to warrant clinical attention. For example, panic or anxiety symptoms are characteristic of alcohol withdrawal.

Independent anxiety disorder (i.e., not induced by a substance/medication). An independent anxiety disorder co-occurring with substance/medication use is distinguished from a substance/medication-induced anxiety disorder by the fact that even though a substance/medication may be taken in high enough amounts to be possibly etiologically related to the anxiety symptoms, the anxiety symptoms are observed at times other than during substance/medication use (i.e., preceding the onset of substance/medication use or persisting for a substantial period of time after substance intoxication, substance withdrawal, or medication use) and would warrant the diagnosis of an independent anxiety disorder.

Delirium. If panic or anxiety symptoms occur exclusively during the course of delirium, they are considered to be an associated feature of the delirium and are not diagnosed separately.

Anxiety disorder due to another medical condition. If the panic or anxiety symptoms are attributed to the physiological consequences of another medical condition (i.e., rather than to the medication taken for the medical condition), anxiety disorder due to another medical condition should be diagnosed. The history often provides the basis for such a judgment. At times, a change in the treatment for the other medical condition (e.g., medication substitution or discontinuation) may be needed to determine whether the medication is the causative agent (in which case the symptoms may be better explained by substance/medication-induced anxiety disorder). If the disturbance is attributable to both another medical condition and substance use, both diagnoses (i.e., anxiety disorder due to another medical condition and substance/medication-induced anxiety disorder) may be given. When there is insufficient evidence to determine whether the panic or anxiety symptoms are attributable to a substance/medication or to another medical condition or are primary (i.e., not attributable to either a

substance or another medical condition), a diagnosis of other specified or unspecified anxiety disorder would be indicated.

Anxiety Disorder Due to Another Medical Condition

Diagnostic Criteria	**F06.4**

A. Panic attacks or anxiety is predominant in the clinical picture.
B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.
C. The disturbance is not better explained by another mental disorder.
D. The disturbance does not occur exclusively during the course of a delirium.
E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Coding note: Include the name of the other medical condition within the name of the mental disorder (e.g., F06.4 anxiety disorder due to pheochromocytoma). The other

medical condition should be coded and listed separately immediately before the anxiety disorder due to the medical condition (e.g., D35.00 pheochromocytoma; F06.4 anxiety disorder due to pheochromocytoma).

Diagnostic Features

The essential feature of anxiety disorder due to another medical condition is clinically significant anxiety that is judged to be best explained as a physiological effect of another medical condition. Symptoms can include prominent anxiety symptoms or panic attacks (Criterion A). The judgment that the symptoms are best explained by the associated physical condition must be based on evidence from the history, physical examination, or laboratory findings (Criterion B). Additionally, it must be judged that the symptoms are not better accounted for by another mental disorder (Criterion C)—in particular, adjustment disorder with anxiety, in which the stressor is the medical condition. In this case, an individual with adjustment disorder is especially distressed about the meaning or the consequences of the associated medical condition. By contrast, there is often a prominent physical component to the anxiety (e.g., shortness of breath) when the anxiety is due to another medical condition. The diagnosis is not made if the anxiety symptoms occur only during the course of a delirium (Criterion D). The anxiety symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E).

In determining whether the anxiety symptoms are attributable to another medical condition, the clinician must first establish the presence of the medical condition. Furthermore, it must be established that anxiety symptoms can be etiologically related to the medical condition through a physiological mechanism before making a judgment that this is the best explanation for the symptoms in a specific individual. A careful and comprehensive assessment of multiple factors is necessary to make this judgment. Several aspects of the clinical presentation should be considered: 1) the presence of a clear temporal association between the onset, exacerbation, or remission of the medical condition and the anxiety symptoms; 2) the presence of features that are atypical of an independent anxiety disorder (e.g., atypical age at onset or course); and 3) evidence in the literature that a known physiological mechanism (e.g., hyperthyroidism) causes anxiety. In addition, the disturbance must not be better explained by an independent anxiety disorder, a substance/medication-induced anxiety disorder, or another mental disorder (e.g., adjustment disorder).

A number of medical conditions are known to include anxiety as a symptomatic manifestation. Examples include endocrine disease (e.g.,

hyperthyroidism, pheochromocytoma, hypoglycemia, hyperadrenocortisolism), cardiovascular disorders (e.g., congestive heart failure, pulmonary embolism, arrhythmia such as atrial fibrillation), respiratory illness (e.g., chronic obstructive pulmonary disease, asthma, pneumonia), metabolic disturbances (e.g., vitamin B_{12} deficiency, porphyria), and neurological illness (e.g., neoplasms, vestibular dysfunction, encephalitis, seizure disorders).

Prevalence

The prevalence of anxiety disorder due to another medical condition is unclear. There appears to be an elevated prevalence of anxiety disorders among individuals with a variety of medical conditions, including asthma, hypertension, ulcers, and arthritis. However, this increased prevalence may be due to reasons other than the anxiety disorder directly causing the medical condition.

Development and Course

The development and course of anxiety disorder due to another medical condition generally follows the course of the underlying illness. This diagnosis is not meant to include primary anxiety disorders that arise in the context of chronic medical illness. This is

important to consider with older adults, who may experience chronic medical illness and then develop independent anxiety disorders secondary to the chronic medical illness.

Diagnostic Markers

Laboratory assessments and/or medical examinations are necessary to confirm the diagnosis of the associated medical condition.

Differential Diagnosis

Delirium and major or mild neurocognitive disorder.

A separate diagnosis of anxiety disorder due to another medical condition is not given if the anxiety disturbance occurs exclusively during the course of a delirium. However, a diagnosis of anxiety disorder due to another medical condition may be given in addition to a diagnosis of major or mild neurocognitive disorder if the anxiety is judged to be a physiological consequence of the pathological process causing the neurocognitive disorder and if anxiety is a prominent part of the clinical presentation.

Mixed presentation of symptoms (e.g., mood and anxiety). If the presentation includes a mix of different types of symptoms, the specific mental disorder due to another medical condition depends on which symptoms predominate in the clinical picture.

Substance/medication-induced anxiety disorder. If there is evidence of recent or prolonged substance use (including medications with psychoactive effects), withdrawal from a substance, or exposure to a toxin, a substance/medication-induced anxiety disorder should be considered. Certain medications are known to increase anxiety (e.g., corticosteroids, estrogens, metoclopramide), and when this is the case, the medication may be the most likely etiology, although it may be difficult to distinguish whether the anxiety is attributable to the medications or to the medical illness itself. When a diagnosis of substance-induced anxiety is being made in relation to recreational or nonprescribed drugs, it may be useful to obtain a urine or blood drug screen or other appropriate laboratory evaluation. Symptoms that develop during or soon after substance intoxication or withdrawal or after medication use may be especially indicative of a substance/medication-induced anxiety disorder, depending on the type, duration, or amount of the substance used. If the disturbance is associated with both another medical condition and substance use, both diagnoses (i.e., anxiety disorder due to another medical condition and substance/medication-induced anxiety disorder) can be given. Features such as onset after age 45 years or the presence of atypical symptoms during a panic attack (e.g., vertigo, loss of consciousness, loss of bladder or bowel control, slurred speech, amnesia) suggest the possibility that another medical condition or a substance may be causing the panic attack symptoms.

Anxiety disorder (not due to a known medical condition).

Anxiety disorder due to another medical condition should be distinguished from other anxiety disorders (especially panic disorder and generalized anxiety disorder). In other anxiety disorders, no specific and direct causative physiological mechanisms associated with another medical condition can be demonstrated. Late age at onset, atypical symptoms, and the absence of a personal or family history of anxiety disorders suggest the need for a thorough assessment to rule out the diagnosis of anxiety disorder due to another medical condition. Anxiety disorders can exacerbate or pose increased risk for medical conditions such as cardiovascular events and myocardial infarction and should not be diagnosed as anxiety disorder due to another medical condition in these cases.

Illness anxiety disorder. Anxiety disorder due to another medical condition should be distinguished from illness anxiety disorder. Illness anxiety disorder is characterized by worry about illness, concern about pain, and bodily preoccupations. In the case of illness anxiety disorder, individuals may or may not have diagnosed medical conditions. Although

an individual with illness anxiety disorder and a diagnosed medical condition is likely to experience anxiety about the medical condition, the medical condition is not physiologically related to the anxiety symptoms.

Adjustment disorders. Anxiety disorder due to another medical condition should be distinguished from adjustment disorders with anxiety or adjustment disorders with anxiety and depressed mood. Adjustment disorder is warranted when individuals experience a maladaptive response to the stress of being diagnosed with or having to manage the medical condition. The reaction to stress usually concerns the meaning or consequences of the medical condition, in contrast with the experience of anxiety or mood symptoms that occur as a physiological consequence of the medical condition. In adjustment disorder, the anxiety symptoms are typically related to coping with the stress of having the medical condition, whereas in anxiety disorder due to another medical condition, individuals are more likely to have prominent physical symptoms and to be focused on issues other than the stress of the illness itself.

Other Specified Anxiety Disorder

F41.8

This category applies to presentations in which symptoms characteristic of an anxiety disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the anxiety disorders diagnostic class, and do not meet criteria for adjustment disorder with anxiety or adjustment disorder with mixed anxiety and depressed mood. The other specified anxiety disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific anxiety disorder. This is done by recording "other specified anxiety disorder" followed by the specific reason (e.g., "generalized anxiety occurring less often than 'more days than not' ").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Limited-symptom attacks.**
2. **Generalized anxiety occurring less often than "more days than not."**
3. ***Khyâl cap* (wind attacks):** See "Culture and Psychiatric Diagnosis" in Section III.
4. ***Ataque de nervios* (attack of nerves):** See "Culture and Psychiatric Diagnosis" in Section III.

Unspecified Anxiety Disorder

F41.9

This category applies to presentations in which symptoms characteristic of an anxiety disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the anxiety disorders diagnostic class, and do not meet criteria for adjustment disorder with anxiety or adjustment disorder with mixed anxiety and depressed mood. The unspecified anxiety disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific anxiety disorder and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Obsessive-Compulsive and Related Disorders

Obsessive-compulsive and related disorders

include obsessive-compulsive disorder (OCD), body dysmorphic disorder, hoarding disorder, trichotillomania (hair-pulling disorder), excoriation (skin-picking) disorder, substance/medication-induced obsessive-compulsive and related disorder, obsessive-compulsive and related disorder due to another medical condition, other specified obsessive-compulsive and related disorder (e.g., nail biting, lip biting, cheek chewing, obsessional jealousy, olfactory reference disorder [olfactory reference syndrome]), and unspecified obsessive-compulsive and related disorder.

OCD is characterized by the presence of obsessions and/or compulsions. *Obsessions* are recurrent and persistent thoughts, urges, or images that are experienced as intrusive and unwanted, whereas *compulsions* are repetitive behaviors or mental acts that an individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly. Some other obsessive-compulsive and related disorders are also characterized by preoccupations and by repetitive behaviors or mental acts in response to the preoccupations. Other obsessive-compulsive and related disorders are characterized primarily by recurrent body-focused repetitive behaviors (e.g., hair pulling, skin picking) and repeated attempts to decrease or stop the behaviors.

The inclusion of a chapter on obsessive-compulsive and related disorders in DSM-5 reflects the increasing evidence of these disorders' relatedness to one another in terms of a range of diagnostic validators as well as the clinical utility of grouping these disorders in the same chapter. Clinicians are encouraged to screen for these conditions in individuals who present with one of them and be aware of overlaps among these conditions.

At the same time, there are important differences in diagnostic validators and treatment approaches across these disorders. Moreover, there are close relationships between the anxiety disorders and some of the obsessive-compulsive and related disorders (e.g., OCD), which is reflected in the sequence of DSM-5 chapters, with obsessive-compulsive and related disorders following anxiety disorders.

The obsessive-compulsive and related disorders differ from developmentally normative preoccupations and rituals by being excessive or persisting beyond developmentally appropriate periods. The distinction between the presence of subclinical symptoms and a clinical disorder requires assessment of a number of factors, including the individual's level of distress and impairment in functioning.

The chapter begins with OCD. It then covers body dysmorphic disorder and hoarding disorder, which are characterized by cognitive symptoms such as perceived defects or flaws in physical appearance or the perceived need to save possessions, respectively. The chapter then covers trichotillomania and excoriation disorder, which are characterized by recurrent body-focused repetitive behaviors. Finally, it covers substance/medication-induced obsessive-compulsive and related disorder, obsessive-compulsive and related disorder due to another medical condition, other specified obsessive-compulsive and related disorder, and unspecified obsessive-compulsive and related disorder.

While the specific content of obsessions and compulsions varies among individuals, certain symptom dimensions are common in OCD, including those of cleaning (contamination obsessions and cleaning compulsions); symmetry (symmetry obsessions and repeating, ordering, and counting compulsions); forbidden or taboo thoughts (e.g., aggressive, sexual, and religious obsessions and related compulsions); and harm (e.g., fears of harm to self or others and related checking compulsions). The tic-related specifier of OCD is used when an individual has a current or past history of a tic disorder.

Body dysmorphic disorder is characterized by preoccupation with one or more perceived defects or flaws in physical appearance that are not

observable or appear only slight to others, and by repetitive behaviors (e.g., mirror checking, excessive grooming, skin picking, or reassurance seeking) or mental acts (e.g., comparing one's appearance with that of other people) in response to the appearance concerns. The appearance preoccupations are not better explained by concerns with body fat or weight in an individual with an eating disorder. Muscle dysmorphia is a form of body dysmorphic disorder that is characterized by the belief that one's body build is too small or is insufficiently muscular.

Hoarding disorder is characterized by persistent difficulty discarding or parting with possessions, regardless of their actual value, as a result of a strong perceived need to save the items and distress associated with discarding them. Hoarding disorder differs from normal collecting. For example, symptoms of hoarding disorder result in the accumulation of a large number of possessions that congest and clutter active living areas to the extent that their intended use is substantially compromised. The excessive acquisition form of hoarding disorder, which characterizes most but not all individuals with hoarding disorder, consists of excessive collecting, buying, or stealing of items that are not needed or for which there is no available space.

Trichotillomania is characterized by recurrent pulling out of one's hair resulting in hair loss, and repeated attempts to decrease or stop hair pulling. Excoriation disorder is characterized by recurrent picking of one's skin resulting in skin lesions and repeated attempts to decrease or stop skin picking. The body-focused repetitive behaviors that characterize these two disorders are not triggered by obsessions or preoccupations; however, they may be preceded or accompanied by various emotional states, such as feelings of anxiety or boredom. They may also be preceded by an increasing sense of tension or may lead to gratification, pleasure, or a sense of relief when the hair is pulled out or the skin is picked. Individuals with these disorders may have varying degrees of conscious awareness of the behavior while engaging in it, with some individuals displaying more focused attention on the behavior (with preceding tension and subsequent relief) and other individuals displaying more automatic behavior (with the behaviors seeming to occur without full awareness).

Substance/medication-induced obsessive-compulsive and related disorder consists of symptoms characteristic of the obsessive-compulsive

and related disorders developed in the context of substance intoxication or withdrawal or after exposure to or withdrawal from a medication. Obsessive-compulsive and related disorder due to another medical condition involves symptoms characteristic of obsessive-compulsive and related disorders that are the direct pathophysiological consequence of another medical condition.

Other specified obsessive-compulsive and related disorder (e.g., nail biting, lip biting, cheek chewing, obsessional jealousy, olfactory reference disorder [olfactory reference syndrome]) and unspecified obsessive-compulsive and related disorder consist of symptoms that cause clinically significant distress or impairment that do not meet criteria for a specific obsessive-compulsive and related disorder in DSM-5 because of atypical presentation or uncertain etiology. These categories are also used for other specific syndromes that are not listed in Section II and when insufficient information is available to diagnose the presentation as another obsessive-compulsive and related disorder.

Those obsessive-compulsive and related disorders that have a cognitive component (i.e., OCD, body dysmorphic disorder, and hoarding disorder) include a specifier for

indicating the individual’s degree of insight with respect to disorder-related beliefs, which ranges from “good or fair insight” to “poor insight” to “absent insight/delusional beliefs.” Those individuals whose degree of insight is in the “absent insight/delusional beliefs” range should not be given an additional diagnosis of a psychotic disorder unless their delusional beliefs involve content that extends beyond what is characteristic of their obsessive-compulsive and related disorder (e.g., an individual with body dysmorphic disorder who is convinced that his or her food has been poisoned).

Obsessive-Compulsive Disorder

Diagnostic Criteria F42.2

A. Presence of obsessions, compulsions, or both:

Obsessions are defined by (1) and (2):

1. Recurrent and persistent thoughts, urges, or images that are experienced, at some time during the disturbance, as intrusive and unwanted, and that in most individuals cause marked anxiety or distress.
2. The individual attempts to ignore or suppress such thoughts, urges, or images, or to neutralize them with some other thought or action (i.e., by performing a compulsion).

Compulsions are defined by (1) and (2):

1. Repetitive behaviors (e.g., hand washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly.
2. The behaviors or mental acts are aimed at preventing or reducing anxiety or distress, or preventing some dreaded event or situation; however, these behaviors or mental acts are not connected in a realistic way with what they are designed to neutralize or prevent, or are clearly excessive.

   Note: Young children may not be able to articulate the aims of these behaviors or mental acts.

B. The obsessions or compulsions are time-consuming (e.g., take more than 1 hour per day) or cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The obsessive-compulsive symptoms are not attributable to the physiological effects of a substance (e.g., a drug of

abuse, a medication) or another medical condition.

D. The disturbance is not better explained by the symptoms of another mental disorder (e.g., excessive worries, as in generalized anxiety disorder; preoccupation with appearance, as in body dysmorphic disorder; difficulty discarding or parting with possessions, as in hoarding disorder; hair pulling, as in trichotillomania [hair-pulling disorder]; skin picking, as in excoriation [skin-picking] disorder; stereotypies, as in stereotypic movement disorder; ritualized eating behavior, as in eating disorders; preoccupation with substances or gambling, as in substance-related and addictive disorders; preoccupation with having an illness, as in illness anxiety disorder; sexual urges or fantasies, as in paraphilic disorders; impulses, as in disruptive, impulse-control, and conduct disorders; guilty ruminations, as in major depressive disorder; thought insertion or delusional preoccupations, as in schizophrenia spectrum and other psychotic disorders; or repetitive patterns of behavior, as in autism spectrum disorder).

Specify if:

With good or fair insight: The individual recognizes that obsessive-compulsive disorder beliefs are definitely or probably not true or that they may or may not be true.

With poor insight: The individual thinks obsessive-compulsive disorder beliefs are probably true.

With absent insight/delusional beliefs: The individual is completely convinced that obsessive-compulsive disorder beliefs are true.

Specify if:

Tic-related: The individual has a current or past history of a tic disorder.

Specifiers

Individuals with obsessive-compulsive disorder (OCD) vary in the degree of insight they have about the accuracy of the beliefs that underlie their obsessive-compulsive symptoms. Many individuals have *good or fair insight* (e.g., the individual believes that the house definitely will not, probably will not, or may or may not burn down if the stove is not checked 30 times). Some have *poor insight* (e.g., the individual believes that the house will probably burn down if the stove is not checked 30 times), and a few (4% or less) have *absent insight/delusional beliefs* (e.g., the individual is convinced that the house will burn down if the stove is not checked 30 times). Insight can vary within an individual over the course of the illness. Poorer insight has been linked to worse long-term outcome.

Up to 30% of individuals with OCD have a lifetime tic disorder. This is most common in men with onset of OCD in childhood. These individuals tend to differ from those without a history of tic disorders in the themes of their OCD symptoms, comorbidity, course, and pattern of familial transmission.

Diagnostic Features

The characteristic symptoms of OCD are the presence of obsessions and compulsions (Criterion A). *Obsessions* are repetitive and persistent thoughts (e.g., of contamination), images (e.g., of violent or horrific scenes), or urges (e.g., to stab someone). Importantly, obsessions are not pleasurable or experienced as voluntary: they are intrusive and unwanted and cause marked distress or anxiety in most individuals. The individual attempts to ignore or suppress these obsessions (e.g., avoiding triggers or using thought suppression) or to neutralize them with another thought or action (e.g., performing a compulsion). *Compulsions* (or rituals) are repetitive behaviors (e.g., washing, checking) or mental acts (e.g., counting, repeating words silently) that the individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly. Most individuals with OCD have both obsessions and compulsions. Obsessions and compulsions are typically thematically related (e.g., thoughts of contamination associated with washing rituals; thoughts of harm associated with repeated checking). Individuals often report that they perform

compulsions to reduce the distress triggered by obsessions or to prevent a feared event (e.g., becoming ill). However, these compulsions either are not connected in a realistic way to the feared event (e.g., arranging items symmetrically to prevent harm to a loved one) or are clearly excessive (e.g., showering for hours each day). Compulsions are not done for pleasure, although individuals may experience temporary relief from anxiety or distress.

The specific content of obsessions and compulsions varies between individuals. However, certain themes, or dimensions, are common, including those of cleaning (contamination obsessions and cleaning compulsions); symmetry (symmetry obsessions and repeating, ordering, and counting compulsions); forbidden or taboo thoughts (e.g., aggressive, sexual, or religious obsessions and related compulsions); and harm (e.g., fears of harm to self or others and checking compulsions). Some individuals also have difficulties discarding and accumulate objects as a consequence of typical obsessions and

compulsions (e.g., fears of harming others); such compulsions must be distinguished from the primary accumulation behaviors seen in hoarding disorder, discussed later in this chapter. These themes occur across different cultures, are relatively consistent over time in adults with the disorder, and may be associated with different neural substrates. Importantly, individuals often have symptoms in more than one dimension.

Criterion B emphasizes that obsessions and compulsions must be time-consuming (e.g., more than 1 hour per day) or cause clinically significant distress or impairment to warrant a diagnosis of OCD. This criterion helps to distinguish the disorder from the occasional intrusive thoughts or repetitive behaviors that are common in the general population (e.g., double-checking that a door is locked). The frequency and severity of obsessions and compulsions vary across individuals with OCD (e.g., some have mild to moderate symptoms, spending 1–3 hours per day obsessing or doing compulsions, whereas others have nearly constant intrusive thoughts or compulsions that can be incapacitating).

Associated Features

Sensory phenomena, defined as physical experiences (e.g., physical sensations, just-right sensations, and feelings of incompleteness) that precede compulsions, are common in OCD. Up to 60% of individuals with OCD report these phenomena.

Individuals with OCD experience a range of affective responses when confronted with situations that trigger obsessions and compulsions. For example, many individuals experience marked anxiety that can include recurrent panic attacks. Others report strong feelings of disgust. While performing compulsions, some individuals report a distressing sense of "incompleteness" or uneasiness until things look, feel, or sound "just right."

It is common for individuals with the disorder to avoid people, places, and things that trigger obsessions and compulsions. For example, individuals with contamination concerns might avoid public situations (e.g., restaurants, public restrooms) to reduce exposure to feared contaminants; individuals with intrusive thoughts about causing harm might avoid social interactions.

Many individuals with OCD have dysfunctional beliefs. These beliefs can include an inflated sense of responsibility and the tendency to overestimate threat; perfectionism and intolerance of uncertainty; and overimportance of thoughts (e.g., believing that having a forbidden thought is as bad as acting on it) and the need to control thoughts. These beliefs, however, are not specific to OCD. The involvement of family or friends in compulsive rituals, termed *accommodation*, can exacerbate or maintain symptoms and is an important target in treatment, especially in children.

Prevalence

The 12-month prevalence of OCD in the United States is 1.2%, with a similar prevalence internationally (including Canada, Puerto Rico, Germany, Taiwan, Korea, and New Zealand; 1.1%–1.8%). Women are affected at a slightly higher rate than men in adulthood, although men are more commonly affected in childhood.

Development and Course

In the United States, the mean age at onset of OCD is 19.5 years, and 25% of cases start by age 14 years. Onset after age 35 years is unusual but does occur. Men have an earlier age at onset than women: nearly 25% of men have onset before age 10 years. The onset of symptoms is typically gradual; however, acute onset can also occur.

If OCD is untreated, the course is usually chronic, often with waxing and waning symptoms. Some individuals have an episodic course, and a minority have a deteriorating course. Without treatment, remission rates in adults are low (e.g., 20% for those reevaluated 40 years later). Onset in childhood or adolescence can lead to a lifetime of OCD.

However, 40% of individuals with onset of OCD in childhood or adolescence may experience remission by early adulthood. The course of OCD is often complicated by the co-occurrence of other disorders (see section “Comorbidity” for this disorder).

Compulsions are more easily diagnosed in children than obsessions are because compulsions are usually observable. However, most children have both obsessions and compulsions (as do most adults). The pattern of symptoms in adults can be stable over time, but it is more variable in children. Some differences in the content of obsessions and compulsions have been reported when children and adolescent samples are compared with adult samples. These differences likely reflect content appropriate to different developmental stages (e.g., higher rates of sexual and religious obsessions in adolescents than in children; higher rates of harm obsessions [e.g., fears of catastrophic events, such as death or illness to self or loved ones] in children and adolescents than in adults).

Risk and Prognostic Factors

Temperamental. Greater internalizing symptoms, higher negative emotionality, and behavioral inhibition in childhood are possible temperamental risk factors.

Environmental. Different environmental factors may increase the risk for OCD. These include adverse perinatal events, premature birth, maternal

tobacco use during pregnancy, physical and sexual abuse in childhood, and other stressful or traumatic events. Some children may develop the sudden onset of obsessive-compulsive symptoms, which has been associated with different environmental factors, including various infectious agents and a postinfectious autoimmune syndrome.

Genetic and physiological. The rate of OCD among first-degree relatives of adults with OCD is approximately two times that among first-degree relatives of those without the disorder; however, among first-degree relatives of individuals with onset of OCD in childhood or adolescence, the rate is increased 10-fold. Familial transmission is due in part to genetic factors (e.g., a concordance rate of 0.57 for monozygotic vs. 0.22 for dizygotic twins). Twin studies suggest that additive genetic effects account for ~40% of the variance in obsessive-compulsive symptoms. Dysfunction in the orbitofrontal cortex, anterior cingulate cortex, and striatum have been most strongly implicated; alterations in frontolimbic, frontoparietal, and cerebellar networks have also been reported.

Culture-Related Diagnostic Issues

OCD occurs across the world. There is substantial similarity across cultures in the gender distribution, age at onset, and comorbidity of OCD. Moreover, around the globe, there is a similar symptom structure involving cleaning, symmetry, hoarding, taboo thoughts, and fear of harm. However, regional variation in symptom expression exists, and cultural factors may shape the content of obsessions and compulsions. For example, obsessions related to sexual content may be reported less frequently in some religious and cultural groups, and obsessions related to violence and aggression may be more common in settings with higher prevalence of urban violence. Attributions of OCD symptoms vary cross-culturally, including physical, social, spiritual, and supernatural causes; specific compulsions and help-seeking options may be reinforced by these cultural attributions.

Sex- and Gender-Related Diagnostic Issues

Men have an earlier age at onset of OCD than women, often in childhood, and are more likely to have comorbid tic disorders. Onset in girls is more typically in adolescence; among adults, OCD is slightly more common in

women than in men. Gender differences in the pattern of symptom dimensions have been reported, with, for example, women more likely to have symptoms in the cleaning dimension and men more likely to have

symptoms in the forbidden thoughts and symmetry dimensions. Onset or exacerbation of OCD, as well as symptoms that can interfere with the mother-infant relationship (e.g., aggressive obsessions such as intrusive violent thoughts of harming the infant, leading to avoidance of the infant), has been reported in the peripartum period. Some women also report exacerbation of OCD symptoms premenstrually.

Association With Suicidal Thoughts or Behavior

A systematic literature review of suicidal ideation and suicide attempts in clinical samples with OCD from multiple countries found a mean rate of lifetime suicide attempts of 14.2%, a mean rate of lifetime suicidal ideation of 44.1%, and a mean rate of current suicidal ideation of 25.9%. Predictors of greater suicide risk were severity of OCD, the symptom dimension of unacceptable thoughts, severity of comorbid depressive and anxiety symptoms, and past history of suicidality. Another international systematic review of 48 studies found a moderate to high significant association between suicidal ideation/suicide attempts and OCD.

A cross-sectional study of 582 outpatients with OCD from Brazil found that 36% reported lifetime suicidal thoughts, 20% had made suicide plans, 11% had already attempted suicide, and 10% presented with current suicidal thoughts. The sexual/religious dimension of OCD and comorbid substance use disorders were associated with suicidal thoughts and suicide plans, impulse-control disorders were associated with current suicidal thoughts and with suicide plans and attempts, and lifetime comorbid major depressive disorder and posttraumatic stress disorder (PTSD) were associated with all aspects of suicidal behaviors.

In a study using Swedish national registry data involving 36,788 individuals with OCD and matched general population control subjects, individuals with OCD had a higher risk of suicide death (OR = 9.8) and

suicide attempt (OR = 5.5), and the increased risk for both outcomes remained substantial even after adjusting for psychiatric comorbidities. Comorbid personality or substance use disorder increased suicide risk, whereas female gender, higher parental education, and a comorbid anxiety disorder were protective factors.

Functional Consequences of Obsessive-Compulsive Disorder

OCD is associated with reduced quality of life as well as high levels of social and occupational impairment. Impairment occurs across many different domains of life and is associated with symptom severity. Impairment can be caused by the time spent obsessing and performing compulsions. Avoidance of situations that can trigger obsessions or compulsions can also severely impair functioning. In addition, specific symptoms can create specific obstacles. For example, obsessions about harm can make relationships with family and friends feel hazardous; the result can be avoidance of these relationships. Obsessions about symmetry can derail the timely completion of school or work projects because the project never feels “just right,” potentially resulting in school failure or job loss. Health consequences can also occur. For example, individuals with contamination concerns may avoid doctors’ offices and hospitals (e.g., because of fears of exposure to germs) or develop dermatological problems (e.g., skin lesions due to excessive washing). Sometimes the symptoms of the disorder interfere with its own treatment (e.g., when medications are considered contaminated). When the disorder starts in childhood or adolescence, individuals may experience developmental difficulties. For example, adolescents may avoid socializing with peers; young adults may struggle when they leave home to live independently. The result can be few significant relationships outside the family and a lack of autonomy and financial independence from their family of origin. In addition, some individuals with OCD try to impose rules and prohibitions on family members because of their obsessions

(e.g., no one in the family can have visitors to the house for fear of contamination), and this can lead to family dysfunction.

Differential Diagnosis

Anxiety disorders. Recurrent thoughts, avoidant behaviors, and repetitive requests for reassurance can also occur in anxiety disorders. However, the recurrent thoughts that are present in generalized anxiety disorder (i.e., worries) are usually about real-life concerns, whereas the obsessions of OCD usually do not involve real-life concerns and can include content that is odd, irrational, or of a seemingly magical nature; moreover, compulsions are usually present and usually linked to the obsessions. Like individuals with OCD, individuals with specific phobia can have a fear reaction to specific objects or situations; however, in specific phobia the feared object is usually much more circumscribed, and rituals are not present. In social anxiety disorder, the feared objects or situations are limited to social interactions or performance situations, and avoidance or reassurance seeking is focused on reducing feelings of embarrassment.

Major depressive disorder. OCD needs to be distinguished from the rumination of major depressive disorder, in which thoughts are usually mood-congruent and not necessarily experienced as intrusive or distressing; moreover, ruminations are not linked to compulsions, as is typical in OCD.

Other obsessive-compulsive and related disorders. In body dysmorphic disorder, the obsessions and compulsions are limited to concerns about physical appearance; and in trichotillomania (hair-pulling disorder), the compulsive behavior is limited to hair pulling in the absence of obsessions. Hoarding disorder symptoms focus exclusively on the persistent difficulty discarding or parting with possessions, marked distress associated with discarding items, and excessive accumulation of objects. However, if an individual has obsessions that are typical of OCD (e.g., concerns about incompleteness or harm), and these obsessions lead to compulsive accumulation (e.g., acquiring all objects in a set to attain a sense of completeness or not discarding old newspapers because they may contain information that could prevent harm), a diagnosis of OCD should be given instead.

Eating disorders.

OCD can be distinguished from anorexia nervosa in that in OCD the obsessions and compulsions are not limited to concerns about weight and food.

Tics (in tic disorder) and stereotyped movements. A *tic* is a sudden, rapid, recurrent, nonrhythmic motor movement or vocalization (e.g., eye blinking, throat clearing). A *stereotyped movement* is a repetitive, seemingly driven, nonfunctional motor behavior (e.g., head banging, body rocking, self-biting). Tics and stereotyped movements are typically less complex than compulsions and are not aimed at neutralizing obsessions. However, distinguishing between complex tics and compulsions can be difficult. Whereas compulsions are usually preceded by obsessions, tics are often preceded by premonitory sensory urges. Some individuals have symptoms of both OCD and a tic disorder, in which case both diagnoses may be warranted.

Psychotic disorders. Some individuals with OCD have poor insight or even delusional OCD beliefs. However, they have obsessions and compulsions (distinguishing their condition from delusional disorder) and do not have other features of schizophrenia or schizoaffective disorder (e.g., hallucinations or disorganized speech). For individuals whose OCD symptoms warrant the "with absent insight/delusional beliefs" specifier, these symptoms should not be diagnosed as a psychotic disorder.

Other compulsive-like behaviors. Certain behaviors are sometimes described as "compulsive," including sexual behavior (in the case of paraphilias), gambling (i.e., gambling disorder), and substance use (e.g., alcohol use disorder). However, these behaviors differ

from the compulsions of OCD in that the person usually derives pleasure from the activity and may wish to resist it only because of its deleterious consequences.

Obsessive-compulsive personality disorder. Although obsessive-compulsive personality disorder and OCD have similar names, the clinical manifestations of these disorders are quite different. Obsessive-compulsive personality disorder is not characterized by intrusive thoughts, images, or

urges or by repetitive behaviors that are performed in response to these intrusive symptoms; instead, it involves an enduring and pervasive maladaptive pattern of excessive perfectionism and rigid control. If an individual manifests symptoms of both OCD and obsessive-compulsive personality disorder, both diagnoses can be given.

Comorbidity

Individuals with OCD often have other psychopathology. Many adults with the disorder in the United States have a lifetime diagnosis of an anxiety disorder (76%; e.g., panic disorder, social anxiety disorder, generalized anxiety disorder, specific phobia) or a depressive or bipolar disorder (63% for any depressive or bipolar disorder, with the most common being major depressive disorder [41%]); a lifetime diagnosis of an impulse-control disorder (56%) or a substance use disorder (39%) is also common. Onset of OCD is usually later than for most comorbid anxiety disorders (with the exception of separation anxiety disorder) and PTSD but often precedes that of depressive disorders. In a study of 214 treatment-seeking adults in the United States with DSM-IV OCD at intake, comorbid obsessive-compulsive personality disorder was found in 23%–32% of individuals followed longitudinally.

Up to 30% of individuals with OCD also have a lifetime tic disorder. A comorbid tic disorder is most common in men with onset of OCD in childhood. These individuals tend to differ from those without a history of tic disorders in the themes of their OCD symptoms, comorbidity, course, and pattern of familial transmission. A triad of OCD, tic disorder, and attention-deficit/hyperactivity disorder can also be seen in children.

Several obsessive-compulsive and related disorders, including body dysmorphic disorder, trichotillomania, and excoriation (skin-picking) disorder, also occur more frequently in individuals with OCD than in those without OCD.

OCD is also much more common in individuals with certain other disorders than would be expected based on its prevalence in the general population; when one of those other disorders is diagnosed, the individual should be assessed for OCD as well. For example, in individuals with schizophrenia or schizoaffective disorder, the prevalence of OCD is

approximately 12%. Rates of OCD are also elevated in bipolar disorder; eating disorders, such as anorexia nervosa and bulimia nervosa; body dysmorphic disorder; and Tourette's disorder.

Body Dysmorphic Disorder

Diagnostic Criteria F45.22

A. Preoccupation with one or more perceived defects or flaws in physical appearance that are not observable or appear slight to others.

B. At some point during the course of the disorder, the individual has performed repetitive behaviors (e.g., mirror checking, excessive grooming, skin picking, reassurance seeking) or mental acts (e.g., comparing his or her appearance with that of others) in response to the appearance concerns.

C. The preoccupation causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The appearance preoccupation is not better explained by concerns with body fat or weight in an individual whose symptoms meet diagnostic criteria for an eating disorder.

Specify if:

With muscle dysmorphia: The individual is preoccupied with the idea that his or her body build is too small or insufficiently muscular. This specifier is used even if the individual is preoccupied with other body areas, which is often the case.

Specify if:

Indicate degree of insight regarding body dysmorphic disorder beliefs (e.g., “I look ugly” or “I look deformed”).

With good or fair insight: The individual recognizes that the body dysmorphic disorder beliefs are definitely or probably not true or that they may or may not be true.

With poor insight: The individual thinks that the body dysmorphic disorder beliefs are probably true.

With absent insight/delusional beliefs: The individual is completely convinced that the body dysmorphic disorder beliefs are true.

Specifiers

Muscle dysmorphia, a form of body dysmorphic disorder occurring almost exclusively in men and adolescent boys, consists of preoccupation with the idea that one’s body is too small or insufficiently lean or muscular. Individuals with this form of the disorder actually have a normal-looking body or are even very muscular. They may also be preoccupied with other body areas, such as skin or hair. A majority (but not all) diet, exercise, and/or lift weights excessively, sometimes causing bodily damage. Some use potentially dangerous anabolic-androgenic steroids and other substances to try to make their body bigger and more muscular.

Individuals with body dysmorphic disorder vary in the degree of insight they have about the accuracy of their body dysmorphic disorder beliefs (e.g., “I look ugly,” “I look deformed”). Insight regarding body dysmorphic disorder beliefs can range from good to absent/delusional (i.e., delusional beliefs consisting of complete conviction that the individual’s view of his or her appearance is accurate and undistorted). On average, insight is poor, and one-third or more of individuals currently have absent insight/delusional body dysmorphic disorder beliefs. Individuals with delusional body dysmorphic disorder tend to have greater morbidity in some areas (e.g., suicidal thoughts or behavior), but this appears to be accounted for by their tendency to have more severe body dysmorphic disorder symptoms.

Diagnostic Features

Individuals with body dysmorphic disorder (formerly known as *dysmorphophobia*) are preoccupied with one or more perceived defects or flaws in their physical appearance, which they believe look ugly, unattractive, abnormal, or deformed (Criterion A). The perceived flaws are not observable or appear only slight to other individuals. Concerns range from looking “unattractive” or “not right” to looking “hideous” or “like a monster.” Preoccupations can focus on one or many body areas, most commonly the skin (e.g., perceived acne, scars, lines, wrinkles, paleness), hair (e.g., “thinning” hair or “excessive” body or facial hair), or nose (e.g., size or shape). However, any body area can be the focus of concern (e.g., eyes, teeth, weight, stomach, breasts, legs, face size or shape, lips, chin, eyebrows, genitals). Some individuals are concerned about perceived asymmetry of body areas. The preoccupations are intrusive, unwanted, time-consuming (occurring, on average, 3–8 hours per day), and usually difficult to resist or control.

Excessive repetitive behaviors or mental acts (e.g., comparing) are performed in response to the preoccupation (Criterion B). The individual feels driven to perform these

behaviors, which are not pleasurable and may increase anxiety and dysphoria. They are typically time-consuming and difficult to resist or control. Common behaviors are comparing one’s appearance with that of other individuals; repeatedly checking perceived defects in mirrors or other reflecting surfaces or examining them directly; taking excessive “selfies”; excessively grooming (e.g., combing, styling, shaving, plucking or pulling hair); seeking reassurance about how the perceived flaws look; touching disliked areas to check them; excessively exercising or weight lifting; and seeking cosmetic procedures. Some individuals excessively tan (e.g., to darken “pale” skin or diminish perceived acne), repeatedly change their clothes (e.g., to camouflage perceived defects), or compulsively shop (e.g., for beauty products). Compulsive skin picking intended to improve perceived skin defects is common and can cause skin damage, infections, or ruptured blood vessels. Camouflaging (i.e., hiding or covering) perceived defects, a very common behavior in individuals with body dysmorphic

disorder, may involve repetitive behaviors (e.g., repeatedly applying makeup, adjusting a hat or clothing, rearranging head hair to cover the forehead or eyes). The preoccupation must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C); usually both are present. Body dysmorphic disorder must be differentiated from an eating disorder. Body dysmorphic disorder by proxy is a form of body dysmorphic disorder in which individuals are preoccupied with defects they perceive in another person's appearance, most often a significant other (e.g., spouse or partner), but sometimes a parent, child, sibling, or stranger.

Associated Features

Many individuals with body dysmorphic disorder have ideas or delusions of reference, believing that other people take special notice of them or mock them because of how they look. Body dysmorphic disorder is associated with high levels of anxiety, social anxiety, social avoidance, depressed mood, negative affectivity (neuroticism), rejection sensitivity, and perfectionism as well as low extroversion and low self-esteem. Body dysmorphic disorder is also associated with elevated hostility and aggressive behavior. Many individuals are ashamed of their appearance and their excessive focus on how they look and are reluctant to reveal their concerns to others. A majority of individuals receive cosmetic treatment to try to improve their perceived defects. Dermatological treatment and surgery are most common, but any type (e.g., dental, electrolysis) may be received. Some individuals perform surgery on themselves. Body dysmorphic disorder appears to respond poorly to such treatments and sometimes becomes worse. Some individuals take legal action or are violent toward the clinician (e.g., surgeon) because they are dissatisfied with the cosmetic outcome.

Body dysmorphic disorder has been associated with abnormalities in emotion recognition, attention, and executive function, as well as information-processing biases and inaccuracies in interpretation of information and social situations. For example, individuals with this disorder tend to have a bias for negative and threatening interpretations of facial expressions and ambiguous scenarios. Body dysmorphic disorder is

also characterized by visual processing abnormalities, with a bias for analyzing and encoding details rather than holistic or configural aspects of visual stimuli.

Prevalence

The point prevalence in a nationwide epidemiological study among U.S. adults was 2.4% (2.5% in women and 2.2% in men). Outside the United States (e.g., Germany), similar studies indicate that the point prevalence is 1.7%–2.9%, with a gender distribution similar to that in the United States. Globally, the point prevalence is 11%–13% among dermatology patients, 13%–15% among general cosmetic surgery patients, 20% in rhinoplasty surgery patients, 11% among adult jaw correction surgery patients, and 5%–10% among adult orthodontia/cosmetic dentistry patients. Among adolescents and college students, point prevalence rates are relatively higher in girls/young women compared with boys/young men.

Development and Course

The mean age at disorder onset is 16–17 years, the median age at onset is 15 years, and the most common age at onset is 12–13 years; in two-thirds of individuals, onset is before age 18. Subclinical body dysmorphic disorder symptoms begin, on average, at age 12 or 13 years. Subclinical concerns usually evolve gradually to the full disorder, although some individuals experience abrupt onset of body dysmorphic disorder. The disorder appears to usually be chronic, although improvement is likely when evidence-based treatment is received. The disorder’s clinical features appear largely similar in children/adolescents and adults. Body dysmorphic disorder occurs in the elderly, but little is known about the disorder in this age group. Individuals with disorder onset before age 18 years have more comorbidity and are more likely to have gradual (rather than acute) disorder onset than those with adult-onset body dysmorphic disorder.

Risk and Prognostic Factors

Environmental.

Body dysmorphic disorder has been associated with high rates of childhood neglect, abuse, and trauma, as well as elevated rates of teasing.

Genetic and physiological. The prevalence of body dysmorphic disorder is elevated in first-degree relatives of individuals with obsessive-compulsive disorder (OCD). Heritability of body dysmorphic disorder symptoms is estimated at 37%–49% in studies of adolescent and young adult twins and may be higher in women. There is shared genetic vulnerability with OCD as well as genetic influences that are specific to body dysmorphic disorder symptoms.

Culture-Related Diagnostic Issues

Body dysmorphic disorder has been reported internationally. Certain features of the disorder appear cross-culturally, such as gender ratio, body areas that are the object of concern, types of repetitive behaviors, and levels of associated distress and impairment. Other features may vary (e.g., in some cultural contexts with a collectivistic focus, such as Japan, body dysmorphic concerns might emphasize the fear of offending others because of the perceived deformity).

Varying cultural standards may be associated with specific body image concerns, such as eyelids in Japan and muscle dysmorphia in Western countries. *Taijin kyofusho*, included in the traditional Japanese diagnostic system, has a subtype similar to body dysmorphic disorder: *shubo-kyofu* ("the phobia of a deformed body"). For more information regarding cultural concepts of distress, refer to the "Culture and Psychiatric Diagnosis" chapter.

Sex- and Gender-Related Diagnostic Issues

Muscle dysmorphia occurs almost exclusively in men, and men are more likely to have a comorbid substance use disorder, whereas women are more likely to have a comorbid eating disorder. Women and men appear to have more similarities than differences in terms of most clinical features— for example, disliked body areas, types of repetitive behaviors, symptom severity, suicidality, comorbidity, illness course, and receipt of cosmetic procedures for body dysmorphic disorder. However, there are some differences. For example, men are more likely to have preoccupations with

their genitals, body build (thinking they are too small or inadequately muscular), and thinning hair, whereas women are more likely to be preoccupied with weight (usually thinking that they weigh too much), breasts/chest, buttocks, legs, hips, and excessive body/facial hair.

Association With Suicidal Thoughts or Behavior

In a systematic review and meta-analysis of 17 studies that examined suicidal thoughts and behaviors across several countries, individuals with body dysmorphic disorder were

four times more likely to have experienced suicidal thoughts (pooled OR = 3.87) and 2.6 times more likely to have made suicide attempts (pooled OR = 2.57) when compared with healthy control subjects and individuals diagnosed with eating disorders, OCD, or any anxiety disorder. Two general population studies in Germany found higher rates of suicidal thoughts—19% vs. 3%; 31.0% vs. 3.5%—and behaviors—7% vs. 1%; 22.2% vs. 2.1%—in individuals diagnosed with body dysmorphic disorder than in those without the diagnosis.

Severity of body dysmorphic disorder strengthens the association of body dysmorphic disorder with suicidal thoughts and behaviors. The relationship between body dysmorphic disorder and elevated suicidal thoughts and behaviors is independent of comorbidity, but certain comorbidities may further strengthen this relationship. A substantial proportion of individuals with body dysmorphic disorder attribute suicidal thoughts or suicide attempts primarily to their appearance concerns.

Individuals with body dysmorphic disorder have many demographic and clinical risk factors that more generally predict suicide death, such as high rates of suicidal thoughts and suicide attempts, unemployment, perceived abuse, poor self-esteem, and high rates of comorbid major depressive disorder, eating disorders, and substance use disorders.

Functional Consequences of Body Dysmorphic Disorder

Nearly all individuals with body dysmorphic disorder experience impaired psychosocial functioning because of their appearance concerns. Impairment can range from moderate (e.g., avoidance of some social situations) to extreme and incapacitating (e.g., being completely housebound).

On average, psychosocial functioning and quality of life are markedly poor. More severe body dysmorphic disorder symptoms are associated with poorer functioning and quality of life. Most individuals experience impairment in their job, academic, or role functioning (e.g., as a parent or caregiver), which is often severe (e.g., performing poorly, missing school or work, not working). About 20% of youths with body dysmorphic disorder report dropping out of school primarily because of their body dysmorphic disorder symptoms. A high proportion of adults and adolescents have been psychiatrically hospitalized.

Differential Diagnosis

Normal appearance concerns and clearly noticeable physical defects. Body dysmorphic disorder differs from normal appearance concerns in being characterized by excessive appearance-related preoccupations and repetitive behaviors that are time-consuming, are usually difficult to resist or control, and cause clinically significant distress or impairment in functioning. Physical defects that are clearly noticeable (i.e., not slight) are not diagnosed as body dysmorphic disorder. However, skin picking as a symptom of body dysmorphic disorder can cause noticeable skin lesions and scarring; in such cases, body dysmorphic disorder should be diagnosed.

Eating disorders. In an individual with an eating disorder, concerns about being fat or overweight are considered a symptom of the eating disorder rather than body dysmorphic disorder. However, weight concerns may occur in body dysmorphic disorder. Eating disorders and body dysmorphic disorder can be comorbid, in which case both should be diagnosed.

Other obsessive-compulsive and related disorders. The preoccupations and repetitive behaviors of body dysmorphic disorder differ from obsessions and compulsions in OCD in that the former focus only on physical appearance. These disorders have other differences, such as poorer insight, more frequent depression, and higher rates of suicidal

ideation in body dysmorphic disorder. When skin picking is intended to improve the appearance of perceived skin defects, body dysmorphic disorder, rather than excoriation (skin-picking) disorder, is diagnosed. When hair removal (plucking, pulling, or other types of removal) is intended to improve perceived defects in the appearance of facial, head, or body hair, body dysmorphic disorder is diagnosed rather than trichotillomania (hair-pulling disorder).

Illness anxiety disorder. Individuals with body dysmorphic disorder are not preoccupied with having or acquiring a serious illness and in clinical samples do not have particularly elevated levels of somatization.

Major depressive disorder. The prominent preoccupation with appearance and excessive repetitive behaviors in body dysmorphic disorder differentiate it from major depressive disorder. However, major depressive disorder and depressive symptoms are common in individuals with body dysmorphic disorder, often appearing to be secondary to the distress and impairment that body dysmorphic disorder causes. Body dysmorphic disorder should be diagnosed in depressed individuals if diagnostic criteria for body dysmorphic disorder are met.

Anxiety disorders. Social anxiety and avoidance are common in body dysmorphic disorder. However, unlike social anxiety disorder, agoraphobia, and avoidant personality disorder, body dysmorphic disorder includes prominent appearance-related preoccupation, which may be delusional, and repetitive behaviors. In addition, the social anxiety and avoidance that are characteristic of body dysmorphic disorder are attributable to concerns about perceived appearance defects and the belief or fear that other people will consider these individuals ugly, ridicule them, or reject them because of their physical features. Unlike generalized anxiety disorder, anxiety and worry in body dysmorphic disorder focus on perceived appearance flaws.

Psychotic disorders. Many individuals with body dysmorphic disorder have delusional appearance beliefs (i.e., complete conviction that their view of their perceived defects is accurate), which is diagnosed as body dysmorphic disorder, with absent insight/delusional beliefs, not as delusional disorder. Appearance-related ideas or delusions of reference are common in body

dysmorphic disorder (i.e., thinking that other people take special notice in a negative way because of the individual's appearance). However, unlike schizophrenia or schizoaffective disorder, body dysmorphic disorder involves prominent appearance preoccupations and related repetitive behaviors; disorganized behavior and other psychotic symptoms are absent (except for appearance beliefs, which may be delusional). For individuals whose obsessive-compulsive and related disorder symptoms warrant the "with absent insight/delusional beliefs" specifier, these symptoms should not be diagnosed as a psychotic disorder.

Other disorders and symptoms. Body dysmorphic disorder should not be diagnosed if the preoccupation is limited to discomfort with or a desire to be rid of one's primary and/or secondary sex characteristics in an individual with *gender dysphoria*. Nor should body dysmorphic disorder be diagnosed if the preoccupation focuses on the belief that one emits a foul or offensive body odor as in olfactory reference disorder (olfactory reference syndrome), which is an example of an other specified obsessive-compulsive and related disorder in DSM-5. *Body integrity dysphoria* (which is included in ICD-11 but not DSM-5) involves a persistent desire to become an amputee in order to correct a mismatch between the individual's sense of how his or her body should be configured and his or her actual anatomical configuration. In contrast to body dysmorphic disorder, the individual does not feel that the limb to be amputated is ugly or defective in any way, just that it should not be there. *Koro*, a culturally related disorder that usually occurs in epidemics in Southeastern Asia, consists of a fear that the penis (labia, nipples, or breasts in females) is shrinking or retracting and will disappear into the abdomen, often accompanied by a belief that death

will result. *Koro* differs from body dysmorphic disorder in several ways, including a focus on death rather than preoccupation with perceived ugliness. *Dysmorphic concern* (which is not a DSM-5 disorder) is a broader construct that is similar to, but not equivalent to, body dysmorphic disorder. It involves symptoms reflecting an overconcern with slight or imagined flaws in appearance.

Comorbidity

Major depressive disorder is the most common comorbid disorder, with onset usually after that of body dysmorphic disorder. Comorbid social anxiety disorder, OCD, and substance-related disorders (including use of anabolic-androgenic steroids in the muscle dysmorphia form of body dysmorphic disorder) are also common.

Hoarding Disorder

Diagnostic Criteria F42.3

A. Persistent difficulty discarding or parting with possessions, regardless of their actual value.
B. This difficulty is due to a perceived need to save the items and to distress associated with discarding them.
C. The difficulty discarding possessions results in the accumulation of possessions that congest and clutter active living areas and substantially compromises their intended use. If living areas are uncluttered, it is only because of the interventions of third parties (e.g., family members, cleaners, authorities).
D. The hoarding causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (including maintaining a safe environment for self and others).
E. The hoarding is not attributable to another medical condition (e.g., brain injury, cerebrovascular disease, Prader-Willi syndrome).
F. The hoarding is not better explained by the symptoms of another mental disorder (e.g., obsessions in obsessive-compulsive disorder, decreased energy in major depressive disorder, delusions in schizophrenia or another psychotic

disorder, cognitive deficits in major neurocognitive disorder, restricted interests in autism spectrum disorder).

Specify if:

With excessive acquisition: If difficulty discarding possessions is accompanied by excessive acquisition of items that are not needed or for which there is no available space.

Specify if:

With good or fair insight: The individual recognizes that hoarding-related beliefs and behaviors (pertaining to difficulty discarding items, clutter, or excessive acquisition) are problematic.

With poor insight: The individual is mostly convinced that hoarding-related beliefs and behaviors (pertaining to difficulty discarding items, clutter, or excessive acquisition) are not problematic despite evidence to the contrary.

With absent insight/delusional beliefs: The individual is completely convinced that hoarding-related beliefs and behaviors (pertaining to difficulty discarding items, clutter, or excessive acquisition) are not problematic despite evidence to the contrary.

Specifiers

With excessive acquisition. Approximately 80%–90% of individuals with hoarding disorder display excessive acquisition. The most frequent form of acquisition is excessive buying, followed by acquisition of free items (e.g., leaflets, items discarded by others). Stealing is less common. Some individuals may deny excessive acquisition when first assessed, yet it may appear later during the course of treatment. Individuals with hoarding disorder typically experience distress if they are unable to or are prevented from acquiring items.

Diagnostic Features

The essential feature of hoarding disorder is persistent difficulty discarding or parting with possessions, regardless of their actual value (Criterion A). The term *persistent* indicates a long-standing difficulty rather than more transient life circumstances that may lead to excessive clutter, such as inheriting property. The difficulty in discarding possessions noted in Criterion A refers to any form of discarding, including throwing away, selling, giving away, or recycling. The main reasons given for this difficulty are the perceived utility or aesthetic value of the items or strong sentimental attachment to the possessions. Some individuals feel responsible for the fate of their possessions and often go to great lengths to avoid being wasteful. Fears of losing important information are also common. The most commonly saved items are newspapers, magazines, clothing, bags, books, mail, and papers, but virtually any item can be saved. The nature of items is not limited to possessions that most other people would define as useless or of limited value. Many individuals collect and save large numbers of valuable things as well, which are often found in piles mixed with other less valuable items.

Individuals with hoarding disorder purposefully save possessions and experience distress (e.g., anxiety, frustration, regret, sadness, guilt) when facing the prospect of discarding them (Criterion B). This criterion emphasizes that the saving of possessions is intentional, which discriminates hoarding disorder from other forms of psychopathology that are characterized by the passive accumulation of items or the absence of distress when possessions are removed.

Individuals accumulate large numbers of items that fill up and clutter active living areas to the extent that their intended use is no longer possible (Criterion C). For example, the individual may not be able to cook in the kitchen, sleep in his or her bed, or sit in a chair. If the space can be used, it is only with great difficulty. *Clutter* is defined as a large group of usually unrelated or marginally related objects piled together in a disorganized fashion in spaces designed for other purposes (e.g., tabletops, floor, hallway). Criterion C emphasizes the “active” living areas of the home, rather than more peripheral areas, such as garages, attics, or basements, that are sometimes cluttered in homes of individuals without hoarding disorder.

However, individuals with hoarding disorder often have possessions that spill beyond the active living areas and can occupy and impair the use of other spaces, such as vehicles, yards, the workplace, and friends' and relatives' houses. In some cases, living areas may be uncluttered only because of the intervention of third parties (e.g., family members, cleaners, local authorities). Individuals who have been forced to clear their homes still have a symptom picture that meets criteria for hoarding disorder because the lack of clutter is attributable to a third-party intervention. Hoarding disorder contrasts with normative collecting behavior, which is organized and selective, although in some cases the actual amount of possessions may be similar to the amount accumulated by an individual with hoarding disorder. Normative collecting does not produce the clutter, distress, or impairment typical of hoarding disorder.

Symptoms (i.e., difficulty discarding and/or clutter) must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning, including maintaining a safe environment for self and others (Criterion D). In some cases, particularly when there is poor insight, the individual may not report distress, and the

impairment may be apparent only to those around the individual. However, any attempts to discard or clear the possessions by third parties result in high levels of distress.

Associated Features

Other common features of hoarding disorder include indecisiveness, perfectionism, avoidance, procrastination, difficulty planning and organizing tasks, and distractibility. Some individuals with hoarding disorder live in unsanitary conditions that may be a logical consequence of severely cluttered spaces and/or that are related to planning and organizing difficulties. *Animal hoarding* can be defined as the accumulation of a large number of animals and a failure to provide minimal standards of nutrition, sanitation, and veterinary care, as well as failure to act on the deteriorating condition of the animals (including disease, starvation, or death) and the

environment (e.g., severe overcrowding, extremely unsanitary conditions). Animal hoarding may be a special manifestation of hoarding disorder. Many individuals who hoard animals also hoard inanimate objects. The most prominent differences between animal and object hoarding are the extent of unsanitary conditions and the poorer insight in animal hoarding.

Prevalence

Nationally representative prevalence studies of hoarding disorder are not available. Community surveys estimate the point prevalence of clinically significant hoarding in the United States and Europe to range between 1.5% and 6%. In a meta-analysis of 12 studies across high-income countries, a prevalence of 2.5% was found, with no gender difference identified. This contrasts with clinical samples, which are predominantly women. In one population-based study in the Netherlands, hoarding symptoms appeared to be almost three times more prevalent in older adults (older than 65 years) compared with younger adults (ages 30–40 years).

Development and Course

Hoarding appears to begin early in life and spans well into the late stages. Hoarding symptoms may first emerge around ages 15–19 years, start interfering with the individual's everyday functioning by the mid-20s, and cause clinically significant impairment by the mid-30s. Participants in clinical research studies are usually in their 50s. Thus, the severity of hoarding increases with each decade of life, especially after age 30. Once symptoms begin, the course of hoarding is often chronic, with few individuals reporting a waxing and waning course.

Pathological hoarding in children appears to be easily distinguished from developmentally adaptive saving and collecting behaviors. Because children and adolescents typically do not control their living environment and discarding behaviors, the possible intervention of third parties (e.g., parents keeping the spaces usable and thus reducing interference) should be considered when making the diagnosis.

Risk and Prognostic Factors

Temperamental.

Indecisiveness is a prominent feature of individuals with hoarding disorder and their first-degree relatives.

Environmental. Individuals with hoarding disorder often retrospectively report stressful and traumatic life events preceding the onset of the disorder or causing an exacerbation.

Genetic and physiological. Hoarding behavior is familial; more than 50% of individuals who hoard report having a relative who also hoards. Twin studies indicate that approximately 50% of the variability in hoarding behavior is attributable to additive genetic factors and the rest to nonshared environmental factors.

Culture-Related Diagnostic Issues

While most of the research has been done in Western, industrialized countries and urban communities, the available data from low- and middle-income countries suggest that hoarding has consistent clinical features cross-culturally, including similarities in severity at clinical presentation and associated cognitions and behaviors. In cultural contexts in which a high value is placed on thrift and storing of possessions, the presence of distress and functional impairment should be the basis for the diagnosis.

Sex- and Gender-Related Diagnostic Issues

The key features of hoarding disorder (i.e., difficulty discarding, excessive amount of clutter) are generally comparable in men and women, but women tend to display more excessive acquisition, particularly excessive buying, than do men.

Functional Consequences of Hoarding Disorder

Clutter impairs basic activities, such as moving through the house, cooking, cleaning, maintaining personal hygiene, and even sleeping. Appliances may be broken, and utilities such as water and electricity may be disconnected, as access for repair work may be difficult. Quality of life is often considerably impaired. In severe cases, hoarding can put individuals at risk

for fire, falling (especially elderly individuals), poor sanitation, and other health risks. Hoarding disorder is associated with occupational impairment, poor physical health, and high social service utilization. Family relationships are frequently under great strain. Conflict with neighbors and local authorities is common, and a substantial proportion of individuals with severe hoarding disorder have been involved in legal eviction proceedings, and some have a history of eviction.

Differential Diagnosis

Other medical conditions. Hoarding disorder is not diagnosed if the symptoms are judged to be a direct consequence of another medical condition (Criterion E), such as traumatic brain injury, surgical resection for treatment of a tumor or seizure control, cerebrovascular disease, infections of the central nervous system (e.g., herpes simplex encephalitis), or neurogenetic conditions such as Prader-Willi syndrome. Damage to the anterior ventromedial prefrontal and cingulate cortices has been particularly associated with the excessive accumulation of objects. In these individuals, the hoarding behavior is not present prior to the onset of the brain damage and appears shortly after the brain damage occurs. Some of these individuals appear to have little interest in the accumulated items and are able to discard them easily or do not care if others discard them, whereas others appear to be very reluctant to discard anything.

Neurodevelopmental disorders. Hoarding disorder is not diagnosed if the accumulation of objects is judged to be a direct consequence of a neurodevelopmental disorder, such as autism spectrum disorder or intellectual developmental disorder (intellectual disability).

Schizophrenia spectrum and other psychotic disorders. Hoarding disorder is not diagnosed if the accumulation of objects is judged to be a direct consequence of delusions or negative symptoms in schizophrenia spectrum and other psychotic disorders.

Major depressive episode. Hoarding disorder is not diagnosed if the accumulation of objects is judged to be a direct consequence of psychomotor retardation, fatigue, or loss of energy during a major depressive episode.

Obsessive-compulsive disorder. Hoarding disorder is not diagnosed if the symptoms are judged to be a direct consequence of typical obsessions or compulsions, such as fears of contamination, harm, or feelings of incompleteness in obsessive-compulsive disorder

(OCD). Feelings of incompleteness (e.g., losing one's identity, or having to document and preserve all life experiences) are the most frequent OCD symptoms associated with this form of hoarding. The accumulation of objects can also be the result of persistently avoiding onerous rituals (e.g., not discarding objects in order to avoid endless washing or checking rituals).

In OCD, the behavior is generally unwanted and highly distressing, and the individual experiences no pleasure or reward from it. Excessive acquisition is usually not present; if excessive acquisition is present, items are acquired because of a specific obsession (e.g., the need to buy items that have been accidentally touched in order to avoid contaminating other people), not because of a genuine desire to possess the items. Individuals who hoard in the context of OCD are also more likely to accumulate bizarre items, such as trash, feces, urine, fingernail and toenail cuttings, hair, used diapers, or rotten food. Accumulation of such items is very unusual in hoarding disorder.

When severe hoarding appears concurrently with other typical symptoms of OCD but is judged to be independent from these symptoms, both hoarding disorder and OCD may be diagnosed.

Neurocognitive disorders. Hoarding disorder is not diagnosed if the accumulation of objects is judged to be a direct consequence of a degenerative disorder, such as neurocognitive disorder associated with frontotemporal degeneration or Alzheimer's disease. Typically, onset of the accumulating behavior is gradual and follows onset of the neurocognitive disorder. The accumulating behavior may be accompanied by self-neglect and severe domestic squalor, alongside other neuropsychiatric symptoms, such as disinhibition, gambling, rituals/stereotypies, tics, and self-injurious behaviors.

Comorbidity

Approximately 75% of individuals with hoarding disorder have a comorbid mood or anxiety disorder. The most common comorbid conditions are major depressive disorder (30%–50%), social anxiety disorder, and generalized anxiety disorder. Approximately 20% of individuals with hoarding disorder also have symptoms that meet diagnostic criteria for OCD. These comorbidities may often be the main reason for consultation, because individuals are unlikely to spontaneously report hoarding symptoms, and hoarding symptoms are often not asked about in routine clinical interviews.

Trichotillomania (Hair-Pulling Disorder)

Diagnostic Criteria F63.3

A. Recurrent pulling out of one's hair, resulting in hair loss.
B. Repeated attempts to decrease or stop hair pulling.
C. The hair pulling causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
D. The hair pulling or hair loss is not attributable to another medical condition (e.g., a dermatological condition).
E. The hair pulling is not better explained by the symptoms of another mental disorder (e.g., attempts to improve a perceived defect or flaw in appearance in body dysmorphic disorder).

Diagnostic Features

The essential feature of trichotillomania (hair-pulling disorder) is the recurrent pulling out of one's own hair (Criterion A). Hair pulling may occur from any region of the body in

which hair grows; the most common sites are the scalp, eyebrows, and eyelids, while less common sites are axillary, facial, pubic, and perirectal regions. Hair-pulling sites may vary over time. Hair pulling may occur in brief episodes scattered throughout the day or during less frequent but more sustained periods that can continue for hours, and such hair pulling may endure for months or years. Criterion A requires that hair pulling lead to hair loss, although individuals with this disorder may pull hair in a widely distributed pattern (i.e., pulling single hairs from all over a site) such that hair loss may not be clearly visible. In addition, individuals may attempt to conceal or camouflage hair loss (e.g., by using makeup, scarves, or wigs). Individuals with trichotillomania have made repeated attempts to decrease or stop hair pulling (Criterion B). Criterion C indicates that hair pulling causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. The term *distress* includes negative affects that may be experienced by individuals with hair pulling, such as feeling a loss of control, embarrassment, and shame. Significant impairment may occur in several different areas of functioning (e.g., social, occupational, academic, and leisure), in part because of avoidance of work, school, or other public situations.

Associated Features

Hair pulling may be accompanied by a range of behaviors or rituals involving hair. Thus, individuals may search for a particular kind of hair to pull (e.g., hairs with a specific texture or color), may try to pull out hair in a specific way (e.g., so that the root comes out intact), or may visually examine or tactilely or orally manipulate the hair after it has been pulled (e.g., rolling the hair between the fingers, pulling the strand between the teeth, biting the hair into pieces, or swallowing the hair).

Hair pulling may also be preceded or accompanied by various emotional states; it may be triggered by feelings of anxiety or boredom, may be preceded by an increasing sense of tension (either immediately before pulling out the hair or when attempting to resist the urge to pull), or may lead to gratification, pleasure, or a sense of relief when the hair is

pulled out. Hair-pulling behavior may involve varying degrees of conscious awareness, with some individuals displaying more focused attention on the hair pulling (with preceding tension and subsequent relief), and other individuals displaying more automatic behavior (in which the hair pulling seems to occur without full awareness). Many individuals report a mix of both behavioral styles. Some individuals experience an “itch-like” or tingling sensation in the scalp that is alleviated by the act of pulling hair. Pain does not usually accompany hair pulling.

Patterns of hair loss are highly variable. Areas of complete alopecia, as well as areas of thinned hair density, are common. When the scalp is involved, there may be a predilection for pulling out hair in the crown or parietal regions. There may be a pattern of nearly complete baldness except for a narrow perimeter around the outer margins of the scalp, particularly at the nape of the neck (“tonsure trichotillomania”). Eyebrows and eyelashes may be completely absent.

Hair pulling does not usually occur in the presence of other individuals, except immediate family members. Some individuals have urges to pull hair from other individuals and may sometimes try to find opportunities to do so surreptitiously. Some individuals may pull hairs from pets, dolls, and other fibrous materials (e.g., sweaters or carpets). Some individuals may deny their hair pulling to others. The majority of individuals with trichotillomania also have one or more other body-focused repetitive behaviors, including skin picking, nail biting, and lip chewing.

Prevalence

In the general population, data from nonrepresentative U.S. samples have suggested that the 12-month prevalence estimate for trichotillomania in adults and adolescents may be in

the range of 1% to 2%. Women are more frequently affected than men in self-identified or clinical samples, at a ratio estimated at 10:1 or greater, but the gender ratio may be closer to 2:1 in community samples. Among children with trichotillomania, boys and girls are more equally represented.

An online survey of more than 10,000 adults ages 18–69 years, representative of the general U.S. population, found that 1.7% identified as having current trichotillomania and that rates did not differ significantly based on gender (1.8% of men and 1.7% of women).

Development and Course

Hair pulling may be seen in infants, and this behavior typically resolves during early development. Onset of hair pulling in trichotillomania most commonly coincides with, or follows the onset of, puberty. Sites of hair pulling may vary over time. The usual course of trichotillomania is chronic, with some waxing and waning if the disorder is untreated. Symptoms may worsen in females premenstrually but not consistently during pregnancy. For some individuals, the disorder may come and go for weeks, months, or years at a time. A minority of individuals remit without subsequent relapse within a few years of onset.

Risk and Prognostic Factors

Genetic and physiological. There is evidence for a genetic vulnerability to trichotillomania. The disorder is more common in individuals with obsessive-compulsive disorder (OCD) and their first-degree relatives than in the general population.

Culture-Related Diagnostic Issues

Trichotillomania appears to manifest similarly across cultures and ethnicities, although there is a paucity of data from non-Western regions.

Diagnostic Markers

Most individuals with trichotillomania admit to hair pulling; thus, dermatopathological diagnosis is rarely required. Skin biopsy and dermoscopy (or trichoscopy) of trichotillomania are able to differentiate the disorder from other causes of alopecia. In trichotillomania, dermoscopy shows a range of characteristic features, including decreased hair density, short vellus hair, and broken hairs with different shaft lengths.

Functional Consequences of Trichotillomania (Hair-Pulling Disorder)

Trichotillomania is associated with distress as well as with social and occupational impairment. There may be irreversible damage to hair growth and hair quality. Infrequent medical consequences of trichotillomania include digit purpura, musculoskeletal injury (e.g., carpal tunnel syndrome; back, shoulder and neck pain), blepharitis, and dental damage (e.g., worn or broken teeth resulting from hair biting). Swallowing of hair (trichophagia) may lead to trichobezoars, with subsequent anemia, abdominal pain, hematemesis, nausea and vomiting, bowel obstruction, and even bowel perforation.

Differential Diagnosis

Normative hair removal/manipulation. Trichotillomania should not be diagnosed when hair removal is performed solely for cosmetic reasons (i.e., to improve physical appearance). Many individuals twist and play with their hair, but this behavior does not usually qualify for a diagnosis of trichotillomania. Some individuals may bite rather than pull hair; again, this does not qualify for a diagnosis of trichotillomania.

Other obsessive-compulsive and related disorders. Individuals with OCD and symmetry concerns may pull out hairs as part of their symmetry rituals, and individuals with body dysmorphic disorder may remove body hair that they perceive as ugly, asymmetrical, or abnormal; in such cases a diagnosis of trichotillomania is not given.

Stereotypic movement disorder. Stereotypic movement disorder can sometimes involve hair-pulling behavior. For example, a child with intellectual developmental disorder (intellectual disability) or autism spectrum disorder may engage in stereotypic head banging, hand or arm biting, and hair pulling when frustrated or angry, and sometimes when excited. This behavior, if impairing, would be diagnosed as stereotypic movement disorder (co-occurring with intellectual developmental disorder or autism spectrum disorder) rather than trichotillomania.

Psychotic disorder. Individuals with a psychotic disorder may remove hair in response to a delusion or hallucination. Trichotillomania is not diagnosed in such cases.

Another medical condition. Trichotillomania is not diagnosed if the hair pulling or hair loss is attributable to another medical condition (e.g., inflammation of the skin or other dermatological conditions). Other causes of noncicatricial (nonscarring) alopecia (e.g., alopecia areata, androgenic alopecia, telogen effluvium) or cicatricial (scarring) (e.g., chronic discoid lupus erythematosus, lichen planopilaris, central centrifugal cicatricial alopecia, pseudopelade, folliculitis decalvans, dissecting folliculitis, acne keloidalis nuchae) should be considered in individuals with hair loss who deny hair pulling. Skin biopsy or dermoscopy can be used to differentiate individuals with trichotillomania from those with dermatological disorders.

Substance-related disorders. Hair-pulling symptoms may be exacerbated by certain substances—for example, stimulants—but it is less likely that substances are the primary cause of persistent hair pulling.

Comorbidity

Trichotillomania is often accompanied by other mental disorders, most commonly major depressive disorder and excoriation (skin-picking) disorder. Repetitive body-focused symptoms other than hair pulling or skin picking (e.g., nail biting) occur in the majority of individuals with trichotillomania and may deserve an additional diagnosis of other specified obsessive-compulsive and related disorder (i.e., other body-focused repetitive behavior disorder).

Excoriation (Skin-Picking) Disorder

Diagnostic Criteria F42.4

A. Recurrent skin picking resulting in skin lesions.
B. Repeated attempts to decrease or stop skin picking.

C. The skin picking causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The skin picking is not attributable to the physiological effects of a substance (e.g., cocaine) or another medical condition (e.g., scabies).

E. The skin picking is not better explained by symptoms of another mental disorder (e.g., delusions or tactile hallucinations in a psychotic disorder, attempts to improve a perceived defect or flaw in appearance in body dysmorphic disorder, stereotypies in stereotypic movement disorder, or intention to harm oneself in nonsuicidal self-injury).

Diagnostic Features

The essential feature of excoriation (skin-picking) disorder is recurrent picking at one's own skin (Criterion A). The most commonly picked sites are the face, arms, and hands, but many individuals pick from multiple body sites. Individuals may pick at healthy skin, at minor skin irregularities, at lesions such as pimples or calluses, or at scabs from previous picking. Most individuals pick with their fingernails, although many use tweezers, pins, or other objects. In addition to skin picking, there may be skin rubbing, squeezing, lancing, and biting. Individuals with excoriation disorder often spend significant amounts of time on their picking behavior, sometimes several hours per day, and such skin picking may endure for months or years. Criterion A requires that skin picking lead to skin lesions, although individuals with this disorder often attempt to conceal or camouflage such lesions (e.g., with makeup or clothing). Individuals with excoriation disorder have made repeated attempts to decrease or stop skin picking (Criterion B).

Criterion C indicates that skin picking causes clinically significant distress or impairment in social, occupational, or other important areas of functioning. The term *distress* includes negative affects that may be

experienced by individuals with skin picking, such as feeling a loss of control, embarrassment, and shame. Significant impairment may occur in several different areas of functioning (e.g., social, occupational, academic, and leisure), in part because of avoidance of social situations.

Associated Features

Skin picking may be accompanied by a range of behaviors or rituals involving skin or scabs. Thus, individuals may search for a particular kind of scab to pull, and they may examine, play with, or mouth or swallow the skin after it has been pulled. Skin picking may also be preceded or accompanied by various emotional states. Skin picking may be triggered by feelings of anxiety or boredom, may be preceded by an increasing sense of tension (either immediately before picking the skin or when attempting to resist the urge to pick), and may lead to gratification, pleasure, or a sense of relief when the skin or scab has been picked. Some individuals report picking in response to a minor skin irregularity or to relieve an uncomfortable bodily sensation. Pain is not routinely reported to accompany skin picking. Some individuals engage in skin picking that is more focused (i.e., with preceding tension and subsequent relief), whereas others engage in more automatic picking (i.e., when skin picking occurs without preceding tension and without full awareness), and many have a mix of both behavioral styles. Skin picking does not usually occur in the presence of other individuals, except immediate family members. Some individuals report picking the skin of others.

Prevalence

An online survey of more than 10,000 adults, ages 18–69 years, age- and gender-matched to the U.S. population, found that 2.1% identified as having current excoriation disorder and 3.1% reported lifetime excoriation disorder. Three-quarters or more of individuals with the disorder are women in community samples.

Development and Course

Although individuals with excoriation disorder may present at various ages, the skin picking most often has onset during adolescence, commonly coinciding with or following the onset of puberty. The disorder frequently begins with a dermatological condition, such as acne. Sites of skin picking may vary over time. The usual course is chronic, with some waxing and waning if untreated. For some individuals, the disorder may come and go for weeks, months, or years at a time.

Risk and Prognostic Factors

Genetic and physiological. There is evidence for a genetic vulnerability to excoriation disorder. The disorder is more common in individuals with obsessive-compulsive disorder (OCD) and their first-degree family members than in the general population.

Culture-Related Diagnostic Issues

There are limited data on the prevalence and clinical characteristics of excoriation disorder across cultures. However, clinical features appear similar in studies of individuals in the United States and other countries.

Diagnostic Markers

Most individuals with excoriation disorder admit to skin picking; therefore, dermatopathological diagnosis is rarely required. However, the disorder may have characteristic features on histopathology.

Functional Consequences of Excoriation (Skin-Picking) Disorder

Excoriation disorder is associated with social and occupational impairment. The majority of individuals with this condition spend at least 1 hour per day picking, thinking about picking, and resisting urges to pick. Many individuals report avoiding social or entertainment events as well as going out in public. A majority of individuals with the disorder also report experiencing work interference from skin picking on at least a daily or

weekly basis. A significant proportion of students with excoriation disorder report having missed school, having experienced difficulties managing responsibilities at school, or having had difficulties studying because of skin picking. Medical complications of skin picking include tissue damage, scarring, and infection and can be life-threatening. Rarely, synovitis of the wrists resulting from chronic picking has been reported. Skin picking often results in significant tissue damage and scarring. It frequently requires antibiotic treatment for infection, and on occasion it may require surgery.

Differential Diagnosis

Psychotic disorder. Skin picking may occur in response to a delusion (i.e., parasitosis) or tactile hallucination (i.e., formication) in a psychotic disorder. In such cases, excoriation disorder should not be diagnosed.

Other obsessive-compulsive and related disorders. Excessive washing compulsions in response to contamination obsessions in individuals with OCD may lead to skin lesions, and skin picking may occur in individuals with body dysmorphic disorder who pick their skin because of appearance concerns; in such cases, excoriation disorder should not be diagnosed. The description of other body-focused repetitive behavior disorder in other specified obsessive-compulsive and related disorder excludes individuals whose symptoms meet diagnostic criteria for excoriation disorder.

Neurodevelopmental disorders. While stereotypic movement disorder may be characterized by repetitive self-injurious behavior, onset is in the early developmental period. For example, individuals with the neurogenetic condition Prader-Willi syndrome may have early onset of skin picking, and their symptoms may meet criteria for stereotypic movement disorder. While tics in individuals with Tourette's disorder may lead to self-injury, the behavior is not tic-like in excoriation disorder.

Dermatitis artefacta. Dermatitis artefacta (also referred to as "factitious dermatitis") is a term used in dermatology to refer to medically unexplained, presumably self-induced skin lesions that the individual denies any role in creating. Cases in which there is evidence of deception on the

individual's part concerning the skin lesions can be diagnosed as either malingering (if the skin picking is motivated by external incentives) or factitious disorder (if the skin picking occurs in the absence of obvious external rewards). In the absence of deception, excoriation disorder can be diagnosed if there are repeated attempts to decrease or stop skin picking.

Other disorders. Excoriation disorder is not diagnosed if the skin picking is primarily attributable to the intention to harm oneself that is characteristic of nonsuicidal self-injury.

Other medical conditions. Excoriation disorder is not diagnosed if the skin picking is primarily attributable to another medical condition. For example, scabies is a dermatological condition invariably associated with severe itching and scratching. However, excoriation disorder may be precipitated or exacerbated by an underlying dermatological condition. For example, acne may lead to some scratching and picking, which may also be associated with comorbid excoriation disorder (so-called acne excoriée). The differentiation between these two clinical situations (acne with some scratching and picking vs. acne with comorbid excoriation disorder) requires an assessment of the extent to which the individual's skin picking has become independent of the underlying dermatological condition.

Substance/medication-induced disorders. Skin-picking symptoms may also be induced by certain substances (e.g., cocaine), in which case excoriation disorder should not be diagnosed. If such skin picking is clinically significant, then a diagnosis of substance/medication-induced obsessive-compulsive and related disorder should be considered.

Comorbidity

Excoriation disorder is often accompanied by other mental disorders. Such disorders include OCD and trichotillomania (hair-pulling disorder), as well as major depressive disorder. Depression comorbidity seems to be more common in women. Repetitive body-focused symptoms other than skin picking and hair pulling (e.g., nail biting) occur in many individuals with excoriation disorder and may deserve an additional diagnosis of other specified obsessive-compulsive and related disorder (i.e., other body-focused repetitive behavior disorder).

Substance/Medication-Induced Obsessive-Compulsive and Related Disorder

Diagnostic Criteria

A. Obsessions, compulsions, skin picking, hair pulling, other body-focused repetitive behaviors, or other symptoms characteristic of the obsessive-compulsive and related disorders predominate in the clinical picture.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by an obsessive-compulsive and related disorder that is not substance/medication-induced. Such evidence of an independent obsessive-compulsive and related disorder could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced obsessive-compulsive and related disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made in addition to a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance/medication]-induced obsessive-compulsive and related disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given. If a mild substance use disorder is comorbid with the substance-induced obsessive-compulsive and related disorder, the 4th position character is “1,” and the clinician should record “mild [substance] use disorder” before the substance-induced obsessive-compulsive and related disorder (e.g., “mild cocaine use disorder with cocaine-induced obsessive-compulsive and related disorder”). If a moderate or severe substance use disorder is comorbid with the substance-induced obsessive-compulsive and related disorder, the 4th position character is “2,” and the clinician should record “moderate [substance] use disorder” or “severe [substance] use disorder,” depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is “9,” and the clinician should record only the substance-induced obsessive-compulsive and related disorder.

	ICD-10-CM

	ICD-10-CM		
	With mild use disorder	**With moderate or severe use disorder**	**Without use disorder**
Amphetamine-type substance (or other stimulant)	F15.188	F15.288	F15.988
Cocaine	F14.188	F14.288	F14.988
Other (or unknown) substance	F19.188	F19.288	F19.988

Specify (see Table 1 in the chapter "Substance-Related and Addictive Disorders," which indicates whether "with onset during intoxication" and/or "with onset during withdrawal" applies to a given substance class; or *specify* "with onset after medication use"):

With onset during intoxication: If criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: If symptoms developed at initiation of medication, with a change in use of medication, or during withdrawal of medication.

Recording Procedures

The name of the substance/medication-induced obsessive-compulsive and related disorder begins with the specific substance (e.g., cocaine) that is presumed to be causing the obsessive-compulsive and related symptoms. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., ropinirole), the code for "other (or unknown) substance" should be

used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the same code should also be used.

To record the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by "with substance/medication-induced obsessive-compulsive and related disorder" (incorporating the name of the specific etiological substance/medication), followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal, with onset after medication use). For example, in the case of repetitive skin-picking occurring during intoxication in a man with a severe cocaine use disorder, the diagnosis is F14.288 severe cocaine use disorder with cocaine-induced obsessive-compulsive and related disorder, with onset during intoxication. A separate diagnosis of the comorbid severe cocaine use disorder is not given. If the substance-induced obsessive-compulsive and related disorder occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F15.988 amphetamine-induced obsessive-compulsive and related disorder, with onset during intoxication). When more than one substance is judged to play a significant role in the development of the obsessive-compulsive and related disorder, each should be listed separately.

Diagnostic Features

The essential features of substance/medication-induced obsessive-compulsive and related disorder are prominent symptoms of an obsessive-compulsive and related disorder (Criterion A) that are judged to be attributable to the effects of a substance (e.g., drug of abuse, medication). The obsessive-compulsive and related disorder symptoms must have developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication or toxin, and the substance/medication must be capable of producing the symptoms (Criterion B). Substance/medication-induced obsessive-compulsive and related disorder due to a prescribed treatment for a mental disorder or other medical condition must have its onset while the individual is receiving the medication. Once the treatment is discontinued, the obsessive-compulsive and related disorder symptoms will usually improve or remit within days to

weeks (depending on the half-life of the substance/medication and the presence of withdrawal). The diagnosis of substance/medication-induced obsessive-compulsive and related disorder should not be given if onset of the obsessive-compulsive and related disorder symptoms precedes the substance/medication use, or if the symptoms persist for a substantial period of time, usually longer than 1 month, from the time of severe intoxication or withdrawal. The substance/medication-induced obsessive-compulsive and related disorder diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and are sufficiently severe to warrant independent clinical attention.

Associated Features

Obsessions, compulsions, hair pulling, skin picking, or other body-focused repetitive behaviors can occur in association with intoxication with the following classes of substances: stimulants (including cocaine) and other (or unknown) substances. Heavy metals and toxins may also cause obsessive-compulsive and related disorder symptoms.

Prevalence

In the U.S. general population, the very limited data that are available indicate that substance/medication-induced obsessive-compulsive and related disorder is very rare.

Differential Diagnosis

Substance intoxication and substance withdrawal. Obsessive-compulsive and related disorder symptoms may occur in the context of substance intoxication and substance withdrawal. The diagnosis of the substance-specific intoxication or substance-specific withdrawal will usually suffice to categorize the symptom presentation. A diagnosis of substance/medication-induced obsessive-compulsive and related disorder either with onset during intoxication or with onset during withdrawal should be made instead of a diagnosis of substance intoxication or substance withdrawal if the

obsessive-compulsive and related disorder symptoms are judged to be in excess of those usually associated with intoxication or withdrawal and are sufficiently severe to warrant clinical attention.

Obsessive-compulsive and related disorder (i.e., not induced by a substance). Substance/medication-induced obsessive-compulsive and related disorder is distinguished from a primary obsessive-compulsive and related disorder by considering the onset, course, and other factors with respect to substances/medications. For drugs of abuse, there must be evidence from the history, physical examination, or laboratory findings for use or intoxication. Substance/medication-induced obsessive-compulsive and related disorder arises only in association with intoxication, whereas a primary obsessive-compulsive and related disorder may precede the onset of substance/medication use. The presence of features that are atypical of a primary obsessive-compulsive and related disorder, such as atypical age at onset of symptoms, may suggest a substance-induced etiology. A primary obsessive-compulsive and related disorder diagnosis is warranted if the symptoms persist for a substantial period of time (about 1 month or longer) after the end of the substance intoxication or the individual has a history of an obsessive-compulsive and related disorder.

Obsessive-compulsive and related disorder due to another medical condition. If the obsessive-compulsive and related symptoms are attributable to another medical condition (i.e., rather than to the medication taken for the other medical condition), obsessive-compulsive and related disorder due to another medical condition should be diagnosed. The history often provides the basis for judgment. At times, a change in the treatment for the other medical condition (e.g., medication substitution or discontinuation) may be needed to determine whether the medication is the causative agent (in which case the symptoms may be better explained by substance/medication-induced obsessive-compulsive and related disorder). If the disturbance is attributable to both another medical condition and substance use, both diagnoses (i.e., obsessive-compulsive and related disorder due to another medical condition and substance/medication-induced obsessive-compulsive and related disorder) may be given. When there is insufficient evidence to determine whether the symptoms are attributable to a substance/medication or to another medical condition or are primary (i.e., attributable to neither a

substance/medication nor another medical condition), a diagnosis of other specified or unspecified obsessive-compulsive and related disorder would be indicated.

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Delirium. If obsessive-compulsive and related disorder symptoms occur exclusively during the course of delirium, they are considered to be an associated feature of the delirium and are not diagnosed separately.

Obsessive-Compulsive and Related Disorder Due to Another Medical Condition

Diagnostic Criteria F06.8

A. Obsessions, compulsions, preoccupations with appearance, hoarding, skin picking, hair pulling, other body-focused repetitive behaviors, or other symptoms characteristic of obsessive-compulsive and related disorder predominate in the clinical picture.
B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.
C. The disturbance is not better explained by another mental disorder.
D. The disturbance does not occur exclusively during the course of a delirium.
E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

With obsessive-compulsive disorder–like symptoms: If obsessive-compulsive disorder–like symptoms predominate in the clinical presentation.

With appearance preoccupations: If preoccupation with perceived appearance defects or flaws predominates in the clinical presentation.

With hoarding symptoms: If hoarding predominates in the clinical presentation.

With hair-pulling symptoms: If hair pulling predominates in the clinical presentation.

With skin-picking symptoms: If skin picking predominates in the clinical presentation.

Coding note: Include the name of the other medical condition in the name of the mental disorder (e.g., F06.8 obsessive-compulsive and related disorder due to cerebral infarction). The other medical condition should be coded and listed separately immediately before the obsessive-compulsive and related disorder due to the medical condition (e.g., I69.398 cerebral infarction; F06.8 obsessive-compulsive and related disorder due to cerebral infarction).

Diagnostic Features

The essential feature of obsessive-compulsive and related disorder due to another medical condition is clinically significant obsessive-compulsive and related symptoms that are judged to be best explained as the direct pathophysiological consequence of another medical condition. Symptoms can include prominent obsessions, compulsions, preoccupations with appearance, hoarding, hair pulling, skin picking, or other body-focused repetitive behaviors (Criterion A). The judgment that the symptoms are best explained by the associated medical condition must be based on evidence from the history, physical examination, or laboratory findings (Criterion B). Additionally, it must be judged that the symptoms are not better explained by another mental disorder (Criterion C). The diagnosis is not made if the

obsessive-compulsive and related symptoms occur only during the course of a delirium (Criterion D). The obsessive-compulsive and related symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E).

In determining whether the obsessive-compulsive and related symptoms are attributable to another medical condition, a medical condition must be present at the time of the onset of the obsessive-compulsive and related symptoms. Furthermore, it must be established that obsessive-compulsive and related symptoms can be etiologically related to the medical condition through a pathophysiological mechanism and that this best explains the symptoms in the individual. Although there are no infallible guidelines for determining whether the relationship between the obsessive-compulsive and related symptoms and the medical condition is etiological, considerations that may provide some guidance in making this diagnosis include the presence of a clear temporal association between the onset, exacerbation, or remission of the medical condition and the obsessive-compulsive and related symptoms; the presence of features that are atypical of a primary obsessive-compulsive and related disorder (e.g., atypical age at onset or course); and evidence in the literature that a known physiological mechanism (e.g., striatal damage due to a cerebral infarction) causes obsessive-compulsive and related symptoms. In addition, the disturbance cannot be better explained by a primary obsessive-compulsive and related disorder, a substance/medication-induced obsessive-compulsive and related disorder, or another mental disorder.

There has been considerable attention to the question of whether obsessive-compulsive and related disorders can be attributed to Group A streptococcal infection. Sydenham’s chorea is the neurological manifestation of rheumatic fever, which is in turn due to Group A streptococcal infection. Sydenham’s chorea is characterized by a combination of motor and nonmotor symptoms. Nonmotor features include obsessions, compulsions, attention deficit, and emotional lability. Although individuals with Sydenham’s chorea may present with non-neuropsychiatric features of acute rheumatic fever, such as carditis and arthritis, they may

present with obsessive-compulsive disorder–like symptoms; such individuals should be diagnosed with obsessive-compulsive and related disorder due to another medical condition.

Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS) has been identified as another postinfectious autoimmune disorder characterized by the sudden onset of obsessions, compulsions, and/or tics accompanied by a variety of acute neuropsychiatric symptoms in the absence of chorea, carditis, or arthritis, after Group A streptococcal infection. However, given that such acute-onset symptoms may be due to a range of other infections or insults, the term *pediatric acute-onset neuropsychiatric syndrome* (PANS) has been used. PANS is characterized by abrupt, dramatic onset of obsessive-compulsive symptoms or severely restricted food intake, together with a range of additional neuropsychiatric symptoms. Assessment guidelines for this syndrome are available.

Associated Features

A number of other medical conditions are known to include obsessive-compulsive and related symptoms as a manifestation. Examples include disorders leading to striatal damage, such as cerebral infarction or Huntington's disorder.

Development and Course

The development and course of obsessive-compulsive and related disorder due to another medical condition generally follows the course of the underlying illness.

Diagnostic Markers

Laboratory assessments and/or medical examinations are necessary to confirm the diagnosis of another medical condition.

Differential Diagnosis

Delirium. A separate diagnosis of obsessive-compulsive and related disorder due to another medical condition is not given if the disturbance occurs

exclusively during the course of a delirium. However, a diagnosis of obsessive-compulsive and related disorder due to

another medical condition may be given in addition to a diagnosis of major neurocognitive disorder (dementia) if the etiology of the obsessive-compulsive symptoms is judged to be a physiological consequence of the pathological process causing the dementia and if obsessive-compulsive symptoms are a prominent part of the clinical presentation.

Mixed presentation of symptoms (e.g., mood and obsessive-compulsive and related symptoms) judged to be due to another medical condition. If the presentation includes a mix of different types of symptoms, the specific mental disorder due to another medical condition depends on which symptoms predominate in the clinical picture.

Substance/medication-induced obsessive-compulsive and related disorders. If there is evidence of recent or prolonged substance use (including medications with psychoactive effects), withdrawal from a substance, or exposure to a toxin, a substance/medication-induced obsessive-compulsive and related disorder should be considered. When a substance/medication-induced obsessive-compulsive and related disorder is being diagnosed in relation to drugs of abuse, it may be useful to obtain a urine or blood drug screen or other appropriate laboratory evaluation. Symptoms that occur during or shortly after (i.e., within 4 weeks of) substance intoxication or withdrawal or after medication use may be especially indicative of a substance/medication-induced obsessive-compulsive and related disorder, depending on the type, duration, or amount of the substance used.

Obsessive-compulsive and related disorders (primary). Obsessive-compulsive and related disorder due to another medical condition should be distinguished from a primary obsessive-compulsive and related disorder. In primary mental disorders, no specific and direct causative physiological mechanisms associated with a medical condition can be demonstrated. Acute-onset symptoms, late age at onset, or atypical symptoms suggest the need for a thorough assessment to rule out the diagnosis of obsessive-compulsive and related disorder due to another medical condition.

Illness anxiety disorder.

Illness anxiety disorder is characterized by a preoccupation with having or acquiring a serious illness. In the case of illness anxiety disorder, individuals may or may not have diagnosed medical conditions.

Associated feature of another mental disorder. Obsessive-compulsive and related symptoms may be an associated feature of another mental disorder (e.g., schizophrenia, anorexia nervosa).

Other specified obsessive-compulsive and related disorder or unspecified obsessive-compulsive and related disorder. These diagnoses are given if it is unclear whether the obsessive-compulsive and related symptoms are primary, substance-induced, or due to another medical condition.

Other Specified Obsessive-Compulsive and Related Disorder

F42.8

This category applies to presentations in which symptoms characteristic of an obsessive-compulsive and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the obsessive-compulsive and related disorders diagnostic class. The other specified obsessive-compulsive and related disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific obsessive-compulsive and related disorder. This is done by recording “other specified obsessive-compulsive and related disorder” followed by the specific reason (e.g., “obsessional jealousy”).

Examples of presentations that can be specified using the “other specified” designation include the following:

1. **Body dysmorphic–like disorder with actual flaws:** This is similar to body dysmorphic disorder except that the defects or flaws in physical appearance are clearly observable by others (i.e., they are more noticeable than “slight”). In such cases, the preoccupation with these flaws is clearly excessive and causes significant impairment or distress.
2. **Body dysmorphic–like disorder without repetitive behaviors:** Presentations that meet body dysmorphic disorder except that the individual has never performed repetitive behaviors or mental acts in response to the appearance concerns.
3. **Other body-focused repetitive behavior disorder:** Presentations involving recurrent body-focused repetitive behaviors other than hair pulling and skin picking (e.g., nail biting, lip biting, cheek chewing) that are accompanied by repeated attempts to decrease or stop the behaviors and that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
4. **Obsessional jealousy:** This is characterized by nondelusional preoccupation with a partner’s perceived infidelity. The preoccupations may lead to repetitive behaviors or mental acts in response to the infidelity concerns; they cause clinically significant distress or impairment in social, occupational, or other important areas of functioning; and they are not better explained by another mental disorder such as delusional disorder, jealous type, or paranoid personality disorder.
5. **Olfactory reference disorder (olfactory reference syndrome):** This is characterized by the individual’s persistent preoccupation with the belief that he or she emits a foul or offensive body odor that is unnoticeable or only slightly noticeable to others; in response to this preoccupation, these individuals often engage in repetitive and excessive behaviors such as repeatedly checking for body odor, excessive showering, or seeking reassurance, as well as excessive attempts to camouflage the perceived odor. These symptoms

cause clinically significant distress or impairment in social, occupational, or other important areas of functioning. In traditional Japanese psychiatry, this disorder is known as *jikoshu-kyofu*, a variant of *taijin kyofusho* (see "Culture and Psychiatric Diagnosis" in Section III).

6. ***Shubo-kyofu:*** A variant of *taijin kyofusho* (see "Culture and Psychiatric Diagnosis" in Section III) that is similar to body dysmorphic disorder and is characterized by excessive fear of having a bodily deformity.
7. ***Koro:*** Related to *dhat syndrome* (see "Culture and Psychiatric Diagnosis" in Section III), an episode of sudden and intense anxiety that the penis in males (or the vulva and nipples in females) will recede into the body, possibly leading to death.

Unspecified Obsessive-Compulsive and Related Disorder

F42.9

This category applies to presentations in which symptoms characteristic of an obsessive-compulsive and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the obsessive-compulsive and related disorders diagnostic class. The unspecified obsessive-compulsive and related disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific obsessive-compulsive and related disorder and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Trauma- and Stressor-Related Disorders

Trauma- and stressor-related disorders include disorders in which exposure to a traumatic or stressful event is listed explicitly as a diagnostic criterion. These include reactive attachment disorder, disinhibited social engagement disorder, posttraumatic stress disorder (PTSD), acute stress disorder, adjustment disorders, and prolonged grief disorder. Placement of this chapter reflects the close relationship between these diagnoses and disorders in the surrounding chapters on anxiety disorders, obsessive-compulsive and related disorders, and dissociative disorders.

Psychological distress following exposure to a traumatic or stressful event is quite variable. In some cases, symptoms can be well understood within an anxiety- or fear-based context. It is clear, however, that many individuals who have been exposed to a traumatic or stressful event exhibit a phenotype in which, rather than anxiety- or fear-based symptoms, the most prominent clinical characteristics are anhedonic and dysphoric symptoms, externalizing angry and aggressive symptoms, or dissociative symptoms. Because of these variable expressions of clinical distress following exposure to catastrophic or aversive events, the aforementioned disorders are grouped under a separate category: *trauma- and stressor-related disorders.* Furthermore, it is not uncommon for the clinical picture to include some combination of the above symptoms (with or without anxiety- or fear-based symptoms). Such a heterogeneous picture has long been recognized in adjustment disorders, as well. Social neglect—that is, the absence of adequate caregiving during childhood—is a diagnostic requirement of both reactive attachment disorder and disinhibited social engagement disorder. Although the two disorders share a common etiology,

the former is expressed as an internalizing disorder with depressive symptoms and withdrawn behavior, while the latter is marked by disinhibition and externalizing behavior. Finally, it has long been recognized that whereas grief, despair, and general dysphoria can be a part of the normal grieving process after the death of a loved one, the expression of such emotions is sometimes abnormally excessive in duration and/or intensity. The diagnosis of prolonged grief disorder has been introduced in this chapter to meet this clinical concern.

Reactive Attachment Disorder

Diagnostic Criteria F94.1

A. A consistent pattern of inhibited, emotionally withdrawn behavior toward adult caregivers, manifested by both of the following:
   1. The child rarely or minimally seeks comfort when distressed.
   2. The child rarely or minimally responds to comfort when distressed.

B. A persistent social and emotional disturbance characterized by at least two of the following:
   1. Minimal social and emotional responsiveness to others.
   2. Limited positive affect.

   3. Episodes of unexplained irritability, sadness, or fearfulness that are evident even during nonthreatening interactions with adult caregivers.

C. The child has experienced a pattern of extremes of insufficient care as evidenced by at least one of the following:

1. Social neglect or deprivation in the form of persistent lack of having basic emotional needs for comfort, stimulation, and affection met by caregiving adults.
2. Repeated changes of primary caregivers that limit opportunities to form stable attachments (e.g., frequent changes in foster care).
3. Rearing in unusual settings that severely limit opportunities to form selective attachments (e.g., institutions with high child-to-caregiver ratios).

D. The care in Criterion C is presumed to be responsible for the disturbed behavior in Criterion A (e.g., the disturbances in Criterion A began following the lack of adequate care in Criterion C).

E. The criteria are not met for autism spectrum disorder.

F. The disturbance is evident before age 5 years.

G. The child has a developmental age of at least 9 months.

Specify if:

Persistent: The disorder has been present for more than 12 months.

Specify current severity:

Reactive attachment disorder is specified as **severe** when a child exhibits all symptoms of the disorder, with each symptom manifesting at relatively high levels.

Diagnostic Features

Reactive attachment disorder is characterized by a pattern of markedly disturbed and developmentally inappropriate attachment behaviors, in which a child rarely or minimally turns preferentially to an attachment figure for comfort, support, protection, and nurturance. The essential feature is absent or grossly underdeveloped attachment between the child and putative caregiving adults. Children with reactive attachment disorder are believed to have the capacity to form selective attachments. However,

because of limited opportunities during early development, they fail to show the behavioral manifestations of selective attachments. That is, when distressed, they show no consistent effort to obtain comfort, support, nurturance, or protection from caregivers. Furthermore, when distressed, children with this disorder do not respond more than minimally to comforting efforts of caregivers. Thus, the disorder is associated with the absence of expected comfort seeking and response to comforting behaviors. As such, children with reactive attachment disorder show diminished or absent expression of positive emotions during routine interactions with caregivers. In addition, their emotion regulation capacity is compromised, and they display episodes of negative emotions of fear, sadness, or irritability that are not readily explained. A diagnosis of reactive attachment disorder should not be made in children who are developmentally unable to form selective attachments. For this reason, the child must have a developmental age of at least 9 months. Diagnostic assessment is enhanced by multiple sources of input, supporting that the symptoms are apparent across contexts.

Associated Features

Because of the shared etiological association with social neglect, reactive attachment disorder often co-occurs with developmental delays, especially in delays in cognition and language. Other associated features include stereotypies and other signs of severe neglect (e.g., malnutrition or signs of poor care).

Prevalence

The prevalence of reactive attachment disorder is unknown, but the disorder is seen relatively rarely in clinical settings. The disorder has been found in young children exposed to severe neglect before being placed in foster care or raised in institutions. The disorder is uncommon, usually occurring in less than 10% of neglected children, even in cases of severe neglect.

Development and Course

Conditions of social neglect are often present in the first months of life in children diagnosed with reactive attachment disorder, even before the disorder is diagnosed. The clinical features of the disorder manifest in a similar fashion between the ages of 9 months and 5 years. That is, signs of absent-to-minimal attachment behaviors and associated emotionally aberrant behaviors are evident in children throughout this age range, although differing cognitive and motor abilities may affect how these behaviors are expressed. Remediation and symptomatic recovery may occur through normative caregiving environments; however, in the absence of enhanced caregiving, the signs of the disorder may persist, at least for several years. Persistent signs of reactive attachment disorder in early adolescence may be associated with problems in social functioning. Less is known about the clinical presentation of reactive attachment disorder in older children, and the diagnosis should be made with caution in children older than 5 years.

Risk and Prognostic Factors

Environmental. Serious social neglect is a diagnostic requirement for reactive attachment disorder and is also the only known risk factor for the disorder. However, the majority of severely neglected children do not develop the disorder. Prognosis for children with the disorder appears to depend on the quality of the caregiving environment following serious neglect.

Culture-Related Diagnostic Issues

There is limited information on reactive attachment behavior in young children from diverse cultural backgrounds around the world. Cultural expectations of attachment behaviors and caregiving practices may influence development of and concern about these patterns of behaviors and their presentations in different settings. Caution should be exercised in making the diagnosis of reactive attachment disorder in cultural contexts in which attachment has not been studied. Symptoms of reactive attachment disorder may be more common in situations where attachment figures have experienced extensive trauma, such as war-zone settings; attachment styles may also vary among migrant and refugee children during the resettlement

period. Variations in nurturing care practices may influence risk of reactive attachment disorder.

Functional Consequences of Reactive Attachment Disorder

Reactive attachment disorder significantly impairs young children's abilities to relate interpersonally to adults or peers and is associated with functional impairment across many domains of early childhood.

Differential Diagnosis

Autism spectrum disorder. Aberrant social behaviors manifest in young children with reactive attachment disorder, but they also are key features of autism spectrum disorder. Specifically, young children with either condition can manifest dampened expression of positive emotions, cognitive and language delays, and impairments in social reciprocity. As a result, reactive attachment disorder must be differentiated from autism spectrum disorder. These

two disorders can be distinguished based on differential histories of neglect and on the presence of restricted interests or ritualized behaviors, specific deficit in social communication, and selective attachment behaviors. Children with reactive attachment disorder have experienced a history of severe social neglect, although it is not always possible to obtain detailed histories about the precise nature of their experiences, especially in initial evaluations. Children with autism spectrum disorder will only rarely have a history of social neglect. The restricted interests and repetitive behaviors characteristic of autism spectrum disorder are not a feature of reactive attachment disorder. These clinical features manifest as excessive adherence to rituals and routines; restricted, fixated interests; and unusual sensory reactions. However, it is important to note that children with either condition can exhibit stereotypic behaviors such as rocking or flapping. Children with either disorder also may exhibit a range of intellectual functioning, but only children with autism spectrum disorder exhibit

selective impairments in social communicative behaviors, such as intentional communication (i.e., impairment in communication that is deliberate, goal-directed, and aimed at influencing the behavior of the recipient). Children with reactive attachment disorder show social communicative functioning comparable to their overall level of intellectual functioning. Finally, children with autism spectrum disorder regularly show attachment behavior typical for their developmental level. In contrast, children with reactive attachment disorder do so only rarely or inconsistently, if at all. Structured observations can help discriminate between the two disorders.

Intellectual developmental disorder (intellectual disability). Developmental delays often accompany reactive attachment disorder, but they should not be confused with the disorder. Children with intellectual developmental disorder should exhibit social and emotional skills comparable to their cognitive skills and do not demonstrate the profound reduction in positive affect and emotion regulation difficulties evident in children with reactive attachment disorder. In addition, developmentally delayed children who have reached a cognitive age of 7–9 months should demonstrate selective attachments regardless of their chronological age. In contrast, children with reactive attachment disorder show lack of preferred attachment despite having attained a developmental age of at least 9 months.

Depressive disorders. Depression in young children is also associated with reductions in positive affect. There is limited evidence, however, to suggest that children with depressive disorders have impairments in attachment. That is, young children who have been diagnosed with depressive disorders still should seek and respond to comforting efforts by caregivers.

Comorbidity

Conditions associated with neglect, including cognitive delays, language delays, and stereotypies, often co-occur with reactive attachment disorder. Medical conditions, such as severe malnutrition, may accompany signs of the disorder. Internalizing symptoms also may co-occur with reactive attachment disorder. A relationship between reactive attachment disorder and externalizing behavior problems or attention-deficit/hyperactivity disorder (ADHD) has been suggested but not clearly established.

Disinhibited Social Engagement Disorder

Diagnostic Criteria F94.2

A. A pattern of behavior in which a child actively approaches and interacts with unfamiliar adults and exhibits at least two of the following:
   1. Reduced or absent reticence in approaching and interacting with unfamiliar adults.
   2. Overly familiar verbal or physical behavior (that is not consistent with culturally sanctioned and with age-appropriate social boundaries).

   3. Diminished or absent checking back with adult caregiver after venturing away, even in unfamiliar settings.
   4. Willingness to go off with an unfamiliar adult with minimal or no hesitation.

B. The behaviors in Criterion A are not limited to impulsivity (as in attention-deficit/hyperactivity disorder) but include socially disinhibited behavior.

C. The child has experienced a pattern of extremes of insufficient care as evidenced by at least one of the following:
   1. Social neglect or deprivation in the form of persistent lack of having basic emotional needs for comfort, stimulation, and affection met by caregiving adults.
   2. Repeated changes of primary caregivers that limit opportunities to form stable attachments (e.g., frequent changes in foster care).
   3. Rearing in unusual settings that severely limit opportunities to form selective attachments (e.g.,

institutions with high child-to-caregiver ratios).

D. The care in Criterion C is presumed to be responsible for the disturbed behavior in Criterion A (e.g., the disturbances in Criterion A began following the pathogenic care in Criterion C).

E. The child has a developmental age of at least 9 months.

Specify if:

Persistent: The disorder has been present for more than 12 months.

Specify current severity:

Disinhibited social engagement disorder is specified as **severe** when the child exhibits all symptoms of the disorder, with each symptom manifesting at relatively high levels.

Diagnostic Features

The essential feature of disinhibited social engagement disorder is a pattern of behavior that involves culturally inappropriate, overly familiar behavior with relative strangers (Criterion A). This overly familiar behavior violates the social boundaries of the culture. A diagnosis of disinhibited social engagement disorder should not be made before children are developmentally able to form selective attachments. For this reason, the child must have a developmental age of at least 9 months.

Associated Features

Because of the shared etiological association with social neglect, disinhibited social engagement disorder may co-occur with developmental delays, especially cognitive and language delays, stereotypies, and other signs of severe neglect, such as malnutrition or poor care. However, signs of the disorder often persist even after these other signs of neglect are no longer present. Therefore, it is not uncommon for children with the disorder to present with no current signs of neglect. Moreover, the condition can present in children who show no signs of disordered attachment. Thus, disinhibited social engagement disorder may be seen in children with a

history of neglect who lack attachments or whose attachments to their caregivers range from disturbed to secure.

Prevalence

The prevalence of disinhibited social engagement disorder is unknown. Nevertheless, the disorder appears to be rare, occurring in a minority of children, even those who have experienced severe early deprivation. In low-income community populations in the United Kingdom, the prevalence is up to 2%.

Development and Course

Conditions of social neglect are often present in the first months of life in children diagnosed with disinhibited social engagement disorder, even before the disorder is diagnosed. As noted by research among children with a history of institutional care, if neglect occurs early and signs of the disorder appear, clinical features of the disorder are moderately stable over time, particularly if conditions of neglect persist.

Signs of disinhibited social engagement disorder have been described from the second year of life through adolescence among children raised in institutional settings, and even into young adulthood. There are some differences in manifestations of the disorder from early childhood to older ages. At the youngest ages, across many cultures, children typically show reticence when interacting with strangers, which is nonpathological, even if they are raised in institutions and foster care. Young children with the disorder, however, fail to show reticence to approach and are found to engage with, and even accompany, unfamiliar adults without hesitation, as shown by research among children with a history of institutionalized care. In preschool children raised in institutional settings in the United Kingdom or the United States, verbal and social intrusiveness appeared most prominent, often accompanied by attention-seeking behavior; preschool children raised in institutional settings across several countries have displayed a pattern of engaging in physical contact with strangers. Verbal and physical overfamiliarity continued through middle childhood,

sometimes accompanied by inauthentic expressions of emotion. In adolescence, indiscriminate behavior may extend to peers. Relative to healthy adolescents, adolescents with the disorder have more “superficial” peer relationships and more peer conflicts. Adult manifestations of the disorder appear to be similar but may include excessive self-disclosure and reduced stranger awareness.

Risk and Prognostic Factors

Temperamental. There is some evidence from research with international adoptees in the United States that both blunted reward sensitivity and decreased inhibitory control are associated with indiscriminate social behavior.

Environmental. Serious social neglect is a diagnostic requirement for disinhibited social engagement disorder. The rationale for this requirement includes research finding a strong association between neglect and features of the disorder. Other factors also have been implicated, such as multiple placement disruptions, borderline personality disorder in the mother, and aberrant caregiving behaviors and low quality of care. All of these contribute to the insufficient care criterion. Still, the majority of severely neglected children do not develop the disorder. The disorder has not been identified in children who experience social neglect only after age 2 years. Prognosis is only modestly associated with quality of the caregiving environment following serious neglect. In many cases, the disorder persists, even in children whose caregiving environment becomes markedly improved.

Genetic and physiological. Various neurobiological factors have been associated with symptoms of the disorder, but findings concerning the nature of such factors and their specific tie to the disorder remain preliminary.

Course modifiers. Caregiving quality seems to moderate the course of disinhibited social engagement disorder, at least in young children. Nevertheless, even after placement in normative caregiving environments, some children show persistent signs of the disorder, through adolescence and into adulthood.

Culture-Related Diagnostic Issues

There is limited cross-cultural information on disinhibited social engagement disorder. Cultural expectations of children's social behaviors may affect their level of disinhibition

toward strangers. The absence of reticence that is characteristic of disinhibited social engagement disorder should exceed culturally accepted norms.

Functional Consequences of Disinhibited Social Engagement Disorder

Disinhibited social engagement disorder significantly impairs young children's abilities to relate interpersonally to adults and peers. Both general social functioning and social competence may be impaired, along with increased risk for peer conflicts and victimization.

Differential Diagnosis

Attention-deficit/hyperactivity disorder. Children with disinhibited social engagement disorder can be distinguished from those with ADHD accompanied by social impulsivity, as the former do not show difficulties with attention or hyperactivity.

Comorbidity

Conditions associated with neglect, including cognitive delays, language delays, and stereotypies, may co-occur with disinhibited social engagement disorder. Autism spectrum disorder may also co-occur. In younger children and in middle childhood, disinhibited social engagement disorder often co-occurs with ADHD and externalizing disorders; this co-occurrence has been proposed to relate to common impairments in cognitive inhibitory control.

Posttraumatic Stress Disorder

Diagnostic Criteria F43.10

Posttraumatic Stress Disorder in Individuals Older Than 6 Years

Note: The following criteria apply to adults, adolescents, and children older than 6 years. For children 6 years and younger, see corresponding criteria below.

A. Exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:

1. Directly experiencing the traumatic event(s).
2. Witnessing, in person, the event(s) as it occurred to others.
3. Learning that the traumatic event(s) occurred to a close family member or close friend. In cases of actual or threatened death of a family member or friend, the event(s) must have been violent or accidental.
4. Experiencing repeated or extreme exposure to aversive details of the traumatic event(s) (e.g., first responders collecting human remains; police officers repeatedly exposed to details of child abuse).

   Note: Criterion A4 does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.

B. Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:

1. Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s).

   Note: In children older than 6 years, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.

2. Recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s).

   Note: In children, there may be frightening dreams without recognizable content.

3. Dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.)

   Note: In children, trauma-specific reenactment may occur in play.

4. Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).
5. Marked physiological reactions to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).

C. Persistent avoidance of stimuli associated with the traumatic event(s), beginning after the traumatic event(s) occurred, as evidenced by one or both of the following:

   1. Avoidance of or efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).
   2. Avoidance of or efforts to avoid external reminders (people, places, conversations, activities, objects, situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).

D. Negative alterations in cognitions and mood associated with the traumatic event(s), beginning or worsening after the

traumatic event(s) occurred, as evidenced by two (or more) of the following:

1. Inability to remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs).
2. Persistent and exaggerated negative beliefs or expectations about oneself, others, or the world (e.g., “I am bad,” “No one can be trusted,” “The world is completely dangerous,” “My whole nervous system is permanently ruined”).
3. Persistent, distorted cognitions about the cause or consequences of the traumatic event(s) that lead the individual to blame himself/herself or others.
4. Persistent negative emotional state (e.g., fear, horror, anger, guilt, or shame).
5. Markedly diminished interest or participation in significant activities.
6. Feelings of detachment or estrangement from others.
7. Persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings).

E. Marked alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

1. Irritable behavior and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects.
2. Reckless or self-destructive behavior.
3. Hypervigilance.
4. Exaggerated startle response.
5. Problems with concentration.

6. Sleep disturbance (e.g., difficulty falling or staying asleep or restless sleep).

F. Duration of the disturbance (Criteria B, C, D, and E) is more than 1 month.

G. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

H. The disturbance is not attributable to the physiological effects of a substance (e.g., medication, alcohol) or another medical condition.

Specify whether:

With dissociative symptoms: The individual's symptoms meet the criteria for posttraumatic stress disorder, and in addition, in response to the stressor, the individual experiences persistent or recurrent symptoms of either of the following:

1. **Depersonalization:** Persistent or recurrent experiences of feeling detached from, and as if one were an outside observer of, one's mental processes or body (e.g., feeling as though one were in a dream; feeling a sense of unreality of self or body or of time moving slowly).
2. **Derealization:** Persistent or recurrent experiences of unreality of surroundings (e.g., the world around the individual is experienced as unreal, dreamlike, distant, or distorted).

Note: To use this subtype, the dissociative symptoms must not be attributable to the physiological effects of a substance (e.g., blackouts, behavior during alcohol intoxication) or another medical condition (e.g., complex partial seizures).

Specify if:

With delayed expression: If the full diagnostic criteria are not met until at least 6 months after the event (although the onset and expression of some symptoms may be immediate).

Posttraumatic Stress Disorder in Children 6 Years and Younger

A. In children 6 years and younger, exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:

   1. Directly experiencing the traumatic event(s).
   2. Witnessing, in person, the event(s) as it occurred to others, especially primary caregivers.
   3. Learning that the traumatic event(s) occurred to a parent or caregiving figure.

B. Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:

   1. Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s).

      Note: Spontaneous and intrusive memories may not necessarily appear distressing and may be expressed as play reenactment.
   2. Recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s).

      Note: It may not be possible to ascertain that the frightening content is related to the traumatic event.
   3. Dissociative reactions (e.g., flashbacks) in which the child feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.) Such trauma-specific reenactment may occur in play.

4. Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).
5. Marked physiological reactions to reminders of the traumatic event(s).

C. One (or more) of the following symptoms, representing either persistent avoidance of stimuli associated with the traumatic event(s) or negative alterations in cognitions and

mood associated with the traumatic event(s), must be present, beginning after the event(s) or worsening after the event(s):

Persistent Avoidance of Stimuli

1. Avoidance of or efforts to avoid activities, places, or physical reminders that arouse recollections of the traumatic event(s).
2. Avoidance of or efforts to avoid people, conversations, or interpersonal situations that arouse recollections of the traumatic event(s).

Negative Alterations in Cognitions

3. Substantially increased frequency of negative emotional states (e.g., fear, guilt, sadness, shame, confusion).
4. Markedly diminished interest or participation in significant activities, including constriction of play.
5. Socially withdrawn behavior.
6. Persistent reduction in expression of positive emotions.

D. Alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

1. Irritable behavior and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects (including extreme temper tantrums).
2. Hypervigilance.
3. Exaggerated startle response.
4. Problems with concentration.
5. Sleep disturbance (e.g., difficulty falling or staying asleep or restless sleep).

E. The duration of the disturbance is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in relationships with parents, siblings, peers, or other caregivers or with school behavior.

G. The disturbance is not attributable to the physiological effects of a substance (e.g., medication or alcohol) or another medical condition.

Specify whether:

With dissociative symptoms: The individual's symptoms meet the criteria for posttraumatic stress disorder, and the individual experiences persistent or recurrent symptoms of either of the following:

1. **Depersonalization:** Persistent or recurrent experiences of feeling detached from, and as if one were an outside observer of, one's mental processes or body (e.g., feeling as though one were in a dream; feeling a sense of unreality of self or body or of time moving slowly).
2. **Derealization:** Persistent or recurrent experiences of unreality of surroundings (e.g., the world around the individual is experienced as unreal, dreamlike, distant, or distorted).

Note: To use this subtype, the dissociative symptoms must not be attributable to the physiological effects of a substance

(e.g., blackouts) or another medical condition (e.g., complex partial seizures).

Specify if:

With delayed expression: If the full diagnostic criteria are not met until at least 6 months after the event (although the onset and expression of some symptoms may be immediate).

Diagnostic Features

The essential feature of posttraumatic stress disorder (PTSD) is the development of characteristic symptoms following exposure to one or more traumatic events. The clinical presentation of PTSD varies. In some individuals, fear-based reexperiencing, emotional, and behavioral symptoms may predominate. In others, anhedonic or dysphoric mood states and negative cognitions may be most prominent. In some other individuals, arousal and reactive-externalizing symptoms are prominent, while in yet others, dissociative symptoms predominate. Finally, some individuals exhibit combinations of these symptom patterns.

The following discussion of specific criteria for PTSD refers to specific criteria for adults; criteria for children 6 years or younger may differ in criterion numbering given differences in applicable criteria for this age group.

The traumatic events in Criterion A all involve actual or threatened death, serious injury, or sexual violence in some way but differ in how the individual is exposed to them, which can be through directly experiencing the traumatic event (Criterion A1), witnessing in person the event as it occurred to others (Criterion A2), learning that the event occurred to a family member or a close friend (Criterion A3), or indirect exposure in the course of occupational duties, through being exposed to grotesque details of an event (Criterion A4). The disorder may be especially severe or long-lasting when the stressor is interpersonal and intentional (e.g., torture, sexual violence).

The directly experienced traumatic events in Criterion A include, but are not limited to, exposure to war as a combatant or civilian, actual or threatened physical assault in which the threat is perceived as imminent and realistic (e.g., physical attack, robbery, mugging, childhood physical abuse), being kidnapped, being taken hostage, terrorist attack, torture, incarceration as a prisoner of war, natural or human-made disasters, and severe motor vehicle accidents.

Sexual trauma includes, but is not limited to, actual or threatened sexual violence or coercion (e.g., forced sexual penetration; alcohol/drug-facilitated nonconsensual sexual penetration; other unwanted sexual contact; and other unwanted sexual experiences not involving contact, such as being forced to watch pornography, exposure to the display of genitals by an exhibitionist, or being the victim of unwanted photography or videotaping of a sexual nature or the unwanted dissemination of these photographs or videos).

Being bullied may qualify as a Criterion A1 experience when there is a credible threat of serious harm or sexual violence. For children, sexually violent events may include developmentally inappropriate sexual experiences without physical violence or injury.

A life-threatening illness or debilitating medical condition is not necessarily considered a traumatic event. Qualifying events of this type include life-threatening medical emergencies (e.g., an acute myocardial infarction, anaphylactic shock) or a particular event in treatment that evokes catastrophic feelings of terror, pain, helplessness, or imminent death (e.g., waking during surgery, debridement of severe burn wounds, emergency cardioversion).

Witnessed events (Criterion A2) include, but are not limited to, observing threatened or serious injury, unnatural death, physical or sexual abuse of another person due to violent assault, domestic violence, accident, war, or disaster. For example, this would include parents witnessing their child in an acute life-endangering incident (e.g., a diving accident) or a medical catastrophe during the course of their child’s illness or ongoing treatment (e.g., a life-threatening hemorrhage).

Indirect exposure through learning about an event (Criterion A3) is limited to events affecting close relatives or friends that were violent or accidental (i.e., death from natural causes does not qualify). Such events

include murder, violent personal assault, combat, terrorist attack, sexual violence, suicide, and serious accident or injury.

The indirect exposure of professionals to the grotesque effects of war, rape, genocide, or abusive violence inflicted on others occurring in the context of their work duties can also result in PTSD and thus is considered to be a qualifying trauma (Criterion A4). Examples

include first responders exposed to serious injury or death and military personnel collecting human remains. Indirect exposure can also occur through photos, videos, verbal accounts, or written accounts (e.g., police officers reviewing crime reports or conducting interviews with crime victims, drone operators, members of the news media covering traumatic events, and psychotherapists exposed to details of their patients' traumatic experiences).

Exposure to multiple traumatic events is common and can take many forms. Some individuals experience different types of traumatic events at different times (e.g., sexual violence during childhood and natural disaster as adults). Others experience the same type of traumatic event at different times or in a series committed by the same person/people over an extended period (e.g., child sexual or physical assault; physical or sexual assault by an intimate partner). Others may experience numerous traumatic events that are the same or different during an extended hazardous period such as deployment or living in a conflict zone. When one is assessing the PTSD criteria in individuals who have experienced multiple traumatic events across their lives, it may be useful to determine if there is a specific, discrete example that the individual considers to be the worst given that the symptomatic expressions of PTSD Criterion B and Criterion C specifically refer to the traumatic event (e.g., recurrent, involuntary, and intrusive distressing recollections of the traumatic event). However, if it is difficult for the individual to identify a worst example, it is appropriate to consider the entire exposure as meeting Criterion A. In addition, some discrete events may incorporate several traumatic event types (e.g., an individual involved in a mass casualty incident sustains a major injury, witnesses

someone else being injured, and then learns that a family member was killed in the incident).

The traumatic event can be reexperienced in various ways. Commonly, the individual has recurrent, involuntary, and intrusive recollections of the event (Criterion B1). Intrusive recollections in PTSD are distinguished from depressive rumination in that they apply only to involuntary and intrusive distressing memories. The emphasis is on recurrent memories of the event that usually include intrusive, vivid, sensory, and emotional components that are distressing and not merely ruminative. A common reexperiencing symptom is distressing dreams that replay the event itself or that are representative or thematically related to the major threats involved in the traumatic event (Criterion B2). The individual may experience dissociative states that typically last a few seconds and rarely are of a longer duration, during which components of the event are relived and the individual behaves as if the event were occurring at that moment (Criterion B3). Such events occur on a continuum, ranging from brief visual or other sensory intrusions about part of the traumatic event without loss of reality orientation to a partial loss of awareness of present surroundings to a complete loss of awareness. These episodes, often referred to as "flashbacks," are typically brief but can be associated with prolonged distress and heightened arousal. For young children, reenactment of events related to trauma may be expressed behaviorally in play or in dissociative states. Intense psychological distress (Criterion B4) or physiological reactivity (Criterion B5) often occurs when the individual is exposed to triggering events or somatic reactions that resemble or symbolize an aspect of the traumatic event (e.g., windy days after a hurricane, seeing someone who resembles one's perpetrator). The triggering cue could also be a physical sensation (e.g., dizziness for survivors of head trauma, rapid heartbeat for a previously traumatized child), particularly for individuals with highly somatic presentations.

Stimuli associated with the trauma are persistently avoided. The individual commonly makes deliberate efforts to avoid thoughts, memories, or feelings (e.g., by utilizing distraction or suppression techniques, including substance use, to avoid internal reminders) (Criterion C1), and to avoid activities, conversations, objects, situations, or people who arouse recollections of it (Criterion C2).

Negative alterations in cognitions or mood associated with the traumatic event begin or worsen after exposure to the event. These negative alterations can take various forms, including an inability to remember key and emotionally painful aspects of the traumatic

event. Such memory loss is typically attributable to dissociative amnesia and is not attributable to head injury or impaired encoding of the memory due to drug or alcohol use (Criterion D1). Individuals with PTSD often report that the traumatic event has irrevocably altered their lives and their view of the world. This is characterized by persistent and exaggerated negative beliefs and expectations regarding important aspects of life applied to themselves, others, the world, or the future (Criterion D2) (e.g., “Bad things will always happen to me”; “The world is dangerous, and I can never be adequately protected”; “I can’t trust anyone ever again”; “My life is permanently ruined”; “I have lost any chance for future happiness”; “My life will be cut short”). Individuals with PTSD may have persistent erroneous cognitions about the causes of the traumatic event that lead them to blame themselves or others (e.g., “It’s all my fault that my uncle abused me”) (Criterion D3). A persistent negative mood state (e.g., fear, dysphoria, horror, anger, guilt, shame) either began or worsened after exposure to the event (Criterion D4). The individual may experience markedly diminished interest or participation in previously enjoyed activities (Criterion D5), may feel detached or estranged from other people (Criterion D6), or may experience a persistent inability to feel positive emotions (especially happiness, joy, satisfaction, or emotions associated with intimacy, tenderness, and sexuality) (Criterion D7).

Negative alterations in arousal and reactivity also begin or get worse after exposure to the event. Individuals with PTSD may exhibit irritable or angry behavior and may engage in aggressive verbal or physical behavior with little or no provocation (e.g., yelling at people, getting into fights, destroying objects) (Criterion E1). They may also engage voluntarily in reckless or self-destructive behavior that is dangerous, that shows a disregard for the physical safety of themselves or others, and that could directly result in serious physical harm or death (Criterion E2). Examples

include, but are not limited to, dangerous driving (e.g., drunk driving, driving at dangerously high speeds), excessive alcohol or drug use, having risky sex (e.g., unprotected sex with a partner whose HIV status is unknown, high number of sexual partners), or self-directed violence including suicidal behaviors. Criterion E2 does not include circumstances in which individuals must engage in dangerous situations as a part of their job (e.g., armed forces members in combat situations or first responders in emergency situations) and take reasonable safety precautions to reduce their risk or when individuals engage in behaviors that may be unwise, unhealthy, or financially harmful but pose no direct risk of immediate serious physical harm or death (e.g., pathological gambling, poor financial decisions, binge eating, unhealthy lifestyles). PTSD is often characterized by a heightened vigilance for potential threats, including those that are related to the traumatic experience (e.g., following a motor vehicle accident, being especially sensitive to the threat potentially caused by cars or trucks) and those not related to the traumatic event (e.g., being fearful of suffering a heart attack) (Criterion E3). Individuals with PTSD may be very reactive to unexpected stimuli, displaying a heightened startle response, or jumpiness, to loud noises or unexpected movements (e.g., jumping markedly in response to a telephone ringing) (Criterion E4). Startle responses are involuntary and reflexive (automatic, instantaneous), and stimuli that evoke exaggerated startle responses (Criterion E4) need not be related at all to the traumatic event. Startle responses are distinguished from the cued physiological arousal responses in Criterion B5, for which there needs to be at least some level of conscious appraisal that the stimulus producing physiological responses is related to the trauma. Concentration difficulties, including difficulty remembering daily events (e.g., forgetting one’s telephone number) or attending to focused tasks (e.g., following a conversation for a sustained period of time), are commonly reported (Criterion E5). Problems with sleep onset and maintenance are common and may be associated with nightmares and safety concerns or with generalized elevated arousal that interferes with adequate sleep (Criterion E6).

The diagnosis of PTSD requires that the duration of the symptoms in Criteria B, C, D, and E be more than 1 month (Criterion F). For a current diagnosis of PTSD, Criteria B, C, D, and E must all be met for more than 1 month, for at least the past month. For a lifetime

diagnosis of PTSD, there must be a period of time lasting more than 1 month during which Criteria B, C, D, and E have all been met for the same 1-month period of time.

A significant subgroup of individuals with PTSD experience persistent dissociative symptoms of either depersonalization (detachment from their bodies) or derealization (detachment from the world around them). This can be indicated by using the “with dissociative symptoms” specifier.

Associated Features

Developmental regression, such as loss of language in young children, may occur. Auditory pseudo-hallucinations, such as having the sensory experience of hearing one’s thoughts spoken in one or more different voices, as well as paranoid ideation, can be present. Following prolonged, repeated, and severe traumatic events (e.g., childhood abuse, torture), the individual may additionally experience difficulties in regulating emotions or maintaining stable interpersonal relationships, or dissociative symptoms. When the traumatic event involves the violent death of someone with whom the individual had a close relationship, symptoms of both prolonged grief disorder and PTSD may be present.

Prevalence

The national lifetime prevalence estimate for PTSD using DSM-IV criteria is 6.8% for U.S. adults. Lifetime prevalence for U.S. adolescents using DSM-IV criteria has ranged from 5.0% to 8.1% and a past 6-month prevalence of 4.9% for adolescents. While definitive, comprehensive population-based data using DSM-5 are not available, findings are beginning to emerge. In two U.S. national epidemiological studies, lifetime DSM-5 PTSD prevalence estimates ranged from 6.1% to 8.3%, and the national 12-month DSM-5 prevalence estimate was 4.7% in both studies. National lifetime DSM-IV PTSD estimates from World Mental Health Surveys in 24 countries varied substantially among countries, income country groups, and WHO regions but was 3.9% overall. In conflict-affected populations worldwide, the point prevalence of PTSD with

functional impairment is 11% after adjustment for age differences across studies.

Rates of PTSD are higher among veterans and others whose vocation increases the risk of traumatic exposure (e.g., police, firefighters, emergency medical personnel). Highest rates (ranging from one-third to more than one-half of those exposed) are found among survivors of rape, military combat and captivity, and ethnically or politically motivated internment and genocide. The prevalence of PTSD may vary across development; children and adolescents, including preschool children, generally have displayed lower prevalence following exposure to serious traumatic events; however, this may be because previous criteria were insufficiently developmentally informed. Racial differences, based on DSM-IV data, show higher rates of PTSD among U.S. Latinx, African Americans, and American Indians compared with Whites. Potential reasons for these prevalence variations include differences in predisposing or enabling factors, such as exposure to past adversity and racism and discrimination, and in availability or quality of treatment, social support, socioeconomic status, and other social resources that facilitate recovery and are confounded with ethnic and racialized background.

Development and Course

PTSD can occur at any age, beginning after the first year of life. Symptoms usually begin within the first 3 months after the trauma, although there may be a delay of months, or even years, before full criteria for the diagnosis are met. There is abundant evidence for what DSM-IV called “delayed onset” but is now called “delayed expression,” with the recognition that some symptoms typically appear immediately and that the delay is in meeting full criteria.

Frequently, an individual’s reaction to a trauma initially meets criteria for acute stress disorder in the immediate aftermath of the trauma. The symptoms of PTSD and the

relative predominance of different symptoms may vary over time. Duration of the symptoms also varies, with complete recovery within 3 months occurring in approximately one-half of adults, while some individuals remain symptomatic for longer than 12 months and sometimes for more than 50 years. Symptom recurrence and intensification may occur in response to reminders of the original trauma, ongoing life stressors, or newly experienced traumatic events.

The clinical expression of reexperiencing can vary across development. Developmental variations in clinical expression inform the use of different criteria in children 6 years and younger and in individuals who are older. Young children may report new onset of frightening dreams without content specific to the traumatic event. Children age 6 and younger may develop PTSD as a result of severe emotional abuse (e.g., threat of abandonment), which can be perceived as life-threatening. During treatment for life-threatening illness (e.g., cancer, solid organ transplantation), the experience of young children of the severity and intensity of the treatment may contribute to risk of developing posttraumatic stress symptoms; the self-appraisal of threat may also contribute to the risk of developing posttraumatic stress symptoms in adolescents. Before age 6 years, young children are more likely to express reexperiencing symptoms through play that refers directly or symbolically to the trauma (see PTSD criteria for children 6 years and younger). They may not manifest fearful reactions at the time of the exposure or during reexperiencing. Parents may report a wide range of emotional or behavioral changes in young children. Children may focus on imagined interventions in their play or storytelling. In addition to avoidance, children may become preoccupied with reminders. Because of young children's limitations in expressing thoughts or labeling emotions, negative alterations in mood or cognition tend to involve primarily mood changes. Children may experience co-occurring traumas (e.g., physical abuse, witnessing domestic violence) and in chronic circumstances may not be able to identify onset of symptomatology. Avoidant behavior may be associated with restricted play or exploratory behavior in young children; reduced participation in new activities in school-age children; or reluctance to pursue developmental opportunities in adolescents (e.g., dating, driving). Older children and adolescents may judge themselves as cowardly. Adolescents may harbor beliefs of being

changed in ways that make them socially undesirable and estrange them from peers and lose aspirations for the future. Irritable or aggressive behavior in children and adolescents can interfere with peer relationships and school behavior. Reckless behavior may lead to accidental injury to self or others, thrill-seeking, or high-risk behaviors. In older individuals, the disorder is associated with negative health perceptions, primary care utilization, and suicidal thoughts. In addition, declining health, worsening cognitive functioning, and social isolation may exacerbate PTSD symptoms.

Risk and Prognostic Factors

Risk factors for PTSD can operate in many ways, including predisposing individuals to trauma or to extreme emotional responses when exposed to traumatic events. Risk (and protective) factors are generally divided into pretraumatic, peritraumatic, and posttraumatic factors.

Pretraumatic Factors

Temperamental. High-risk factors include childhood emotional problems by age 6 years (e.g., externalizing or anxiety problems) and prior mental disorders (e.g., panic disorder, depressive disorder, PTSD, or obsessive-compulsive disorder [OCD]). Individual differences in premorbid personality may influence the trajectory of response to trauma and treatment outcomes. Personality traits associated with negative emotional responses such as depressed mood and anxiousness represent risk factors for the development of PTSD. Such traits might be captured in measures of negative affectivity (neuroticism) on

standardized personality scales. Premorbid trait impulsivity tends to be associated with externalizing manifestations of PTSD and comorbidities of the externalizing spectrum, including substance use disorder or aggressive behavior.

Environmental. As documented among U.S. civilians and veterans, these risk factors include lower socioeconomic status; lower education; exposure

to prior trauma (especially during childhood); childhood adversity (e.g., economic deprivation, family dysfunction, parental separation or death); lower intelligence; ethnic discrimination and racism; and a family psychiatric history. Social support prior to event exposure is protective.

Genetic and physiological. The risk of developing PTSD following traumatic exposure has been demonstrated to be modestly heritable in twin studies and molecular studies. Genome-wide association data from a large multiethnic cohort support the heritability of PTSD and demonstrate three robust genome-wide significant loci that vary by geographic ancestry. Susceptibility to PTSD may also be influenced by epigenetic factors. Genome-wide association data from U.S. veterans identify eight significant regions in Americans of European descent associated with intrusive reexperiencing symptoms of PTSD; data from the United Kingdom also support these associations.

Peritraumatic Factors

Environmental. These include severity (dose) of the trauma, perceived life threat, personal injury, interpersonal violence (particularly trauma perpetrated by a caregiver or involving a witnessed threat to a caregiver in children), and, for military personnel, being a perpetrator, witnessing atrocities, or killing the enemy. Finally, dissociation, fear, panic, and other peritraumatic responses that occur during the trauma and persist afterward are risk factors.

Posttraumatic Factors

Temperamental. These include negative appraisals, inappropriate coping strategies, and development of acute stress disorder.

Environmental. These include subsequent exposure to repeated upsetting reminders, subsequent adverse life events, and financial or other trauma-related losses. Posttraumatic experiences such as forced migration and high levels of daily stressors may contribute to different conditional risks of PTSD across cultural contexts. Exposure to racial and ethnic discrimination has been associated with a more chronic course among African American and Latinx adults. Social support (including family stability, for children) is a protective factor that moderates outcome after trauma.

Culture-Related Diagnostic Issues

Different demographic, cultural, and occupational groups have different levels of exposure to traumatic events, and the relative risk of developing PTSD following a similar level of exposure (e.g., religious persecution) may also vary across cultural, ethnic, and racialized groups. Variation in the type of traumatic exposure (e.g., genocide), the impact on disorder severity of the meaning attributed to the traumatic event (e.g., inability to perform funerary rites after a mass killing), the ongoing sociocultural context (e.g., residing among unpunished perpetrators in postconflict settings), exposure to racial and ethnic discrimination, and other cultural factors (e.g., acculturative stress in migrants) may influence the risk of onset and severity of PTSD across cultural groups. Some communities are exposed to pervasive and ongoing traumatic environments, rather than isolated Criterion A events; in these communities, the predictive power of individual traumatic events for the development of PTSD may diminish. In cultures where social image (e.g., maintaining a family's "face") is emphasized, public defamation or shaming may magnify the impact of Criterion A events. Some cultures may attribute PTSD syndromes to negative supernatural experiences.

The clinical expression of the symptoms or symptom clusters of PTSD can vary culturally in both adults and children. In many non-Western groups, avoidance is less commonly observed, whereas somatic symptoms (e.g., dizziness, shortness of breath, heat sensations) are more common; other symptoms that vary cross-culturally are distressing dreams, amnesia not related to head injury, and reckless but nonsuicidal behavior. Negative moods, especially anger, are common cross-culturally in individuals with PTSD, as are distressing dreams and sleep paralysis. Across cultures, somatic symptoms are frequent, occurring in both children and adults, especially after sexual trauma. Symptoms that vary cross-culturally in relation to PTSD among children include intrusive thoughts, diminished participation in activities, inability to experience positive emotions, irritability, aggression, and hypervigilance. Distressing dreams, flashbacks,

psychological distress upon exposure to trauma cues, and efforts to avoid memories and thoughts are common in children with PTSD across cultures.

In certain cultural contexts, it may be normative to respond to traumatic events with negative beliefs about oneself or with spiritual attributions that may appear exaggerated to others. For example, blaming oneself may be consistent with ideas of karma in South and East Asia, destiny or "spoiled medicine law" in West Africa, and cultural differences in locus of control and conceptions of self.

In many populations around the world, there are cultural concepts of distress that resemble PTSD and are characterized by diverse manifestations of psychological distress attributed to frightening or traumatic experiences. Thus, cultural concepts of distress influence the expression of PTSD and the range of its comorbid disorders (see "Culture and Psychiatric Diagnosis" in Section III).

Sex- and Gender-Related Diagnostic Issues

PTSD is more prevalent among women than among men across the life span. Lifetime prevalence of PTSD ranges from 8.0% to 11.0% for women and 4.1% to 5.4% for men based on two large U.S. population-based studies using DSM-5 criteria. Some of the increased risk for PTSD in women appears to be attributable to a greater likelihood of exposure to childhood sexual abuse, sexual assault, and other forms of interpersonal violence, which carry the highest risk for development of PTSD. Women in the general population also experience PTSD for a longer duration than do men. However, other factors likely contributing to the higher prevalence in women include gender differences in the emotional and cognitive processing of trauma, as well as effects of reproductive hormones. When responses of men and women to specific stressors are compared, gender differences in risk for PTSD persist. On the other hand, PTSD symptom profiles and factor structures are similar between men and women.

Association With Suicidal Thoughts or Behavior

Traumatic events such as childhood abuse or sexual trauma increase an individual's suicide risk in both civilians and veterans. PTSD is associated with suicidal thoughts, suicide attempts, and death from suicide. The

presence of PTSD has been associated with an increased likelihood of transitioning from suicidal thoughts to a suicide plan or attempt, and this effect of PTSD occurs independently of the increased risk of mood disorders on the likelihood of suicidal behaviors. Among adolescents there is also a significant relationship between PTSD and suicidal thoughts or behavior even after adjustment for the effects of comorbidity.

Functional Consequences of Posttraumatic Stress Disorder

PTSD is associated with high impairment in social, occupational, and physical functioning; reduced quality of life; and physical health problems. Impaired functioning is exhibited

across social, interpersonal, developmental, educational, physical health, and occupational domains. In community and veteran samples, PTSD is associated with poor social and family relationships, absenteeism from work, lower income, and lower educational and occupational success.

Differential Diagnosis

Adjustment disorders. In adjustment disorders, the stressor can be of any severity or type rather than a stressor involving exposure to actual or threatened death, serious injury, or sexual violence as required by PTSD Criterion A. The diagnosis of an adjustment disorder is used when the response to a stressor that meets PTSD Criterion A does not meet all other PTSD criteria (or criteria for another mental disorder). An adjustment disorder is also diagnosed when the symptom pattern of PTSD occurs in response to a stressor that does not meet PTSD Criterion A (e.g., spouse leaving, being fired).

Other posttraumatic disorders and conditions. Not all psychopathology that occurs in individuals exposed to an extreme stressor should necessarily be attributed to PTSD. The PTSD diagnosis requires that trauma exposure precede the onset or exacerbation of pertinent symptoms. If the symptom response pattern to the extreme stressor meets criteria for another mental

disorder, these diagnoses should be given instead of, or in addition to, PTSD. Other diagnoses and conditions are excluded if they are better explained by PTSD (e.g., symptoms of panic disorder that occur only after exposure to traumatic reminders). If severe, symptom response patterns to the extreme stressor that meet criteria for another mental disorder may warrant a separate diagnosis (e.g., dissociative amnesia) in addition to PTSD.

Acute stress disorder. Acute stress disorder is distinguished from PTSD because the symptom pattern in acute stress disorder is restricted to a duration of 3 days to 1 month following exposure to the traumatic event.

Anxiety disorders and obsessive-compulsive disorder. In OCD, there are recurrent intrusive thoughts, but these meet the definition of an obsession. In addition, the intrusive thoughts are not related to an experienced traumatic event, compulsions are usually present, and other symptoms of PTSD or acute stress disorder are typically absent. Neither the arousal and dissociative symptoms of panic disorder nor the avoidance, irritability, and anxiety of generalized anxiety disorder are associated with a specific traumatic event. The symptoms of separation anxiety disorder are clearly related to separation from home or family, rather than to a traumatic event.

Major depressive disorder. Major depression may or may not be preceded by a traumatic event and should be diagnosed if full criteria have been met. Specifically, major depressive disorder does not include any PTSD Criterion B or C symptoms. Nor does it include a number of symptoms from PTSD Criterion D or E. However, if full criteria for PTSD are also met, both diagnoses may be given.

Attention-deficit/hyperactivity disorder. Both ADHD and PTSD may include problems in attention, concentration, and learning. In contrast to ADHD, where the problems in attention, concentration, and learning must have their onset prior to age 12, in PTSD the symptoms have their onset following exposure to a Criterion A traumatic event. In PTSD, disruptions in the individual’s attention and concentration can be attributable to alertness to danger and exaggerated startle responses to reminders of the trauma.

Personality disorders. Interpersonal difficulties that had their onset, or were greatly exacerbated, after exposure to a traumatic event may be an

indication of PTSD, rather than a personality disorder, in which such difficulties would be expected independently of any traumatic exposure.

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Dissociative disorders. Dissociative amnesia, dissociative identity disorder, and depersonalization-derealization disorder may or may not be preceded by exposure to a traumatic event or may or may not have co-occurring PTSD symptoms. When full PTSD criteria are also met, however, the PTSD "with dissociative symptoms" subtype should be considered.

Functional neurological symptom disorder (conversion disorder). New onset of somatic symptoms within the context of posttraumatic distress might be an indication of PTSD rather than functional neurological symptom disorder.

Psychotic disorders. Flashbacks in PTSD must be distinguished from illusions, hallucinations, and other perceptual disturbances that may occur in schizophrenia, brief psychotic disorder, and other psychotic disorders; depressive and bipolar disorders with psychotic features; delirium; substance/medication-induced disorders; and psychotic disorders due to another medical condition. PTSD flashbacks are distinguished from these other perceptual disturbances by being directly related to the traumatic experience and by occurring in the absence of other psychotic or substance-induced features.

Traumatic brain injury. Some types of traumatic events increase risk of both PTSD and traumatic brain injury (TBI) because they can produce head injuries (e.g., military combat, bomb blasts, child physical abuse, intimate partner violence, violent crime, motor vehicle or other accidents). In such cases, individuals presenting with PTSD may also have TBI, and those presenting with TBI may also have PTSD. Individuals with PTSD who also have TBI may have persistent postconcussive symptoms (e.g., headaches, dizziness, sensitivity to light or sound, irritability, concentration deficits). However, such symptoms may also occur in non-brain-injured populations, including individuals with PTSD. Because symptoms of PTSD and TBI-related neurocognitive symptoms can overlap, a differential diagnosis between PTSD and neurocognitive disorder symptoms attributable to TBI may be possible based on the presence of symptoms that are distinctive to

each presentation. Whereas reexperiencing and avoidance are characteristic of PTSD and not the effects of TBI, persistent disorientation and confusion are more specific to TBI (neurocognitive effects) than to PTSD. TBI-related memory problems concerning the traumatic event are typically attributable to injury-related inability to encode the information, whereas PTSD-related memory problems typically reflect dissociative amnesia. Sleep difficulties are common to both disorders.

Comorbidity

Individuals with PTSD are more likely than those without PTSD to have symptoms that meet diagnostic criteria for at least one other mental disorder, such as depressive, bipolar, anxiety, or substance use disorders. PTSD is also associated with increased risk of major neurocognitive disorder. In a U.S.-based study, women were more likely to develop PTSD following a mild TBI. Although most young children with PTSD also have at least one other diagnosis, the patterns of comorbidity are different than in adults, with oppositional defiant disorder and separation anxiety disorder predominating.

Acute Stress Disorder

Diagnostic Criteria F43.0

A. Exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:
   1. Directly experiencing the traumatic event(s).
   2. Witnessing, in person, the event(s) as it occurred to others.

   3. Learning that the event(s) occurred to a close family member or close friend. **Note:** In cases of actual or

threatened death of a family member or friend, the event(s) must have been violent or accidental.

4. Experiencing repeated or extreme exposure to aversive details of the traumatic event(s) (e.g., first responders collecting human remains, police officers repeatedly exposed to details of child abuse).

   Note: This does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.

B. Presence of nine (or more) of the following symptoms from any of the five categories of intrusion, negative mood, dissociation, avoidance, and arousal, beginning or worsening after the traumatic event(s) occurred:

Intrusion Symptoms

1. Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s). **Note:** In children, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.
2. Recurrent distressing dreams in which the content and/or affect of the dream are related to the event(s). **Note:** In children, there may be frightening dreams without recognizable content.
3. Dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.) **Note:** In children, trauma-specific reenactment may occur in play.
4. Intense or prolonged psychological distress or marked physiological reactions in response to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).

Negative Mood

5. Persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings).

Dissociative Symptoms

6. An altered sense of the reality of one's surroundings or oneself (e.g., seeing oneself from another's perspective, being in a daze, time slowing).
7. Inability to remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs).

Avoidance Symptoms

8. Efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).
9. Efforts to avoid external reminders (people, places, conversations, activities, objects, situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).

Arousal Symptoms

10. Sleep disturbance (e.g., difficulty falling or staying asleep, restless sleep).
11. Irritable behavior and angry outbursts (with little or no provocation), typically expressed as verbal or physical aggression toward people or objects.
12. Hypervigilance.
13. Problems with concentration.
14. Exaggerated startle response.

C. Duration of the disturbance (symptoms in Criterion B) is 3 days to 1 month after trauma exposure.

Note: Symptoms typically begin immediately after the trauma, but persistence for at least 3 days and up to a month is needed to meet disorder criteria.

D. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

E. The disturbance is not attributable to the physiological effects of a substance (e.g., medication or alcohol) or another medical condition (e.g., mild traumatic brain injury) and is not better explained by brief psychotic disorder.

Diagnostic Features

The essential feature of acute stress disorder is the development of characteristic symptoms lasting from 3 days to 1 month following exposure to one or more traumatic events (Criterion A), which are the same type as described in PTSD Criterion A (for more information, see "Diagnostic Features" for PTSD).

The clinical presentation of acute stress disorder may vary by individual but typically involves an anxiety response that includes some form of reexperiencing of or reactivity to the traumatic event. Presentations may include intrusion symptoms, negative mood, dissociative symptoms, avoidance symptoms, and arousal symptoms (Criterion B1–B14). In some individuals, a dissociative or detached presentation can predominate, although these individuals typically will also display strong emotional or physiological reactivity in response to trauma reminders. In other individuals, there can be a strong anger response in which reactivity is characterized by irritable or possibly aggressive responses.

Intrusion symptoms (Criterion B1–B4) are the same as described in PTSD Criterion B1–B5 (for discussion of these symptoms, see "Diagnostic Features" for PTSD; note that acute stress disorder Criterion B4 comprises PTSD Criterion B4 and B5). Individuals with acute stress disorder may have a persistent inability to feel positive emotions (e.g., happiness, joy, satisfaction, or emotions associated with intimacy, tenderness, sexuality) but can experience negative emotions such as fear, sadness, anger, guilt, or

shame (Criterion B5). Alterations in awareness can include *depersonalization,* a detached sense of oneself (e.g., seeing oneself from the other side of the room), or *derealization,* having a distorted view of one's surroundings (e.g., perceiving that things are moving in slow motion, seeing things in a daze, not being aware of events that one would normally encode) (Criterion B6). Some individuals also report an inability to remember an important aspect of the traumatic event that was presumably encoded. This symptom is attributable to dissociative amnesia and is not attributable to head injury, alcohol, or drugs (Criterion B7). Stimuli associated with the trauma are persistently avoided. The individual commonly makes deliberate efforts to avoid thoughts, memories, or feelings (e.g., by using distraction or suppression techniques, including substance use, to avoid internal reminders) (Criterion B8), and to avoid activities, conversations, objects, situations, or people who arouse recollections of it (Criterion B9).

It is very common for individuals with acute stress disorder to experience problems with sleep onset and maintenance, which may be associated with nightmares and safety concerns or with generalized elevated arousal that interferes with adequate sleep (Criterion B10). Individuals with acute stress disorder may exhibit irritable behavior and may even engage in aggressive verbal or physical behavior with little or no provocation (e.g., yelling at people, getting into fights, destroying objects) (Criterion B11). Acute stress disorder is often characterized by a heightened vigilance for potential threats, including those that are related to the traumatic experience (e.g., following a motor vehicle accident, being especially sensitive to the threat potentially caused by cars or trucks) and those not related to the traumatic event (e.g., being fearful of suffering a heart attack) (Criterion B12). Concentration difficulties (Criterion B13) include difficulty remembering familiar facts (e.g.,

forgetting one's telephone number) or daily events (e.g., having recently read part of a book or newspaper) or attending to focused tasks (e.g., following a conversation for a sustained period of time).

Individuals with acute stress disorder may be very reactive to unexpected stimuli, displaying a heightened startle response or jumpiness to

loud noises (e.g., in response to a telephone ringing) or unexpected movements (Criterion B14). Startle responses are involuntary and reflexive (automatic, instantaneous), and stimuli that evoke exaggerated startle responses (Criterion B14) need not be related to the traumatic event.

The full symptom picture must last for at least 3 days after the traumatic event but should not last longer than 1 month (Criterion C). Symptoms that occur immediately after the event but resolve in less than 3 days would not meet criteria for acute stress disorder.

Associated Features

Individuals with acute stress disorder commonly engage in catastrophic or extremely negative thoughts about their role in the traumatic event, their response to the traumatic experience, or the likelihood of future harm. For example, an individual with acute stress disorder may feel excessively guilty about not having prevented the traumatic event or about not adapting to the experience more successfully. Individuals with acute stress disorder may also interpret their symptoms in a catastrophic manner, such that flashback memories or emotional numbing may be interpreted as a sign of diminished mental capacity. It is common for individuals with acute stress disorder to experience panic attacks in the initial month after trauma exposure that may be triggered by trauma reminders or may apparently occur spontaneously. Additionally, individuals with acute stress disorder may display chaotic or impulsive behavior. For example, individuals may drive recklessly, make irrational decisions, or gamble excessively. In children, there may be significant separation anxiety, possibly manifested by excessive needs for attention from caregivers. In the case of bereavement following a death that occurred in traumatic circumstances, the symptoms of acute stress disorder can involve acute grief reactions. In such cases, reexperiencing, dissociative, and arousal symptoms may involve reactions to the loss, such as intrusive memories of the circumstances of the individual’s death, disbelief that the individual has died, and anger about the death. Postconcussive symptoms (e.g., headaches, dizziness, sensitivity to light or sound, irritability, concentration deficits), which occur frequently following mild traumatic brain injury (TBI), are also frequently seen in individuals with acute stress disorder. Postconcussive symptoms are equally

common in brain-injured and non-brain-injured populations, and the frequent occurrence of postconcussive symptoms could be attributable to acute stress disorder symptoms.

Prevalence

The prevalence of acute stress disorder in recently trauma-exposed populations (i.e., within 1 month of trauma exposure) varies according to the nature of the event and the context in which it is assessed. In research conducted in Australia, the United Kingdom, and the United States, acute stress disorder was identified in less than 20% of cases following traumatic events that do not involve interpersonal assault—for example, motor vehicle accidents, mild TBI, severe burns, and industrial accidents. Higher rates (i.e., 19%–50%) were usually found following interpersonal traumatic events (e.g., assault, rape).

Development and Course

By definition, acute stress disorder cannot be diagnosed until 3 days after a traumatic event. Although acute stress disorder may progress to posttraumatic stress disorder (PTSD) after 1 month, it may also be a transient stress response that remits within 1 month of trauma exposure and does not result in PTSD. Approximately half of individuals who eventually develop PTSD initially present with acute stress disorder. Longitudinal analyses indicate

that acute stress symptoms can remit, remain constant, or worsen over time, largely as a result of ongoing life stressors or further traumatic events.

The forms of reexperiencing can vary across development. Unlike adults or adolescents, young children may report frightening dreams without content that clearly reflects aspects of the trauma (e.g., waking in fright in the aftermath of the trauma but being unable to relate the content of the dream to the traumatic event). Children age 6 years and younger are more likely than older children to express reexperiencing symptoms through play that refers directly or symbolically to the trauma. For example,

a very young child who survived a fire may draw pictures of flames. Young children also do not necessarily manifest fearful reactions at the time of the exposure or even during reexperiencing. Parents typically report a range of emotional expressions, such as anger, shame, or withdrawal, and even excessively bright positive affect, in young children who are traumatized. Although children may avoid reminders of the trauma, they sometimes become preoccupied with reminders (e.g., a young child bitten by a dog may talk about dogs constantly yet avoid going outside because of fear of coming into contact with a dog).

Risk and Prognostic Factors

Temperamental. Risk factors include prior mental disorder, high levels of negative emotional responses such as depressed mood and anxiousness (also termed *negative affectivity* or *neuroticism*), greater perceived severity of the traumatic event, and an avoidant coping style. A tendency to make catastrophic appraisals of the traumatic experience, often characterized by exaggerated appraisals of future harm, guilt, or hopelessness, is strongly predictive of acute stress disorder.

Environmental. First and foremost, an individual must be exposed to a traumatic event to be at risk for acute stress disorder. Risk factors for the disorder include a history of prior trauma.

Genetic and physiological. Elevated reactivity, as reflected by acoustic startle response, prior to trauma exposure increases the risk for developing acute stress disorder.

Culture-Related Diagnostic Issues

The profile of symptoms of acute stress disorder may vary cross-culturally, particularly with respect to dissociative symptoms, nightmares, avoidance, and somatic symptoms (e.g., dizziness, shortness of breath, heat sensations, pain complaints). Acute stress reactions may be shaped by cultural values and norms regarding the expression of extreme emotions, even in extraordinary situations. Cultural concepts of distress shape the local symptom profiles of acute stress disorder. Some cultural groups may display variants of dissociative responses, such as possession or trancelike

behaviors in the initial month after trauma exposure. Panic symptoms may be salient in acute stress disorder among Cambodians because of the association of traumatic exposure with panic-like *khyâl* attacks, and *ataque de nervios* among Latin Americans may also follow a traumatic exposure. For more information regarding cultural concepts of distress, refer to the Section III chapter “Culture and Psychiatric Diagnosis.”

Sex- and Gender-Related Diagnostic Issues

Acute stress disorder is more prevalent among women than among men in studies across multiple countries. The increased risk for the disorder in women may be attributable to a greater likelihood of exposure to the types of traumatic events with a high conditional risk for acute stress disorder, such as rape, other interpersonal violence, and childhood trauma, including sexual abuse. Other factors likely contributing to the higher prevalence in women include gender differences in the emotional and cognitive processing of trauma. Sex-linked

neurobiological differences in stress response as well as sociocultural factors may also contribute to women’s increased risk for acute stress disorder.

Functional Consequences of Acute Stress Disorder

Impaired functioning in social, interpersonal, or occupational domains has been shown across survivors of accidents, assault, and rape who develop acute stress disorder. The extreme levels of anxiety that may be associated with acute stress disorder may interfere with sleep, energy levels, and capacity to attend to tasks. Avoidance in acute stress disorder can result in generalized withdrawal from many situations that are perceived as potentially threatening, which can lead to nonattendance of medical appointments, avoidance of driving to important appointments, and absenteeism from work.

Differential Diagnosis

Adjustment disorders. In adjustment disorders, the stressor can be of any severity rather than of the severity and type required by Criterion A of acute stress disorder. The diagnosis of an adjustment disorder is used when the response to a Criterion A event does not meet the criteria for acute stress disorder (or another specific mental disorder) and when the symptom pattern of acute stress disorder occurs in response to a stressor that does not meet Criterion A for exposure to actual or threatened death, serious injury, or sexual violence (e.g., spouse leaving, being fired). For example, severe stress reactions to life-threatening illnesses that may include some acute stress disorder symptoms may be more appropriately described as an adjustment disorder. Some forms of acute stress response do not include acute stress disorder symptoms and may be characterized by anger, depression, or guilt. These responses are more appropriately described as primarily an adjustment disorder. Depressive or anger responses in an adjustment disorder may involve rumination about the traumatic event, as opposed to involuntary and intrusive distressing memories in acute stress disorder.

Panic disorder. Spontaneous panic attacks are very common in acute stress disorder. However, panic disorder is diagnosed only if panic attacks are unexpected and there is anxiety about future attacks or maladaptive changes in behavior associated with fear of dire consequences of the attacks.

Dissociative disorders. Severe dissociative responses (in the absence of characteristic acute stress disorder symptoms) may be diagnosed as derealization/depersonalization disorder. If severe amnesia of the trauma persists in the absence of characteristic acute stress disorder symptoms, the diagnosis of dissociative amnesia may be indicated.

Posttraumatic stress disorder. Acute stress disorder is distinguished from PTSD because the symptom pattern in acute stress disorder must resolve within 1 month of the traumatic event. If the symptoms persist for more than 1 month and meet criteria for PTSD, the diagnosis is changed from acute stress disorder to PTSD.

Obsessive-compulsive disorder. In obsessive-compulsive disorder, there are recurrent intrusive thoughts, but these meet the definition of an obsession. In addition, the intrusive thoughts are not related to an experienced

traumatic event, compulsions are usually present, and other symptoms of acute stress disorder are typically absent.

Psychotic disorders. Flashbacks in acute stress disorder must be distinguished from illusions, hallucinations, and other perceptual disturbances that may occur in schizophrenia, other psychotic disorders, depressive or bipolar disorder with psychotic features, a delirium, substance/medication-induced disorders, and psychotic disorders due to another medical condition. Acute stress disorder flashbacks are distinguished from these other perceptual disturbances by being directly related to the traumatic experience and by occurring in the absence of other psychotic or substance-induced features.

Traumatic brain injury. When a brain injury occurs in the context of a traumatic event (e.g., traumatic accident, bomb blast, acceleration/deceleration trauma), symptoms of acute stress disorder may appear. An event causing head trauma may also constitute a psychological traumatic event, and TBI-related neurocognitive symptoms are not mutually exclusive and may occur concurrently. Symptoms previously termed *postconcussive* (e.g., headaches, dizziness, sensitivity to light or sound, irritability, concentration deficits) can occur in brain-injured and non-brain-injured populations, including individuals with acute stress disorder. Because symptoms of acute stress disorder and TBI-related neurocognitive symptoms can overlap, a differential diagnosis between acute stress disorder and neurocognitive disorder symptoms attributable to TBI may be possible based on the presence of symptoms that are distinctive to each presentation. Whereas reexperiencing and avoidance are characteristic of acute stress disorder and not the effects of TBI, persistent disorientation and confusion are more specific to TBI (neurocognitive effects) than to acute stress disorder. Furthermore, differential is aided by the fact that symptoms of acute stress disorder persist for up to only 1 month following trauma exposure.

Adjustment Disorders

Diagnostic Criteria

A. The development of emotional or behavioral symptoms in response to an identifiable stressor(s) occurring within 3 months of the onset of the stressor(s).

B. These symptoms or behaviors are clinically significant, as evidenced by one or both of the following:

   1. Marked distress that is out of proportion to the severity or intensity of the stressor, taking into account the external context and the cultural factors that might influence symptom severity and presentation.
   2. Significant impairment in social, occupational, or other important areas of functioning.

C. The stress-related disturbance does not meet the criteria for another mental disorder and is not merely an exacerbation of a preexisting mental disorder.

D. The symptoms do not represent normal bereavement and are not better explained by prolonged grief disorder.

E. Once the stressor or its consequences have terminated, the symptoms do not persist for more than an additional 6 months.

Specify whether:

F43.21 With depressed mood: Low mood, tearfulness, or feelings of hopelessness are predominant.

F43.22 With anxiety: Nervousness, worry, jitteriness, or separation anxiety is predominant.

F43.23 With mixed anxiety and depressed mood: A combination of depression and anxiety is predominant.

F43.24 With disturbance of conduct: Disturbance of conduct is predominant.

F43.25 With mixed disturbance of emotions and conduct: Both emotional symptoms (e.g., depression, anxiety) and a

disturbance of conduct are predominant.

F43.20 Unspecified: For maladaptive reactions that are not classifiable as one of the specific subtypes of adjustment disorder.

Specify if:

Acute: This specifier can be used to indicate persistence of symptoms for less than 6 months.

Persistent (chronic): This specifier can be used to indicate persistence of symptoms for 6 months or longer. By definition, symptoms cannot persist for more than 6 months after the termination of the stressor or its consequences. The persistent specifier therefore applies when the duration of the disturbance is longer than 6 months in response to a chronic stressor or to a stressor that has enduring consequences.

Specifiers

By definition, an adjustment disorder must resolve within 6 months of the termination of the stressor or its consequences. However, the symptoms may persist for a prolonged period (i.e., longer than 6 months) if they occur in response to a persistent stressor (e.g., a chronic disabling other medical condition) or to a stressor that has enduring consequences (e.g., the financial and emotional difficulties resulting from a divorce). The duration of the symptoms of an adjustment disorder can be indicated by using the acute or persistent (chronic) specifiers. The acute specifier is used to indicate persistence of symptoms for less than 6 months. The persistent (chronic) specifier is used to indicate persistence of symptoms for 6 months or longer. This latter specifier therefore applies when the duration of the disturbance is longer than 6 months in response to a persistent stressor or to a stressor that has enduring consequences.

Diagnostic Features

The presence of emotional or behavioral symptoms in response to an identifiable stressor is the essential feature of adjustment disorders (Criterion A). The stressor may be a single event (e.g., a termination of a romantic relationship), or there may be multiple stressors (e.g., marked business difficulties and marital problems). Stressors may be recurrent (e.g., associated with seasonal business crises, unfulfilling sexual relationships) or continuous (e.g., a persistent painful illness with increasing disability, living in a crime-ridden neighborhood). Stressors may affect a single individual, an entire family, or a larger group or community (e.g., a natural disaster). Some stressors may accompany specific developmental events (e.g., going to school, leaving a parental home, reentering a parental home, getting married, becoming a parent, failing to attain occupational goals, retirement).

Adjustment disorders may be diagnosed following the death of a loved one when the intensity, quality, or persistence of grief reactions exceeds what normally might be expected, when cultural, religious, or age-appropriate norms are taken into account and the grief reaction does not meet criteria for prolonged grief disorder.

Prevalence

Adjustment disorders are common, although prevalence may vary widely as a function of the population studied and the assessment methods used. The percentage of individuals in outpatient mental health treatment in the United States with a principal diagnosis of an adjustment disorder ranges from approximately 5% to 20%. Rates of adjustment disorder may be higher in women, as noted by research in Denmark. In Australian, Canadian, Israeli, and U.S. hospital psychiatric consultation settings, an adjustment disorder was often the most common diagnosis in the 1990s, frequently reaching 50%.

Development and Course

By definition, the disturbance in adjustment disorders begins within 3 months of onset of a stressor. If the stressor is an acute event (e.g., being fired from a job), the onset of the disturbance is usually immediate (i.e., within a few days) and the duration is relatively brief

(i.e., no more than a few months). If the stressor or its consequences persist, the adjustment disorder may also continue to be present and become the persistent form. By definition, if symptoms persist beyond 6 months after the stressor or its consequences have ceased, the diagnosis of adjustment disorder would no longer apply.

Risk and Prognostic Factors

Environmental. Persons from disadvantaged life circumstances experience a high rate of stressors and may be at increased risk for adjustment disorders.

Culture-Related Diagnostic Issues

Because the nature, meaning, and experience of the stressors and the evaluation of the response to stressors may vary across cultures, cultural context is key in determining whether the adjustment response is maladaptive. Migrants and refugees may experience stressful major contextual and cultural changes that can make this assessment challenging. Suffering is assumed to be an intrinsic aspect of normal life in some cultural contexts, such that distressful reactions to stressful life events may not be viewed as maladaptive or worthy of treatment. Self-immolation is also a risk associated with adjustment disorder in some cultural contexts.

Association With Suicidal Thoughts or Behavior

Adjustment disorders are associated with an increased risk of suicide attempts and suicide. Among migrant populations, including Turkish migrants in Western Europe and South Asian or South East Asian migrants in Gulf countries, adjustment disorder was found to be among the most common diagnoses associated with suicide-related behavior.

Functional Consequences of Adjustment Disorders

The subjective distress or impairment in functioning associated with adjustment disorders is frequently manifested as decreased performance at work or school and temporary changes in social relationships. An

adjustment disorder may complicate the course of illness in individuals who have another medical condition (e.g., decreased compliance with the recommended medical regimen; increased length of hospital stay).

Differential Diagnosis

Major depressive disorder. If an individual has symptoms that meet criteria for a major depressive disorder in response to a stressor, the diagnosis of an adjustment disorder is not applicable. The symptom profile of major depressive disorder differentiates it from adjustment disorders.

Posttraumatic stress disorder and acute stress disorder. In adjustment disorders, the stressor can be of any severity rather than of the severity and type required by Criterion A of acute stress disorder and posttraumatic stress disorder (PTSD). In distinguishing adjustment disorders from these two posttraumatic diagnoses, there are both timing and symptom profile considerations. Adjustment disorders can be diagnosed immediately and persist up to 6 months after exposure to the traumatic event, whereas acute stress disorder can only occur between 3 days and 1 month of exposure to the stressor, and PTSD cannot be diagnosed until at least 1 month has passed since the occurrence of the traumatic stressor. The required symptom profiles for PTSD and acute stress disorder differentiate them from the adjustment disorders. With regard to symptom profiles, an adjustment disorder may be diagnosed following a traumatic event when an individual exhibits symptoms of either acute stress disorder or PTSD that do not meet or exceed the diagnostic threshold for either disorder. Because adjustment disorder cannot persist for more than 6 months after termination of the stressor or its consequences, cases in which symptoms occurring in response

to a traumatic event that fall short of the diagnostic threshold for PTSD and that persist for longer than 6 months should be diagnosed as other specified trauma- and stressor-related disorder. An adjustment disorder should also be diagnosed for individuals who have not been exposed to a traumatic event

meeting Criterion A for PTSD, but who otherwise exhibit the full symptom profile of either acute stress disorder or PTSD.

Personality disorders. With regard to personality disorders, some personality features may be associated with a vulnerability to situational distress that may resemble an adjustment disorder. The lifetime history of personality functioning will help inform the interpretation of distressed behaviors to aid in distinguishing a long-standing personality disorder from an adjustment disorder. In addition to some personality disorders incurring vulnerability to distress, stressors may also exacerbate personality disorder symptoms. In the presence of a personality disorder, if the symptom criteria for an adjustment disorder are met, and the stress-related disturbance exceeds what may be attributable to maladaptive personality disorder symptoms (i.e., Criterion C is met), then the diagnosis of an adjustment disorder should be made.

Bereavement. Clinically significant acute bereavement-related distress may sometimes be diagnosed as an adjustment disorder if the bereavement is judged to be out of proportion to what would be expected or significantly impairs self-care and interpersonal relations. When such symptoms persist for more than 12 months after the death, the diagnosis is either prolonged grief disorder if full criteria are met or else other specified trauma- and stressor-related disorder.

Psychological factors affecting other medical conditions. In psychological factors affecting other medical conditions, specific psychological entities (e.g., psychological symptoms, behaviors, other factors) exacerbate a medical condition. These psychological factors can precipitate, exacerbate, or put an individual at risk for medical illness, or they can worsen an existing condition. In contrast, an adjustment disorder is a reaction to the stressor (e.g., having a medical illness).

Normative stress reactions. When bad things happen, most people get upset. This is not an adjustment disorder. The diagnosis should only be made when the magnitude of the distress (e.g., alterations in mood, anxiety, or conduct) exceeds what would normally be expected (which may vary in different cultures) or when the adverse event precipitates functional impairment.

Comorbidity

Adjustment disorders can accompany most mental disorders and any medical condition. Adjustment disorders can be diagnosed in addition to another mental disorder only if the latter does not explain the particular symptoms that occur in reaction to the stressor. For example, an individual may develop an adjustment disorder, with depressed mood, after losing a job and at the same time have a diagnosis of obsessive-compulsive disorder. Or, an individual may have a depressive or bipolar disorder and an adjustment disorder as long as the criteria for both are met. Adjustment disorders are common accompaniments of medical illness and may be the major psychological response to a medical condition.

Prolonged Grief Disorder

Diagnostic Criteria F43.8

A. The death, at least 12 months ago, of a person who was close to the bereaved individual (for children and adolescents, at least 6 months ago).

B. Since the death, the development of a persistent grief response characterized by one or both of the following symptoms, which have been present most days to a clinically significant degree. In addition, the symptom(s) has occurred nearly every day for at least the last month:
   1. Intense yearning/longing for the deceased person.
   2. Preoccupation with thoughts or memories of the deceased person (in children and adolescents, preoccupation may focus on the circumstances of the death).

C. Since the death, at least three of the following symptoms have been present most days to a clinically significant

degree. In addition, the symptoms have occurred nearly every day for at least the last month:

1. Identity disruption (e.g., feeling as though part of oneself has died) since the death.
2. Marked sense of disbelief about the death.
3. Avoidance of reminders that the person is dead (in children and adolescents, may be characterized by efforts to avoid reminders).
4. Intense emotional pain (e.g., anger, bitterness, sorrow) related to the death.
5. Difficulty reintegrating into one's relationships and activities after the death (e.g., problems engaging with friends, pursuing interests, or planning for the future).
6. Emotional numbness (absence or marked reduction of emotional experience) as a result of the death.
7. Feeling that life is meaningless as a result of the death.
8. Intense loneliness as a result of the death.

D. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

E. The duration and severity of the bereavement reaction clearly exceed expected social, cultural, or religious norms for the individual's culture and context.

F. The symptoms are not better explained by another mental disorder, such as major depressive disorder or posttraumatic stress disorder, and are not attributable to the physiological effects of a substance (e.g., medication, alcohol) or another medical condition.

Diagnostic Features

Prolonged grief disorder represents a prolonged maladaptive grief reaction that can be diagnosed only after at least 12 months (6 months in children

and adolescents) have elapsed since the death of someone with whom the bereaved had a close relationship (Criterion A). Although in general this time frame reliably discriminates normal grief from grief that continues to be severe and impairing, the duration of adaptive grief may vary individually and cross-culturally. The condition involves the development of a persistent grief response characterized by intense yearning or longing for the deceased person (often with intense sorrow and frequent crying) or preoccupation with thoughts or memories of the deceased, although in children and adolescents, this preoccupation may focus on the circumstances of the death. The intense yearning/longing or the preoccupation has been present most days to a clinically significant degree and has occurred nearly every day for at least the last month (Criterion B). Moreover, since the death, at least three additional symptoms have been present most days to a clinically significant degree and have occurred nearly every day for at least the past month. These symptoms include identity disruption since the death (e.g., feeling as though part of oneself has died) (Criterion C1); a marked sense of disbelief about the death (Criterion C2); avoidance of reminders that the person is dead, which in children and adolescents may be characterized by efforts to avoid reminders (Criterion C3); intense emotional pain (e.g., anger, bitterness, guilt) since the death (Criterion C4); having difficulty reintegrating into personal relationships and activities since the death (e.g., problems

engaging with friends, pursuing interests, or planning for the future) (Criterion C5); emotional numbness (absence or marked reduction of emotional experience) as a result of the death (Criterion C6); feeling that life is meaningless as a result of the death (Criterion C7); or intense loneliness as a consequence of the death (Criterion C8).

The symptoms of prolonged grief disorder must result in clinically significant distress or impairment in social, occupational, or other important areas of functioning in the bereaved individual (Criterion D). The nature, duration, and severity of the bereavement reaction must clearly exceed expected social, cultural, or religious norms for the individual’s culture and context (Criterion E). Although there are variations in how grief can

manifest, the symptoms of prolonged grief disorder occur across genders and in diverse social and cultural groups.

Associated Features

Individuals with symptoms of prolonged grief disorder often experience maladaptive cognitions about the self, guilt about the death, and diminished future life expectancy and life goals. Somatic complaints commonly accompany the condition and may be related to comorbid depression and anxiety, social identity disruption, and increased health care visits; the somatic symptoms may be associated with those that were experienced by the deceased (e.g., changes in appetite). Harmful health behaviors related to decreased self-care and concern are also common in individuals with symptoms of prolonged grief disorder. Hallucinations about the deceased (e.g., hearing the deceased person's voice) may occur during normal grief but may be more common in individuals with symptoms of prolonged grief disorder; hallucinations experienced by individuals with prolonged grief disorder symptoms may be associated with disruptions of social identity and purpose related to the death (e.g., confusion about one's role in life, feeling of meaninglessness). Other associated features of prolonged grief disorder include bitterness, anger, or restlessness; blaming others for the death; and decreased sleep quantity and quality.

Prevalence

The prevalence of DSM-5 prolonged grief disorder in adults is unknown. Meta-analysis of studies across four continents that used a different definition for prolonged grief disorder with at least a 6-month duration postloss suggests a pooled prevalence of 9.8%; however, there was substantial methodological heterogeneity across studies (e.g., in symptom definitions, measures, duration of bereavement), which affected the prevalence findings. Populations with elevated exposure to trauma may have higher prevalence rates. Mean prevalence of prolonged grief presentations may be higher in high-income Western countries than in high- and upper-middle-income Asian countries, but recent studies in China have revealed higher rates and substantial variation. Prevalence of persistent complex bereavement disorder (included in DSM-5 Section III, "Conditions

for Further Study") among bereaved U.S. youth in the community was estimated at 18%.

Development and Course

There are limited data on the course of prolonged grief disorder across the life span. Symptoms usually begin within the initial months after the death, although there may be a delay before the full syndrome appears. Preliminary evidence suggests that course may be especially prolonged among parents after the death of a child. The course of prolonged grief disorder may be complicated by comorbid posttraumatic stress disorder, which is more common in situations of bereavement following the violent death of a loved one (e.g., murder, suicide) when grief for the bereaved may be accompanied by personal life threat and/or witnessing of violent and potentially gruesome death. Older age may be associated with a higher risk of developing the disorder after the death of a loved one. Older adults with prolonged grief disorder symptoms may be at elevated risk for progressive cognitive decline.

In children, distress may be expressed in play and behavior, developmental regressions, and anxious or protest behavior at times of separation and reunion. Young children may experience symptoms of prolonged grief disorder in specific ways because of their age. The loss of a primary caregiver may be particularly traumatic for a young child, given the disorganizing effects of the caregiver's absence. Young children may protest or become angry when daily care activities are performed differently than by the deceased (e.g., cooking, discipline, bedtime rituals). They may express intense insecurity about their future, often manifested as worries about the health and safety of caregivers and about themselves, with repeated questions about death. They may engage in searching for the deceased because they do not understand the permanence of death. Young children tend toward somatic manifestations such as disturbances in sleep, eating, digestion, and level of energy. They may express yearning in thought and play as a wish, literally, to physically reunite with the deceased

to overcome the painful physical separation (e.g., to climb a ladder to heaven or lie in the ground next to a parent). Young children typically do not understand or describe numbing, whereas adolescents may describe "not feeling anything."

In children and adolescents, ongoing preoccupation with the circumstances of the death might involve focusing on distressing aspects of physical deterioration over the course of a fatal illness and/or the inability of a caregiver to perform vital caregiving functions. Identity disruption may include feeling profoundly different from others, often in response to loss reminders (e.g., making Mother's Day cards at school, watching a friend enjoy a hobby with a sibling). Children and adolescents may verbally, in their behavior, or through emotional withdrawal show reluctance to join adults in activities that serve as loss reminders. They may experience intense emotional pain over feeling deprived ("robbed") of the deceased's help with ongoing developmental tasks (e.g., onset of menses). Separation distress may be predominant in younger children, and distress over disruptions in social identity (e.g., confusion about purpose in life) and risk for comorbid depression can increasingly manifest in older children and adolescents.

Failure to achieve age-appropriate developmental milestones and transitions is a manifestation of failure to reintegrate into life roles. For older children and adolescents, feeling that life is meaningless without the person who died may include giving up on developmental aspirations ("It's not worth trying if they can't be here"), not caring about risky behavior ("So what if I get hurt or die?"), or feeling that their future is "ruined." Older children and adolescents may be apprehensive over sharing a similar fate as the deceased, including premature death. Loneliness may be intensified by keeping grief private, sometimes over not wanting to add to the distress of a grieving caregiver or to avoid presumed stigma from peers.

Risk and Prognostic Factors

Environmental. Risk for prolonged grief disorder symptoms is heightened by increased dependency on the deceased prior to the death, by the death of a child, by violent or unexpected deaths, and by economic stressors. The disorder has a higher prevalence following the death of a spouse/partner or

child compared with other kinship relationships to the deceased. Disturbances in caregiver availability and support increase the risk for bereaved children.

Culture-Related Diagnostic Issues

The symptoms of prolonged grief disorder are observed across cultural settings, but grief responses may manifest in culturally specific ways, including in expected duration, and show historical variation. For example, across cultures, nightmares about the deceased may be especially distressing because of their attributed significance; the prevalence of hallucinations of the deceased or of grief-related somatic symptoms may vary; and indirect expressions of prolonged grief disorder–related functional impairment (e.g., unhealthy

behaviors like drinking or poor self-care) may be more prevalent than direct expressions of grief. The inability to carry out funerary rituals in some cultures may worsen symptoms of prolonged grief disorder, possibly because of interpretation of their impact on the spiritual status of the deceased. Some studies suggest higher prevalence of the symptoms of prolonged grief disorder in African Americans relative to non-Hispanic Whites; the cause for these elevations requires further study in areas such as differential exposure to sudden or violent death. Differences in mourning practices may contribute to the cultural prescription or prohibition of specific grief expressions, and cultural norms about the social status of the bereaved may affect grief intensity and duration, such as different levels of support or societal sanction toward remarriage depending on the gender of the bereaved. Diagnosis of the disorder requires that the persistent and severe responses go beyond cultural norms of grief responses and not be better explained by culturally specific mourning rituals.

Sex- and Gender-Related Diagnostic Issues

Some studies find higher disorder prevalence or symptom severity among bereaved women, but other studies conclude the gender disparity is small

and/or not statistically significant.

Association With Suicidal Thoughts or Behavior

Individuals with symptoms of prolonged grief disorder are at heightened risk for suicidal ideation, even after adjustment for the effect of major depression and PTSD. The association of prolonged grief disorder symptoms and suicidal ideation is consistent across the life span and cross-nationally. However, the existing literature does not establish whether suicidal ideation associated with symptoms of prolonged grief disorder is linked to a higher incidence of suicidal behavior. Stigma, isolation, thwarted belongingness, avoidance, and psychological distress in bereaved individuals are associated with suicidal ideation. Compared with individuals whose bereavement is due to nonviolent causes, individuals whose prolonged grief disorder symptoms are the result of a violent loss (e.g., homicide, suicide, accident) are at greater risk for suicidal ideation. Similarly, individuals who experience the death of a child, especially if the child is younger than 25, are more likely to develop prolonged grief disorder symptoms that are associated with suicidal ideation.

Functional Consequences of Prolonged Grief Disorder

Symptoms of prolonged grief disorder are associated with impairments in work and social functioning and with harmful health behaviors, such as increased tobacco and alcohol use. They are also associated with marked increases in risks for serious medical conditions, including cardiac disease, hypertension, cancer, immunological deficiency, and reduced quality of life. Long-term developmental consequences among children and adolescents include premature school withdrawal, diminished educational aspirations, and reduced academic attainment; young women in particular may be hesitant to marry as they transition to adulthood. Impaired cognitive functioning may be associated with symptoms of prolonged grief disorder, especially in middle-age and older adults.

Differential Diagnosis

Normal grief.

Prolonged grief disorder is distinguished from normal grief by the presence of severe grief reactions that persist at least 12 months (6 months in children or adolescents) after the death of a person who was close to the bereaved individual. It is only when severe levels of grief response persist for the specified duration following the death, interfere with the individual's capacity to function, and exceed cultural, social, or religious norms that prolonged grief disorder is diagnosed. In evaluating the requirement for clinically significant symptoms to be present most days over the past month, it should be noted that marked increases in grief severity can be seen in normal grieving around calendar

days that are reminders of the loss, such as the anniversary of the death, birthdays, wedding anniversaries, and holidays; this exacerbation of grief severity does not by itself, in the absence of persistent grief at other times, constitute evidence of prolonged grief disorder.

Depressive disorders. Prolonged grief disorder, major depressive disorder, and persistent depressive disorder share several symptoms, including low mood, crying, and suicidal thinking. However, in prolonged grief disorder the distress is focused on feelings of loss and separation from a loved one rather than reflecting generalized low mood. Major depressive disorder may also be preceded by the death of a loved one, with or without comorbid prolonged grief disorder.

Posttraumatic stress disorder. Individuals who experience bereavement as a result of violent or accidental death may develop both PTSD and prolonged grief disorder. Both conditions can involve intrusive thoughts and avoidance. Whereas intrusions in PTSD revolve around the traumatic event (which may have caused the death of a loved one), intrusive memories in prolonged grief disorder focus on thoughts about many aspects of the relationship with the deceased, including positive aspects of the relationship and distress over the separation. Unlike avoidance in PTSD, which is manifested by avoidance of memories, thoughts, or feelings associated with the traumatic event that led to the death of the loved one (e.g., memories of the fatal automobile accident that killed the loved one), the avoidance in

prolonged grief disorder is of reminders that the loved one is no longer present (e.g., avoidance of activities carried out together with the deceased). Moreover, reexperiencing memories in PTSD tend to be more perceptual, with the individual reporting that the memory feels like it is occurring in the "here and now," which tends not to be the case in prolonged grief disorder. In prolonged grief disorder, there is also a yearning for the deceased, which is absent in PTSD.

Separation anxiety disorder. Separation anxiety disorder is characterized by anxiety about separation from current attachment figures, whereas prolonged grief disorder involves distress about separation from a deceased person.

Psychotic disorder. Hallucinations about the deceased (e.g., seeing the deceased in a favorite chair) or transient sensations about the presence of the deceased (e.g., by touch, voice, or sight) are common cross-culturally during normal grief, may be experienced as reassuring, and often occur while the individual is falling asleep (hypnagogic). To receive a diagnosis of psychotic disorder, individuals with prolonged grief disorder must also endorse other symptoms of psychosis, such as delusions, disorganized thinking, or negative symptoms.

Comorbidity

The most common comorbid disorders with symptoms of prolonged grief disorder are major depressive disorder, PTSD, and substance use disorders. PTSD is more frequently comorbid with prolonged grief disorder symptoms when the death occurred in violent or accidental circumstances. Separation anxiety disorder involving major living attachment figures may be comorbid with symptoms of prolonged grief disorder.

Other Specified Trauma- and Stressor-Related Disorder

F43.8

This category applies to presentations in which symptoms characteristic of a trauma- and stressor-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the

full criteria for any of the disorders in the trauma- and stressor-related disorders diagnostic class. The other specified trauma- and stressor-related disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific trauma- and stressor-related disorder. This is done by recording "other specified trauma- and stressor-related disorder" followed by the specific reason (e.g., "persistent response to trauma with PTSD-like symptoms").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Adjustment-like disorders with delayed onset of symptoms that occur more than 3 months after the stressor.**
2. **Adjustment-like disorders with prolonged duration of more than 6 months without prolonged duration of stressor.**
3. **Persistent response to trauma with PTSD-like symptoms** (i.e., symptoms occurring in response to a traumatic event that fall short of the diagnostic threshold for PTSD and that persist for longer than 6 months, sometimes referred to as "subthreshold/partial PTSD").
4. ***Ataque de nervios:*** See "Culture and Psychiatric Diagnosis" in Section III.
5. **Other cultural syndromes:** See "Culture and Psychiatric Diagnosis" in Section III.

Unspecified Trauma- and Stressor-Related Disorder

F43.9

This category applies to presentations in which symptoms characteristic of a trauma- and stressor-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the trauma- and stressor-related disorders diagnostic class. The unspecified trauma- and stressor-related disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific trauma- and stressor-related disorder and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Dissociative Disorders

Dissociative disorders are characterized by a disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior. Dissociative symptoms can potentially disrupt every area of psychological functioning. This chapter includes dissociative identity disorder, dissociative amnesia, depersonalization/derealization disorder, other specified dissociative disorder, and unspecified dissociative disorder.

Dissociative disorders are frequently found in the aftermath of a wide variety of psychologically traumatic experiences in children, adolescents, and adults. Throughout this chapter, “traumatic experiences” refers to experiences that result in psychological sequelae, as opposed to the physical impact that can cause traumatic brain injury. Therefore, in DSM-5, the dissociative disorders are placed next to, but are not part of, the trauma- and stressor-related disorders, reflecting the close relationship between these diagnostic classes. Both acute stress disorder and posttraumatic stress disorder include dissociative symptoms, such as amnesia, flashbacks, numbing, and depersonalization/derealization.

Dissociative symptoms are experienced as unbidden intrusions into awareness and behavior, with accompanying losses of continuity in subjective experience (i.e., ‘‘positive’’ dissociative symptoms such as division of identity, depersonalization, and derealization) and/or inability to access information or to control mental functions that normally are readily amenable to access or control (i.e., “negative” dissociative symptoms such as amnesia).

Across cultural contexts, risk factors for dissociative pathology include earlier onset of trauma; neglect and sexual, physical, and emotional abuse by parents; cumulative early life trauma and adversities; and repeated

sustained trauma or torture associated with captivity (e.g., experienced by prisoners of war, victims of trafficking).

Depersonalization/derealization disorder is characterized by clinically significant persistent or recurrent depersonalization (i.e., experiences of unreality or detachment from one's mind, self, or body) and/or derealization (i.e., experiences of unreality or detachment from one's surroundings). These alterations of experience are accompanied by intact reality testing. There is no evidence of any distinction between predominantly depersonalization and predominantly derealization symptoms. Individuals with this disorder can have depersonalization, derealization, or both.

Dissociative amnesia is characterized by an inability to recall autobiographical information that is inconsistent with normal forgetting. The amnesia may be localized (i.e., an event or period of time), selective (i.e., a specific aspect of an event), or generalized (i.e., identity and life history). In dissociative amnesia, memory deficits are primarily retrograde and often associated with traumatic experiences (e.g., lack of recall of third grade when the individual was kidnapped and held hostage). Although some individuals with amnesia promptly notice that they have gaps or a sense of fragmentation in their remote memory, most individuals with dissociative disorders are initially unaware of their amnesia or minimize or rationalize the deficits. For them, awareness of amnesia occurs when they realize that they do not recall their personal identity or when circumstances make these individuals aware that important autobiographical information is missing (e.g., when they discover

evidence or are told of past events that they cannot recall). Generalized dissociative amnesia with loss of a major part or all of the individual's life history and/or identity is rare.

Dissociative identity disorder is characterized by a) the presence of two or more distinct personality states or an experience of possession and b) recurrent episodes of dissociative amnesia. The fragmentation/division of identity may vary across cultural contexts (e.g., possession-form presentations) and with circumstance. Thus, individuals may experience discontinuities in identity and memory that may not be immediately evident

to others or are obscured by attempts to hide dysfunction. Individuals with dissociative identity disorder experience recurrent, inexplicable intrusions into their conscious functioning and sense of self (e.g., voices; dissociated actions and speech; intrusive thoughts, emotions, and impulses); alterations of sense of self (e.g., attitudes, preferences, and feeling like their body or actions are not their own); odd changes of perception (e.g., depersonalization or derealization, such as feeling detached, as if watching themself from outside their body); and intermittent functional neurological symptoms. Stress often produces transient exacerbation of dissociative symptoms that makes them more evident.

The residual category of *other specified dissociative disorder* includes presentations in which symptoms characteristic of a dissociative disorder that cause clinically significant distress or impairment predominate but do not meet the criteria for any of the specific dissociative disorders. Examples include identity disturbances associated with less-than-marked discontinuities in sense of self and agency, alterations of identity, or episodes of possession in the absence of a history of episodes of dissociative amnesia; identity disturbance due to prolonged and intensive coercive persuasion as may occur in sects/cults or terrorist organizations; acute dissociative reactions to stressful events that last less than 1 month; and dissociative trance, which is characterized by an acute narrowing or complete loss of awareness of immediate surroundings that manifests as profound unresponsiveness or insensitivity to environmental stimuli.

Dissociative Identity Disorder

Diagnostic Criteria F44.81

A. Disruption of identity characterized by two or more distinct personality states, which may be described in some cultures as an experience of possession. The disruption in identity involves marked discontinuity in sense of self and sense of agency, accompanied by related alterations in affect, behavior, consciousness, memory, perception, cognition,

and/or sensory-motor functioning. These signs and symptoms may be observed by others or reported by the individual.

B. Recurrent gaps in the recall of everyday events, important personal information, and/or traumatic events that are inconsistent with ordinary forgetting.

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The disturbance is not a normal part of a broadly accepted cultural or religious practice.

   Note: In children, the symptoms are not better explained by imaginary playmates or other fantasy play.

E. The symptoms are not attributable to the physiological effects of a substance (e.g., blackouts or chaotic behavior during alcohol intoxication) or another medical condition (e.g., complex partial seizures).

Diagnostic Features

The defining feature of dissociative identity disorder is the presence of two or more distinct personality states or an experience of possession (Criterion A). The overtness or covertness of these personality states varies as a function of psychological motivation, current level of stress, cultural context, internal conflicts and dynamics, and emotional resilience, among other factors. Sustained periods of identity confusion/alteration may occur when psychosocial pressures are severe and/or prolonged. In those cases of dissociative identity disorder that present as the individual being possessed by external identities (e.g., spirits, demons) (possession-form dissociative identity disorder), and in a small proportion of non-possession-form cases, manifestations of alternate identities are readily observable. Most individuals with non-possession-form dissociative identity disorder do not overtly display, or only subtly display, their discontinuity of identity, and

only a minority present to clinical attention with discernible alternation of identities. The elaboration of dissociative personality states with different names, wardrobes, hairstyles, handwritings, accents, and so forth, occurs in only a *minority* of individuals with the non-possession-form dissociative identity disorder and is *not* essential to diagnosis. In those cases where alternate personality states cannot be directly observed, the presence of distinct personality states can be identified by sudden alterations or discontinuities in the individual's sense of self and sense of agency (Criterion A), and recurrent dissociative amnesias (Criterion B).

Criterion A symptoms are related to discontinuities of experience that can affect any aspect of an individual's functioning. Individuals with dissociative identity disorder may report the feeling that they have suddenly become depersonalized observers of their own speech and actions, which they may feel powerless to stop (i.e., impaired sense of self and impaired sense of agency). These individuals may also report perceptions of voices (e.g., a child's voice, voices commenting on the individual's thoughts or behavior, persecutory voices and command hallucinations). In some cases, hearing voices is specifically denied, but the individual reports multiple, perplexing, independent thought streams over which the individual experiences no control. Individuals with dissociative identity disorder may report hallucinations in all sensory modalities: auditory, visual, tactile, olfactory, and gustatory.

Strong emotions, impulses, thoughts, and even speech or other actions may suddenly materialize, without a sense of personal ownership or control (i.e., lack of sense of agency). Conversely, thoughts and emotions may unexpectedly vanish, and speech and actions are abruptly inhibited. These experiences are frequently reported as ego-dystonic and puzzling. Attitudes, outlooks, and personal preferences (e.g., about food, activities, gender identity) may suddenly shift. Individuals may report that their bodies feel different (e.g., like a small child, the opposite gender, different ages simultaneously). Alterations in sense of self and agency may be accompanied by a feeling that attitudes, emotions, and behaviors—even the individual's own body—are "not mine" or are "not under my control." Although most Criterion A symptoms are subjective, these sudden discontinuities in speech, affect, and behavior may be witnessed by family, friends, or the clinician.

In most individuals with dissociative identity disorder, switching/shifting of states is subtle and may occur with only subtle changes in overt presentation. State switching may be more overt in the possession form of dissociative identity disorder. In general, the individual with dissociative identity disorder experiences himself or herself as multiple, simultaneously overlapping and interfering states.

Dissociative amnesia (Criterion B) manifests in several major domains: 1) gaps in any aspect of autobiographical memory (e.g., important life events like getting married or giving birth, lack of recall of all school experiences before high school); 2) lapses in memory of recent events or well-learned skills (e.g., how to do one's job, use a computer, cook or drive); and 3) discovery of possessions that the individual has no recollection of ever owning (e.g., clothing, weapons, tools, writings or drawings that he or she must have created).

Dissociative fugues, with amnesia for travel, are common. Individuals may report suddenly finding themselves in another city, at work, or even at home: in the closet, under the bed, or running out of the house. Amnesia in individuals with dissociative identity disorder is not limited to stressful or traumatic events; it can involve everyday events as well. Individuals may report major gaps in ongoing memory (e.g., experiencing "time loss," "blackouts," or "coming to" in the midst of doing something). Dissociative amnesia may be apparent to others (e.g., the individual does not recall something others witnessed that he or she did or said, cannot remember his or her own name, or may fail to recognize spouse, children, or close friends). Minimization or rationalization of amnesia is common.

Possession-form identities in dissociative identity disorder typically manifest behaviorally as if a "spirit," supernatural being, or outside person has taken control, with the individual speaking or acting in a distinctly different manner. For example, an individual's behavior may give the appearance that her identity has been replaced by the "ghost" of a girl who died by suicide in the same community years before, speaking and acting as though she were still alive. The identities that arise during possession-form dissociative identity disorder present recurrently, are unwanted and

involuntary, and cause clinically significant distress or impairment (Criterion C). However, the majority of possession states that occur around the world are usually part of a broadly accepted cultural or religious practice and therefore do not meet criteria for dissociative identity disorder (Criterion D).

Associated Features

Individuals with dissociative identity disorder typically present with comorbid depression, anxiety, substance abuse, self-injury, or another common symptom. Nonepileptic seizures and other functional neurological symptoms are prominent in some presentations of dissociative identity disorder, especially in some non-Western settings. Some individuals, especially in Western settings, may present with apparently refractory neurological symptoms, such as headaches, seizures, or symptoms suggestive of multiple sclerosis.

Individuals with dissociative identity disorder often conceal, or are not fully aware of, disruptions in consciousness, amnesia, or other dissociative symptoms. Many individuals with dissociative identity disorder report dissociative flashbacks during which they experience a sensory reliving of a previous event as though it were occurring in the present, often with a change of identity, a partial or complete loss of contact with or disorientation to current reality during the flashback, and a subsequent amnesia for the content of the flashback. Individuals with the disorder typically report multiple types of interpersonal maltreatment during childhood and adulthood. Other overwhelming early life events, such as multiple long, painful, early-life medical procedures, also may be reported. Nonsuicidal self-injury is frequent. On standardized measures, these individuals report higher levels of hypnotizability and dissociative symptoms compared with other clinical groups and healthy control subjects. Some individuals experience transient psychotic phenomena or episodes.

Among personality features, avoidant personality features most often rate highest in individuals with dissociative identity disorder, and some individuals with dissociative identity disorder are so avoidant that they prefer to be alone. When decompensated, some individuals with dissociative identity disorder display features of borderline personality

disorder (i.e., self-destructive high-risk behaviors, and mood instability). Many individuals with dissociative identity disorder display attachment problems but typically do not exhibit frantic activity to avoid being abandoned. Some have stable long-term relationships, albeit frequently dysfunctional and/or abusive ones, from which they may have difficulty extricating themselves. Obsessional personality features are common in dissociative identity disorder, more so than histrionic personality features. A subgroup of

individuals with dissociative identity disorder have narcissistic and/or antisocial personality features.

Prevalence

The 12-month prevalence of dissociative identity disorder among adults in a small U.S. community study was 1.5%. Lifetime prevalence of dissociative identity disorder was 1.1% in a representative sample of community-based women in mid-eastern Turkey.

Development and Course

The disorder may first manifest at almost any age from early childhood to late life. Children usually do not present with identity shifting, instead presenting primarily with independently acting, imaginary companions, or as personified “mood” states (Criterion A phenomena). Dissociation in children may generate problems with memory, concentration, and attachment, and may be associated with traumatic play. In adolescents, dissociative identity disorder commonly comes to clinical attention because of externalizing symptoms, suicidal/self-destructive behavior, or rapid behavioral shifts often ascribed to other disorders such as attention-deficit/hyperactivity disorder or childhood bipolar disorder. Some children with dissociative identity disorder can also be quite aggressive and irritable. Older individuals with dissociative identity disorder may present with symptoms that appear to be late-life mood disorders, obsessive-compulsive

disorder, paranoia, psychotic mood disorders, or even cognitive disorders attributable to dissociative amnesia.

Overt identity alteration/confusion may be triggered by many factors, such as later traumatic experiences (e.g., sexual assault), or even seemingly inconsequential stressors, like a minor motor vehicle accident. The experience of other major or cumulative life stressors may also worsen symptoms, including life events such as the individual's children reaching the same age at which the individual was significantly abused or traumatized. The death of, or the onset of a fatal illness in, the individual's abuser(s) is another example of an event that may worsen symptoms. Individuals with dissociative identity disorder are at high risk for adult interpersonal trauma such as rape, intimate partner violence, and sexual exploitation, including ongoing incestuous abuse into adulthood, as well as adult trafficking.

Risk and Prognostic Factors

Environmental. In the context of family and attachment pathology, early life trauma (e.g., neglect and physical, sexual, and emotional abuse, usually before ages 5–6 years) represents a risk factor for dissociative identity disorder. In studies from diverse geographic regions, about 90% of the individuals with the disorder report multiple types of early neglect and childhood abuse, often extending into late adolescence. Some individuals report that maltreatment primarily occurred outside the family, in school, church, and/or neighborhoods, including being bullied severely. Other forms of repeated early-life traumatic experiences include multiple, painful childhood medical and surgical procedures; war; terrorism; or being trafficked beginning in childhood. Onset has also been described after prolonged and often transgenerational exposure to dysfunctional family dynamics (e.g., overcontrolling parenting, insecure attachment, emotional abuse) in the absence of clear neglect or sexual or physical abuse.

Genetic and physiological. Twin studies suggest that genetics account for around 45%–50% of the interindividual variance in dissociative symptoms, with nonshared, stressful, and traumatic environmental experiences accounting for most of the additional variance. Several brain regions have been implicated in the pathophysiology of dissociative identity disorder,

including the orbitofrontal cortex, hippocampus, parahippocampal gyrus, and amygdala.

Course modifiers. Ongoing sexual, physical, and emotional trauma often leads to significant difficulties in later functioning. Poorer outcome in adults is commonly related to severe psychosocial stressors, revictimization, ongoing sexual or physical abuse or exploitation, intimate partner violence, refractory substance use, eating disorders, severe medical illness, enmeshment with the individual's abusive family of origin, or ongoing involvement in criminal subgroups. Poorer functioning may also be related to perpetration of child maltreatment or intimate partner violence by individuals with dissociative identity disorder.

Culture-Related Diagnostic Issues

Many features of dissociative identity disorder can be influenced by the individual's sociocultural background. In settings where possession symptoms are common (e.g., rural areas in low- and middle-income countries, among certain religious groups in the United States and Europe), all or some of the fragmented identities may take the form of possessing spirits, deities, demons, animals, or mythical figures. Acculturation or prolonged intercultural contact may shape the presentation of the other identities (e.g., identities in India may speak English exclusively and wear Western clothes). Possession-form dissociative identity disorder can be distinguished from culturally accepted possession states in that the former is involuntary, distressing, and uncontrollable; involves conflict between the individual and his or her surrounding family, social, or work milieu; and is manifested at times and in places that violate cultural or religious norms. Combined dissociative-psychosis episodes may be more common in cultural contexts with marked communal violence or oppression and limited opportunity for redress.

Sex- and Gender-Related Diagnostic Issues

Women with dissociative identity disorder predominate in adult clinical settings but not in child/adolescent clinical settings or in general population studies. Few differences in symptom profiles, clinical history, and childhood trauma history have been found in comparisons between men and women with dissociative identity disorder, except that women may have higher rates of somatization.

Association With Suicidal Thoughts or Behavior

Suicidal behavior is frequent. Over 70% of outpatients with dissociative identity disorder have attempted suicide; multiple attempts are common, and other self-injurious and high-risk behaviors are highly prevalent. Individuals with dissociative identity disorder have multiple interacting risk factors for self-destructive and/or suicidal behavior. These include cumulative, severe early- and later-life trauma; high rates of comorbid posttraumatic stress disorder (PTSD), depressive disorders, and substance use disorders; and personality disorder features. Dissociation itself is an independent risk factor for multiple suicide attempts. Greater severity of dissociative symptom scores is associated with a higher frequency of suicide attempts and nonsuicidal self-injury among individuals with dissociative disorders.

Functional Consequences of Dissociative Identity Disorder

Some children and adolescents with dissociative identity disorder may function poorly in school and in relationships. Others do well in school, experiencing it as a respite. In adults impairment varies widely, from apparently minimal (e.g., in high-functioning professionals) to profound. The symptoms of higher-functioning individuals may impair their relational, marital, family, and parenting functions more than their occupational and professional life, although the latter also may be affected. Many impaired individuals show improvement in occupational and personal functioning over time, while some individuals with dissociative

identity disorder may be impaired in most activities of living and function at the level of chronic and persistent mental illness.

Differential Diagnosis

Dissociative amnesia. Both dissociative identity disorder and dissociative amnesia are characterized by gaps in the recall of everyday events, important personal information, or traumatic events that are inconsistent with ordinary forgetting. Dissociative identity disorder is distinguished from dissociative amnesia by the additional presence of identity disruption characterized by two or more distinct personality states.

Depersonalization/derealization disorder. The essential feature of depersonalization/derealization disorder is persistent or recurrent episodes of depersonalization, derealization, or both. Individuals with depersonalization/derealization disorder do not experience the presence of personality/identity states with alterations of self and agency, nor do they typically report dissociative amnesia.

Major depressive disorder. Most individuals with dissociative identity disorder endorse a lifelong negative posttraumatic emotional state, often with childhood onset, and their symptoms may appear to meet the criteria for a major depressive episode. Moreover, posttraumatic reactivity to times of year when trauma occurred (anniversary reactions), primarily manifesting with more despondency, distress, and suicidal ideation, may also appear to be major depressive disorder, with seasonal pattern. However, individuals with major depressive disorder or persistent depressive disorder do not experience dissociative fluctuations in self and agency and dissociative amnesia. It is important to assess if all or most identity states are experiencing the adverse mood state, since mood disorder symptoms may fluctuate because they are experienced in some identity states, but not others.

Bipolar disorders. Dissociative identity disorder is commonly misdiagnosed as bipolar disorder, typically bipolar II disorder, with mixed features. The relatively rapid shifts in behavioral state in individuals with dissociative identity disorder—usually within minutes or hours—are atypical for even the most rapid-cycling individuals with bipolar disorders. These state

alterations are due to rapidly shifting dissociative states and/or fluctuating posttraumatic intrusions. Sometimes these shifts are accompanied by rapid changes in levels of activation, but these usually last minutes to hours, not days, and are associated with activation of specific identity states. Elevated or depressed mood may be experienced as loculated in specific identities, through overlap/interference phenomena. Usually, the individual with dissociative identity disorder does not have a classic bipolar sleep disturbance (e.g., reduced need for sleep), instead suffering from chronic, severe nightmares and nocturnal flashbacks that interrupt sleep.

Posttraumatic stress disorder. A majority of individuals with dissociative identity disorder will have symptoms that meet diagnostic criteria for comorbid PTSD. Dissociative symptoms characteristic of dissociative identity disorder should be differentiated from the dissociative amnesia, dissociative flashbacks, and depersonalization/derealization characteristic of acute stress disorder, PTSD, or the dissociative subtype of PTSD. Dissociative amnesia in PTSD typically manifests only for specific traumatic events or aspects of traumatic events, as opposed to the chronic, complex dissociative amnesia characteristic of dissociative identity disorder. Depersonalization/derealization symptoms in the dissociative subtype of PTSD are related to specific posttraumatic reminders. Depersonalization/derealization symptoms in dissociative identity disorder may occur not only in response to posttraumatic reminders, but also in an ongoing fashion in daily life, including in response to stressful interpersonal interactions and when there is overlap/interference between states.

Schizophrenia and other psychotic disorders. Individuals with dissociative identity disorder may experience symptoms that can superficially appear similar to those of psychotic

disorders. These include auditory hallucinations and symptoms characteristic of intrusions of personality states into the individual’s awareness; these symptoms can seemingly resemble some of the Schneiderian first-rank symptoms formerly considered indicative of schizophrenia (e.g., thought broadcasting, thought insertion, thought

withdrawal, hearing voices keeping up a running commentary about the individual). For example, hearing different personality states discussing the individual can resemble auditory hallucinations of voices arguing in schizophrenia. The individual with dissociative identity disorder may also experience the thoughts or emotions of an intruding personality state, which can resemble thought insertion in schizophrenia, as well as experience the sudden disappearance of these thoughts or emotions, which can resemble thought withdrawal. Such experiences in an individual with schizophrenia are usually accompanied by delusional beliefs about the cause of those symptoms (i.e., thoughts being inserted by an outside force), whereas individuals with dissociative identity disorder typically experience these symptoms as ego-alien and frightening. Individuals with dissociative identity disorder may also report a range of visual, tactile, olfactory, gustatory, and somatic hallucinations, which are usually related to autohypnotic, posttraumatic, and dissociative factors, such as partial flashbacks, in contrast to individuals with schizophrenia, whose hallucinations are primarily auditory and less commonly visual. Dissociative identity disorder and psychotic disorders are therefore distinguished by symptoms characteristic of one of these conditions and not the other (e.g., dissociative amnesia in dissociative identity disorder and not in psychotic disorders). Finally, individuals with schizophrenia have low hypnotic capacity, whereas individuals with dissociative identity disorder have the highest hypnotic capacity among all clinical groups.

Substance/medication-induced disorders. Individuals with dissociative identity disorder frequently have a current or past history of substance use disorders. Symptoms associated with the physiological effects of a substance (e.g., blackouts) should be distinguished from dissociative amnesia in dissociative identity disorder if the substance in question is judged to be etiologically related to the memory loss.

Personality disorders. Individuals with dissociative identity disorder often present identities that appear to encapsulate a variety of severe personality disorder features, suggesting a differential diagnosis of personality disorder, especially of the borderline type. Importantly, however, the individual's longitudinal variability in personality style (attributable to inconsistency among identities) differs from the pervasive and persistent dysfunction in

affect management and interpersonal relationships typical of those with personality disorders.

Posttraumatic amnesia due to brain injury. Both dissociative identity disorder and traumatic brain injury (TBI) are characterized by gaps in memory. Other characteristics of TBI include loss of consciousness, disorientation and confusion, or, in more severe cases, neurological signs and symptoms. A neurocognitive disorder due to TBI manifests either immediately after brain injury occurs or immediately after the individual recovers consciousness after the injury, and persists past the acute postinjury period. The cognitive presentation of a neurocognitive disorder following TBI is variable and includes difficulties in the domains of complex attention, executive function, and learning and memory, as well as slowed speed of information processing and disturbances in social cognition. While depersonalization is not uncommon following TBI, the additional neurocognitive features noted above help distinguish it from dissociative amnesia that is part of dissociative identity disorder. Moreover, dissociative amnesia occurring in the context of dissociative identity disorder is accompanied by a marked discontinuity in sense of self and sense of agency, which are not features of TBI.

Functional neurological symptom disorder (conversion disorder). Functional neurological symptom disorder may be distinguished from dissociative identity disorder by the absence of identity alteration characterized by two or more distinct personality states or an

experience of possession. Dissociative amnesia in functional neurological symptom disorder is more limited and circumscribed (e.g., amnesia for a nonepileptic seizure).

Factitious disorder and malingering. Individuals who feign dissociative identity disorder usually do not report the subtle symptoms of intrusion characteristic of the disorder; instead they tend to overreport media-based symptoms of the disorder, such as dramatic dissociative amnesia and melodramatic switching behaviors, while underreporting less-publicized comorbid symptoms, such as depression. Individuals who feign dissociative

identity disorder tend to be relatively undisturbed by or may even seem to enjoy "having" the disorder, or may ask clinicians to "find" traumatic memories. In contrast, most individuals with genuine dissociative identity disorder are ashamed of and overwhelmed by their symptoms, deny the diagnosis, underreport their symptoms, and display minimization and avoidance of their trauma history.

Individuals who feign the symptoms of dissociative identity disorder usually create limited, stereotyped alternate identities, with feigned amnesia related only to the events for which gain is sought, with apparent switching behaviors and amnesia only displayed while being observed. They may present an "all-good" identity and an "all-bad" identity in hopes of gaining exculpation for a crime.

Comorbidity

Disorders that are comorbid with dissociative identity disorder include PTSD, depressive disorders, substance-related disorders, feeding and eating disorders, obsessive-compulsive disorder, antisocial personality disorder, and other specified personality disorder with avoidant, obsessive-compulsive, or borderline personality traits. The most common forms of functional neurological symptom disorder include nonepileptic seizures, gait disturbances, and paralyses. Most commonly, nonepileptic seizures resemble grand mal seizures or complex partial seizures with temporal lobe foci; others may mimic absence or partial seizures.

Dissociative Amnesia

Diagnostic Criteria F44.0

A. An inability to recall important autobiographical information, usually of a traumatic or stressful nature, that is inconsistent with ordinary forgetting.

Note: Dissociative amnesia most often consists of localized or selective amnesia for a specific event or events; or

generalized amnesia for identity and life history.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The disturbance is not attributable to the physiological effects of a substance (e.g., alcohol or other drug of abuse, a medication) or a neurological or other medical condition (e.g., partial complex seizures, transient global amnesia, sequelae of a closed head injury/traumatic brain injury, other neurological condition).

D. The disturbance is not better explained by dissociative identity disorder, posttraumatic stress disorder, acute stress disorder, somatic symptom disorder, or major or mild neurocognitive disorder.

Coding note: The code for dissociative amnesia without dissociative fugue is **F44.0.** The code for dissociative amnesia with dissociative fugue is **F44.1.**

Specify if:

F44.1 With dissociative fugue: Apparently purposeful travel or bewildered wandering that is associated with amnesia for identity or for other important autobiographical information.

Specifiers

The specifier “with dissociative fugue” applies when dissociative amnesia occurs in the context of a dissociative fugue, which is characterized by apparently purposeful travel or bewildered wandering that is associated with amnesia for identity or other important autobiographical information.

Diagnostic Features

The defining characteristic of dissociative amnesia is an inability to recall important autobiographical information that 1) should be successfully

stored in memory and 2) ordinarily would be freely recollected (Criterion A). Dissociative amnesia is conceptualized as a potentially reversible memory retrieval deficit. In this way, among others, it differs from the amnesias attributable to neurobiological damage or toxicity that impair memory storage or retrieval.

A variety of types of dissociative amnesia may manifest. In general, the memory deficit in dissociative amnesia is *retrograde* and, except in rare cases, is not associated with ongoing amnesia for contemporary life events. *Retrospective memory impairments* include not only lost memories of traumatic experiences but also lost memories of everyday life during which no trauma occurred. Most commonly, individuals with dissociative amnesia report *localized amnesia*—a failure to recall events during a circumscribed period of time; and/or *selective amnesia*—the individual can recall some, but not all, of the events during a circumscribed period of time. In *systematized amnesia* the individual fails to recall a specific category of important information (e.g., fragmentary recall of home growing up, but continuous memory for school; no recall of a violent older sibling; lack of recall of a specific room in the individual's childhood home). Individuals rarely overtly complain of symptoms of these forms of dissociative amnesia and attempt to minimize and rationalize the memory loss.

Generalized dissociative amnesia involves a complete loss of memory for most or all of the individual's life history. Individuals with generalized amnesia may forget personal identity (e.g., a woman loses memory of her entire life history after giving in to repeated pressure from a close friend to engage in a sexual relationship), lose previous knowledge about the world (e.g., recent political events, how to use current technology), and less commonly lack access to well-learned skills (e.g., what contact lenses are and how to put them in). Generalized dissociative amnesia has an acute onset; the perplexity, disorientation, and purposeless wandering of individuals with generalized amnesia usually bring them to the attention of the police or psychiatric emergency services. Dissociative fugue is commonly associated with generalized dissociative amnesia and can be indicated by using the "with dissociative fugue" specifier. Generalized dissociative amnesia may be more common among combat veterans, sexual assault victims, and individuals experiencing extreme emotional stress or

conflict. In continuous amnesia (i.e., anterograde dissociative amnesia), an individual forgets each new event as it occurs.

Individuals with dissociative amnesia are frequently unaware (or only partially aware) of their memory problems. They may recall some traumatic events, or parts of traumatic events, but not others of the same type. Many, especially those with localized amnesia, minimize the importance of their memory loss and may become uncomfortable when prompted to address it.

Associated Features

Many individuals with dissociative amnesia are chronically impaired in their ability to form and sustain satisfactory relationships. Histories of trauma, especially child abuse, and victimization are common. Some individuals with dissociative amnesia report dissociative flashbacks (i.e., behavioral reexperiencing of traumatic events). Many have a history of

nonsuicidal self-injury, suicide attempts, and other high-risk behaviors. Depressive and functional neurological symptoms are common, as are depersonalization, auto-hypnotic symptoms, and high hypnotizability. Sexual dysfunctions are common. Mild traumatic brain injury (TBI) may precede dissociative amnesia.

Prevalence

The 12-month prevalence for dissociative amnesia among adults in a small U.S. community study was 1.8%.

Development and Course

Dissociative amnesia has been observed in young children, adolescents, adults, and geriatric populations. Amnesia in children younger than 12 may be the most difficult to evaluate because they often have difficulty understanding questions about amnesia, and interviewers may find it difficult to formulate child-friendly questions about memory and amnesia, especially in younger children. Observations of apparent dissociative amnesia are often difficult to differentiate from inattention, absorption,

daydreaming, anxiety, oppositional behavior, and learning disorders. Reports from several different sources (e.g., teacher, therapist, case worker) may be needed to diagnose amnesia in children. Some traumatized adolescents with dissociative amnesia are less likely to come to clinical attention because of lower levels of posttraumatic stress disorder (PTSD) intrusive symptoms and less externalizing behavior. Dissociative fugue behavior in children and adolescents may be limited by the child's life space (e.g., a child in a fugue "coming to" after bicycling to an unfamiliar neighborhood, a teenager finding herself having taken public transportation to a nearby town).

Onset of generalized amnesia is usually sudden. Individuals may experience multiple episodes of this type of dissociative amnesia. A single episode may predispose to future episodes. In between episodes of amnesia, the individual may or may not appear to be acutely symptomatic. Some episodes of acute generalized amnesia resolve rapidly (e.g., when the individual is removed from combat or some other stressful situation, and/or is brought to clinical attention). A substantial subgroup of individuals develop highly impairing, debilitating, chronic autobiographical memory deficits, such that even "relearning" their life history does not ameliorate the memory loss.

Removal from the traumatic circumstances generating acute, generalized dissociative amnesia (e.g., combat) may bring about a rapid return of memory. The memory loss of individuals with dissociative fugue may be particularly refractory. Later life trauma, life stresses, or losses may precede breakdown of long-standing autobiographical memory deficits related to childhood or adult trauma, with the onset of acute PTSD, mood disorders, substance abuse, and dangerousness to self or others, among other symptoms.

Risk and Prognostic Factors

Environmental. Severe, acute, or chronic traumatization is the main risk factor for dissociative amnesia. Cumulative early life trauma and adversities, especially physical and sexual abuse, are the major risk factors for dissociative amnesia for childhood and adolescence. More severe sexual abuse, multiple episodes of childhood sexual abuse, and sexual abuse by a

relative, especially with betrayal by a close attachment figure, may increase the extent of childhood autobiographical memory disturbances. Individuals with dissociative amnesia may deny recall of specific childhood traumas (e.g., sexual assault), even those documented in medical or social service reports, although the individual can recall other similar traumatic events, both before and after the amnestic event. Severe cumulative adult trauma (e.g., repeated combat, trafficking, prisoner-of-war or concentration camp

experiences) also may result in extensive localized, selective, and/or systematized dissociative amnesia. Generalized dissociative amnesia may be more common among individuals who have recently experienced extreme acute traumas (e.g., brutal military combat, rape, torture, often in the context of inability to escape) and/or a prior history of major social dislocation, asylum-seeking, or refugee status. Others develop generalized amnesia in the context of profound psychological conflict from which the individual also feels unable to escape. Virtually all individuals who develop generalized dissociative amnesia in the context of psychological conflict report past histories of severe early life and/or adult traumatization. Extreme acute traumatic experiences also may engender major psychological conflicts (e.g., a woman develops generalized amnesia after experiencing a brutal rape resulting in an unwanted pregnancy and becomes suicidal; on assessment, she reveals that her religion views abortion as murder and suicide as a major sin).

Genetic and physiological. Quantitative genetic studies suggest that genetics account for about 50% of the interindividual variance in dissociative symptoms, with nonshared, stressful environmental experiences accounting for most of the additional variance. Candidate gene studies suggest a gene x environment interplay with earlier and more chronic childhood traumatic experiences leading to significant increases in dissociative symptoms later in life.

Culture-Related Diagnostic Issues

In cultural contexts where possession is part of normative religious or spiritual practice, dissociative amnesia and fugue may be interpreted as resulting from pathological possession. In contexts or situations where individuals feel highly constrained by social circumstances or cultural traditions, the precipitants of dissociative amnesia often do not involve frank trauma. Instead, the amnesia may be preceded by severe psychological stresses or conflicts (e.g., marital conflict, other family disturbances, attachment problems, or conflicts attributable to restriction or oppression).

Association With Suicidal Thoughts or Behavior

Suicidal and other self-destructive behaviors are common in individuals with dissociative amnesia. The psychological forces producing generalized amnesia may be extreme, and suicidal thoughts, impulses, plans, and behavior are a risk when amnesia decreases. Case reports suggest that suicidal behavior may be a particular risk when the amnesia remits suddenly and overwhelms the individual with intolerable memories.

Functional Consequences of Dissociative Amnesia

Impairments in individuals with dissociative amnesia resulting from childhood/adolescent traumatization range from limited to severe. Some of these individuals may be chronically impaired in their ability to form and sustain satisfactory attachments. Some may become highly successful in occupational functioning but often do so by compulsive overwork. Individuals with acute generalized dissociative amnesia usually have impairment in all aspects of functioning. A substantial subgroup of individuals with generalized amnesia develop a highly impairing, chronic autobiographical memory deficit that even relearning their life history does not ameliorate. These individuals experience a highly debilitated, chronic course with poor overall functioning in most domains of life.

Differential Diagnosis

Dissociative identity disorder. Recurrent episodes of dissociative amnesia may be attributable to dissociative identity disorder. Individuals with

dissociative amnesia may report depersonalization and auto-hypnotic symptoms, as do individuals with dissociative

identity disorder. Individuals with dissociative identity disorder report pervasive discontinuities in sense of self and agency, accompanied by many other dissociative symptoms. Amnesias in dissociative identity disorder, in addition to retrospective autobiographical memory deficits, include ongoing amnesia (“time loss”) for everyday events and interpersonal interactions; finding unexplained possessions; perplexing major fluctuations in skills and knowledge; and frequent, brief amnesic gaps during interpersonal interactions.

Posttraumatic stress disorder. Some individuals with PTSD cannot recall part or all of a specific traumatic event (e.g., a rape victim who cannot recall most events for the entire day of the rape). When that amnesia extends to events beyond the immediate time of the trauma, a comorbid diagnosis of dissociative amnesia may be warranted. Individuals with the dissociative subtype of PTSD may also report dissociative amnesia in addition to depersonalization/derealization.

Neurocognitive disorders. In major neurocognitive disorders, there is typically evidence of neural tissue damage accompanied by a decline in cognitive function with deficits in attention, executive function, learning and memory, language, and perceptual-motor and social cognition that impair capacity for independent everyday activities. Memory loss for personal information is usually embedded in cognitive, linguistic, affective, attentional, and behavioral disturbances. Generally, awareness of personal identity is spared until late in the course of the neurocognitive disorder. In neurocognitive disorders, retrograde amnesia is almost always accompanied by anterograde amnesia. Anterograde dissociative amnesia can be confused with delirium. However, medical, laboratory, toxicological, and neurological workups, including imaging studies, are normal. Careful, repeated evaluations over time will show that as in other forms of dissociative amnesia, there are no true neurocognitive deficits.

Substance-related disorders.

In the context of repeated intoxication with alcohol or other substances/medications, there may be episodes of “blackouts” or periods for which the individual has no memory, or partial memory (“grayouts”). To aid in distinguishing these episodes from dissociative amnesia, a longitudinal history should show that the amnestic episodes occur only in the context of intoxication. However, the distinction may be difficult when the individual with dissociative amnesia also misuses alcohol or other substances, particularly in the context of stressful situations that may also exacerbate dissociative symptoms. This can be a more complex differential diagnosis when the substance use begins in childhood or adolescence, generally in the context of intrafamilial abuse, neglect, and substance-related disorders. Sequential observation of these individuals after detoxification, along with carefully taken history, usually can distinguish the memory loss attributable to long-standing substance use from dissociative amnesia. Some individuals with comorbid dissociative amnesia and substance use disorders will attempt to minimize their dissociative amnesia and attribute memory problems solely to the substance use. Prolonged use of alcohol or other substances may result in a substance-induced neurocognitive disorder that may be associated with impaired cognitive function. However, in this context the protracted history of substance use and the persistent deficits associated with the neurocognitive disorder would serve to distinguish it from dissociative amnesia, where there is typically no evidence of persistent impairment in intellectual functioning.

Posttraumatic amnesia due to brain injury. Amnesia may occur in the context of a TBI when there has been an impact to the head or other mechanisms of rapid movement or displacement of the brain within the skull. Other characteristics of TBI include loss of consciousness, disorientation and confusion, or, in more severe cases, neurological signs and symptoms (e.g., abnormalities on neuroimaging, a new onset of seizures or a marked worsening of a preexisting seizure disorder, visual field cuts, anosmia). A neurocognitive disorder attributable to TBI must present either immediately after brain injury occurs or immediately after the individual recovers consciousness after the injury, and persist past the

acute postinjury period. The cognitive presentation of a neurocognitive disorder following TBI is variable and includes difficulties in the domains of complex attention, executive function, learning and memory, as well as slowed speed of information processing and disturbances in social cognition. The patterns of memory deficits are typical of neurocognitive disorders. Mild TBI may precede acute dissociative amnesia presentations, but the dissociative memory deficits are out of proportion to the TBI head trauma and typically follow the dissociative, not the neurocognitive, patterns.

Seizure disorders. Individuals with seizure disorders may exhibit complex behavior during seizures or postictally with subsequent amnesia. Some individuals with a seizure disorder engage in nonpurposive wandering that is limited to the period of seizure activity. Conversely, behavior during a dissociative fugue is usually purposeful, complex, and goal-directed and may last for days, weeks, or longer. Occasionally, individuals with a seizure disorder will report that some autobiographical memories have been "wiped out" as the seizure disorder progresses. Such memory loss is not associated with psychological trauma or adversities and appears to occur randomly. In seizure disorders, serial electroencephalograms usually show abnormalities. Telemetric electroencephalographic monitoring generally shows an association between the episodes of amnesia and seizure activity. Dissociative and epileptic amnesias may coexist.

Memory deficits associated with electroconvulsive therapy. Memory deficits after electroconvulsive therapy (ECT) most commonly occur for the day of ECT administration. More extensive retrograde and even anterograde amnesia after ECT is usually unrelated to stressful or traumatic life epochs, and generally remits after the ECT series concludes. ECT in severely depressed individuals with dissociative disorders does not worsen dissociation, and memory access may improve as depression remits.

Catatonic stupor. Mutism in catatonic stupor may suggest dissociative amnesia, but failure of recall is usually absent. Other catatonic symptoms (e.g., rigidity, posturing, negativism) are usually present. Catatonic symptoms in children can be associated with trauma, abuse, and/or

deprivation. Unlike in dissociative amnesia, the pattern of memory loss in catatonia is only for the catatonic episode.

Acute dissociative reactions to stressful events (other specified dissociative disorder).

The acute dissociative reactions to stressful events example of other specified dissociative disorder is characterized by a combination of dissociative symptoms that occur together acutely in response to stressful events and typically last less than 1 month. Amnestic episodes that occur as part of these reactions are accompanied by other prominent dissociative symptoms, have a short duration (hours or days), and tend to be circumscribed to limited periods or events in a person's life (micro-amnesias).

Factitious disorder and malingering. There is no test, battery of tests, or set of procedures that invariably distinguishes dissociative amnesia from feigned amnesia. Feigned amnesia is more common in individuals with 1) acute, florid dissociative amnesia; 2) financial, sexual, or legal problems; 3) a wish to escape stressful circumstances; 4) a desire to seem to be a more interesting patient; and/or 5) a plan to engage in litigation for "recovered memories." However, dissociative amnesia can be associated with those same circumstances and can coexist with deliberate feigning. Many individuals who malinger amnesia confess spontaneously or when confronted.

Memory changes with aging or mild neurocognitive disorder. Memory decrements in mild neurocognitive disorder differ from those of dissociative amnesia; in mild neurocognitive disorder, memory changes manifest as difficulty in learning and retaining new information. This is often measured in tests of verbal learning of word lists or a brief story with evaluation of immediate and delayed recall. With normal cognitive aging, individuals may also have similar weaknesses in immediate and delayed recall of new information,

although normal aging may also affect information processing speed and other complex executive function tasks in addition to memory.

Comorbidity

As is common in individuals with a history of trauma, many comorbidities co-occur with dissociative amnesia, particularly as dissociative amnesia begins to remit. A wide variety of affective phenomena may surface, including dysphoria, grief, rage, shame, guilt, and psychological conflict and turmoil. Individuals may engage in nonsuicidal self-injury and other high-risk behaviors. These individuals may have symptoms that meet diagnostic criteria for persistent depressive disorder, major depressive disorder, or subthreshold depression (other specified depressive disorder). Many individuals with dissociative amnesia develop PTSD at some point during their life, especially when the traumatic antecedents of their amnesia are brought into conscious awareness. Many of these individuals may show symptoms of the dissociative subtype of PTSD. Many individuals with dissociative amnesia have symptoms that meet diagnostic criteria for a comorbid somatic symptom and related disorder (and vice versa), particularly functional neurological symptom disorder (conversion disorder). Substance-related and addictive disorders may be comorbid with dissociative amnesia, as well as feeding and eating disorders and sexual dysfunctions. The most common comorbid personality disorder is other specified personality disorder (with mixed personality disorder features), which often includes avoidant, obsessive-compulsive, dependent, and borderline features.

Depersonalization/Derealization Disorder

Diagnostic Criteria F48.1

A. The presence of persistent or recurrent experiences of depersonalization, derealization, or both:

   1. **Depersonalization:** Experiences of unreality, detachment, or being an outside observer with respect to one's thoughts, feelings, sensations, body, or actions (e.g.,

perceptual alterations, distorted sense of time, unreal or absent self, emotional and/or physical numbing).

2. **Derealization:** Experiences of unreality or detachment with respect to surroundings (e.g., individuals or objects are experienced as unreal, dreamlike, foggy, lifeless, or visually distorted).

B. During the depersonalization or derealization experiences, reality testing remains intact.

C. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, medication) or another medical condition (e.g., seizures).

E. The disturbance is not better explained by another mental disorder, such as schizophrenia, panic disorder, major depressive disorder, acute stress disorder, posttraumatic stress disorder, or another dissociative disorder.

Diagnostic Features

The essential features of depersonalization/derealization disorder are persistent or recurrent episodes of depersonalization, derealization, or both. Episodes of depersonalization are characterized by a feeling of unreality or detachment from, or unfamiliarity with, the individual's whole self or from aspects of the self (Criterion A1). The individual may feel detached from his or her entire being (e.g., "I am no one," "I have no self"). He or she may

also feel subjectively detached from aspects of the self, including feelings (e.g., hypoemotionality: "I know I have feelings, but I don't feel them"), thoughts (e.g., "My thoughts don't feel like my own," "head filled with cotton"), whole body or body parts, or sensations (e.g., touch, proprioception, hunger, thirst, libido). There may also be a diminished sense

of agency (e.g., feeling robotic, like an automaton; lacking control of speech or movements). The depersonalization experience can sometimes be one of a split self, with one part observing and one participating, known as an "out-of-body experience" in its most extreme form. The unitary symptom of "depersonalization" consists of several symptom factors: anomalous body experiences (i.e., unreality of the self and perceptual alterations); emotional or physical numbing; and temporal distortions with anomalous subjective recall.

Episodes of derealization are characterized by a feeling of unreality or detachment from, or unfamiliarity with, the world, be it individuals, inanimate objects, or all surroundings (Criterion A2). The individual may feel as if he or she were in a fog, dream, or bubble, or as if there were a veil or a glass wall between the individual and the world around. Surroundings may be experienced as artificial, colorless, or lifeless. Derealization is commonly accompanied by subjective visual distortions, such as blurriness, heightened acuity, widened or narrowed visual field, two-dimensionality or flatness, exaggerated three-dimensionality, or altered distance or size of objects (i.e., macropsia or micropsia). Auditory distortions can also occur, whereby voices or sounds are muted or heightened. In addition, Criterion C requires that the symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning, and Criteria D and E describe exclusionary diagnoses.

Associated Features

Individuals with depersonalization/derealization disorder may have difficulty describing their symptoms and may think they are "crazy" or "going crazy." Another common experience is the fear of irreversible brain damage. A commonly associated symptom is a subjectively altered sense of time (i.e., too fast or too slow), as well as a subjective difficulty in vividly recalling past memories and owning them as personal and emotional. Vague somatic symptoms, such as head fullness, tingling, or lightheadedness, are not uncommon. Individuals may experience extreme rumination or obsessional preoccupation (e.g., constantly obsessing about whether they really exist, or checking their perceptions to determine whether they appear real). Varying degrees of anxiety and depression are also common

associated features. Individuals with the disorder have been found to have physiological hyporeactivity to emotional stimuli. Neural substrates of interest include the hypothalamic-pituitary-adrenocortical axis, inferior parietal lobule, and prefrontal cortical-limbic circuits.

Prevalence

Transient depersonalization/derealization symptoms lasting hours to days are common in the general population. The 12-month prevalence of depersonalization/derealization disorder is thought to be markedly less than for transient symptoms, although precise estimates for the disorder are unavailable. In general, approximately one-half of all adults have experienced at least one lifetime episode of depersonalization/derealization. However, symptomatology that meets full criteria for depersonalization/derealization disorder is markedly less common than transient symptoms. One-month prevalence in the United Kingdom is approximately 1%–2%.

Development and Course

The mean age at onset of depersonalization/derealization disorder is 16 years, although the disorder can start in early or middle childhood; a minority cannot recall ever not having had the symptoms. Less than 20% of individuals experience onset after age 20 years and only 5% after age 25 years. Onset in the fourth decade of life or later is highly unusual.

Onset can range from extremely sudden to gradual. Duration of depersonalization/derealization disorder episodes can vary greatly, from brief (hours or days) to prolonged (weeks, months, or years). Given the rarity of disorder onset after age 40 years, in such cases the individual should be examined more closely for underlying medical conditions (e.g., brain lesions, seizure disorders, sleep apnea). The course of the disorder is often persistent. About one-third of cases involve discrete episodes; another third, continuous symptoms from the start; and still another third, an initially episodic course that eventually becomes continuous.

While in some individuals the intensity of symptoms can wax and wane considerably, others report an unwavering level of intensity that in extreme cases can be constantly present for years or decades. Internal and external factors that affect symptom intensity vary between individuals, yet some typical patterns are reported. Exacerbations can be triggered by stress, worsening mood or anxiety symptoms, novel or overstimulating settings, and physical factors such as lighting or lack of sleep.

Risk and Prognostic Factors

Temperamental. Individuals with depersonalization/derealization disorder are characterized by harm-avoidant temperament, immature defenses, and both disconnection and overconnection schemata. Immature defenses such as idealization/devaluation, projection, and acting out result in denial of reality and poor adaptation. *Cognitive disconnection schemata* reflect defectiveness and emotional inhibition and subsume themes of abuse, neglect, and deprivation. *Overconnection schemata* involve impaired autonomy with themes of dependency, vulnerability, and incompetence.

Environmental. There is a clear association between the disorder and childhood interpersonal traumas in a substantial portion of individuals, although this association is not as prevalent or as extreme in the nature of the traumas as in other dissociative disorders, such as dissociative identity disorder. In particular, emotional abuse and emotional neglect have been most strongly and consistently associated with the disorder. Other stressors can include physical abuse; witnessing domestic violence; growing up with a seriously impaired, mentally ill parent; or unexpected death or suicide of a family member or close friend. Sexual abuse is a much less common antecedent but can be encountered. The most common proximal precipitants of the disorder are severe stress (interpersonal, financial, occupational), depression, anxiety (particularly panic attacks), and illicit drug use. Symptoms may be specifically induced by substances such as tetrahydrocannabinol, hallucinogens, ketamine, MDMA (3,4-methylenedioxymethamphetamine; “ecstasy”), and salvia. Marijuana use may precipitate new-onset panic attacks and depersonalization/derealization symptoms simultaneously.

Culture-Related Diagnostic Issues

Volitionally induced experiences of depersonalization/derealization can be a part of meditative practices that are prevalent in many religious, spiritual, and cultural contexts and should not be diagnosed as a disorder. However, there are individuals who initially induce these states intentionally but over time lose control over them and may develop a fear and aversion for related practices. Cultural frameworks may affect the level of distress or perceived severity associated with uncontrolled depersonalization/derealization experiences by providing explanations for them (e.g., spiritual/supernatural causes), which may alleviate individuals' fears that they are "losing their mind."

Functional Consequences of Depersonalization/Derealization Disorder

Symptoms of depersonalization/derealization disorder are highly distressing and are associated with major morbidity. The affectively flattened and robotic demeanor that these

individuals often demonstrate may appear incongruent with the extreme emotional pain reported by those with the disorder. Impairment is often experienced in both interpersonal and occupational spheres, largely as a result of the hypoemotionality with others, subjective difficulty in focusing and retaining information, and a general sense of disconnectedness from life.

Differential Diagnosis

Illness anxiety disorder. Although individuals with depersonalization/derealization disorder can present with vague somatic complaints as well as fears of permanent brain damage, the diagnosis of depersonalization/derealization disorder is characterized by the presence of a constellation of typical depersonalization/derealization symptoms and the absence of other manifestations of illness anxiety disorder.

Major depressive disorder.

Feelings of numbness, deadness, apathy, and being in a dream are not uncommon in major depressive episodes. However, in depersonalization/derealization disorder, such symptoms are associated with further symptoms of the disorder. If the depersonalization/derealization clearly precedes the onset of a major depressive episode or clearly continues after its resolution, the diagnosis of depersonalization/derealization disorder applies.

Obsessive-compulsive disorder. Some individuals with depersonalization/derealization disorder can become obsessively preoccupied with their subjective experience or develop rituals checking on the status of their symptoms. However, other symptoms of obsessive-compulsive disorder unrelated to depersonalization/derealization are not present.

Other dissociative disorders. In order to diagnose depersonalization/derealization disorder, the symptoms should not occur in the context of another dissociative disorder, such as dissociative identity disorder. Differentiation from dissociative amnesia and functional neurological symptom disorder (conversion disorder) is simpler, as the symptoms of these disorders do not overlap with those of depersonalization/derealization disorder.

Panic attacks. Depersonalization/derealization is one of the symptoms of panic attacks, increasingly common as panic attack severity increases. Therefore, depersonalization/derealization disorder should not be diagnosed when the symptoms occur only during panic attacks that are part of panic disorder, social anxiety disorder, or specific phobia. In addition, it is not uncommon for depersonalization/derealization symptoms to first begin in the context of new-onset panic attacks or as panic disorder progresses and worsens. In such presentations, the diagnosis of depersonalization/derealization disorder can be made if 1) the depersonalization/derealization component of the presentation is very prominent from the start, clearly exceeding in duration and intensity the occurrence of actual panic attacks; or 2) the depersonalization/derealization continues after panic disorder has remitted or has been successfully treated.

Psychotic disorders. The presence of intact reality testing specifically regarding the depersonalization/derealization symptoms is essential to

differentiating depersonalization/derealization disorder from psychotic disorders. Rarely, positive-symptom schizophrenia can pose a diagnostic challenge when nihilistic delusions are present. For example, an individual may complain that he or she is dead or the world is not real; this could be either a subjective experience that the individual knows is not true or a delusional conviction.

Substance/medication-induced disorders. Depersonalization/derealization associated with the physiological effects of substances during acute intoxication or withdrawal is not diagnosed as depersonalization/derealization disorder. The most common precipitating substances are the illicit drugs marijuana, hallucinogens, ketamine, ecstasy, and salvia. In

about 15% of all cases of depersonalization/derealization disorder, the symptoms are precipitated by ingestion of such substances. If the symptoms persist for some time in the absence of any further substance or medication use, the diagnosis of depersonalization/derealization disorder applies. This diagnosis is usually easy to establish since the vast majority of individuals with this presentation become highly phobic and aversive to the triggering substance and do not use it again.

Traumatic brain injury. Depersonalization/derealization symptoms are typical in traumatic brain injury (TBI) but are distinguished from depersonalization/derealization disorder by onset of symptoms following TBI and the lack of other symptoms of depersonalization/derealization disorder.

Dissociative symptoms due to another medical condition. Features such as onset after age 40 years or the presence of atypical symptoms and course in any individual suggest the possibility of an underlying medical condition. In cases with dissociative symptoms, it is essential to conduct a thorough medical and neurological evaluation, which may include standard laboratory studies, viral titers, an electroencephalogram, vestibular testing, visual testing, sleep studies, and/or brain imaging. When the suspicion of an underlying seizure disorder proves difficult to confirm, an ambulatory

electroencephalogram may be indicated; although temporal lobe epilepsy is most commonly implicated, parietal and frontal lobe epilepsy may also be associated.

Comorbidity

In a convenience sample of adults recruited for a number of depersonalization research studies, lifetime comorbidities were high for unipolar depressive disorder and for any anxiety disorder, with a significant proportion of the sample having both disorders. Comorbidity with posttraumatic stress disorder was low. The three most commonly co-occurring personality disorders were avoidant, borderline, and obsessive-compulsive.

Other Specified Dissociative Disorder

F44.89

This category applies to presentations in which symptoms characteristic of a dissociative disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the dissociative disorders diagnostic class. The other specified dissociative disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific dissociative disorder. This is done by recording “other specified dissociative disorder” followed by the specific reason (e.g., “dissociative trance”).

Examples of presentations that can be specified using the “other specified” designation include the following:

1. **Chronic and recurrent syndromes of mixed dissociative symptoms:** This category includes identity disturbance associated with less-than-marked discontinuities in sense of self and agency, or alterations of identity or episodes of

possession in an individual who reports no dissociative amnesia.

2. **Identity disturbance due to prolonged and intense coercive persuasion:** Individuals who have been subjected to intense coercive persuasion (e.g., brainwashing, thought reform, indoctrination while captive, torture, long-term political imprisonment, recruitment by sects/cults or by terror organizations) may present with prolonged changes in, or conscious questioning of, their identity.

3. **Acute dissociative reactions to stressful events:** This category is for acute, transient conditions that typically last less than 1 month, and sometimes only a few hours or days. These conditions are characterized by constriction of consciousness; depersonalization; derealization; perceptual disturbances (e.g., time slowing, macropsia); microamnesias; transient stupor; and/or alterations in sensory-motor functioning (e.g., analgesia, paralysis).
4. **Dissociative trance:** This condition is characterized by an acute narrowing or complete loss of awareness of immediate surroundings that manifests as profound unresponsiveness or insensitivity to environmental stimuli. The unresponsiveness may be accompanied by minor stereotyped behaviors (e.g., finger movements) of which the individual is unaware and/or that he or she cannot control, as well as transient paralysis or loss of consciousness. The dissociative trance is not a normal part of a broadly accepted collective cultural or religious practice.

Unspecified Dissociative Disorder

F44.9

This category applies to presentations in which symptoms characteristic of a dissociative disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the dissociative disorders diagnostic class. The unspecified dissociative disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific dissociative disorder and includes presentations for which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Somatic Symptom and Related Disorders

This chapter includes the diagnoses of somatic symptom disorder, illness anxiety disorder, functional neurological symptom disorder (conversion disorder), psychological factors affecting other medical conditions, factitious disorder, other specified somatic symptom and related disorder, and unspecified somatic symptom and related disorder. All of the disorders in this chapter share a common feature: the prominence of somatic symptoms and/or illness anxiety associated with significant distress and impairment. Individuals with disorders with prominent somatic symptoms or illness anxiety are commonly encountered in primary care and other medical settings but are less commonly encountered in psychiatric and other mental health settings. These reconceptualized diagnoses, based on a reorganization of DSM-IV somatoform disorder diagnoses, are more useful for primary care and other medical (nonpsychiatric) clinicians.

The major diagnosis in this diagnostic class, somatic symptom disorder, emphasizes diagnosis made on the basis of the presence of symptoms and signs (distressing somatic symptoms plus abnormal thoughts, feelings, and behaviors in response to these symptoms) rather than the absence of a medical explanation for somatic symptoms. A distinctive characteristic of many individuals with somatic symptom disorder is not the somatic symptoms per se, but instead the way they present and interpret them. Incorporating affective, cognitive, and behavioral components into the criteria for somatic symptom disorder provides a more comprehensive and accurate reflection of the true clinical picture than can be achieved by assessing the somatic complaints alone.

The principles behind the changes in the somatic symptom and related diagnoses from DSM-IV are crucial in understanding the DSM-5 diagnoses.

The DSM-IV term *somatoform disorders* was confusing and was replaced by *somatic symptom and related disorders*. In DSM-IV there was a great deal of overlap across the somatoform disorders and a lack of clarity about the boundaries of diagnoses. Although individuals with these disorders primarily present in medical rather than mental health settings, nonpsychiatric physicians found the DSM-IV somatoform diagnoses difficult to understand and use. The current DSM-5 classification recognizes this overlap by reducing the total number of disorders as well as their subcategories.

The previous criteria overemphasized the centrality of symptoms being unexplained by recognized pathophysiological processes. Such symptoms are present to various degrees, particularly in functional neurological symptom disorder, but somatic symptom disorders can also accompany recognized medical conditions (i.e., those disorders related to clearly recognized pathophysiological processes). The reliability of determining that a somatic symptom is unexplained by a recognized pathophysiological process related to a recognized medical condition is limited, and grounding a diagnosis on the absence of an explanation is problematic and reinforces mind-body dualism. It is not appropriate to give an individual a mental disorder diagnosis solely because a recognized medical condition cannot be demonstrated. Furthermore, the presence of a recognized medical condition does not exclude the possibility of a comorbid mental disorder, including a somatic symptom and related disorder. Perhaps because of the predominant focus on lack of medical explanation in DSM-IV, individuals regarded these diagnoses as pejorative and demeaning,

implying that their physical symptoms were not “real.” The DSM-5 classification defines the major diagnosis, somatic symptom disorder, on the basis of positive symptoms (distressing somatic symptoms plus abnormal thoughts, feelings, and behaviors in response to these symptoms). In functional neurological symptom disorder and pseudocyesis (other specified somatic symptom and related disorder), the emphasis is on demonstrating clinical evidence of incompatibility with recognized pathophysiological processes.

It is important to note that some other mental disorders may initially manifest with primarily somatic symptoms (e.g., major depressive disorder, panic disorder). Such diagnoses may account for the somatic symptoms, or they may occur alongside one of the somatic symptom and related disorders in this chapter. There is also considerable medical comorbidity among individuals with somatic symptom and related disorders. Although somatic symptoms are frequently associated with psychological distress and psychopathology, some somatic symptom and related disorders can arise spontaneously, and their causes can remain obscure. Anxiety disorders and depressive disorders may accompany somatic symptom and related disorders. The somatic component adds severity and complexity to depressive and anxiety disorders and results in higher severity, functional impairment, and even refractoriness to traditional treatments. In rare instances, the degree of preoccupation may be so severe as to warrant consideration of a delusional disorder diagnosis.

A number of factors may contribute to somatic symptom and related disorders. These include genetic and biological vulnerability (e.g., increased sensitivity to pain), early traumatic experiences (e.g., violence, abuse, deprivation), medical iatrogenesis (e.g., reinforcement of the sick role, excessive referrals and diagnostic testing), and learning (e.g., lack of reinforcement of nonsomatic expressions of distress), as well as sociocultural norms that minimize or stigmatize psychological suffering as compared with physical suffering. Differences in medical care across cultural contexts affect the presentation, recognition, and management of these somatic presentations. Variations in symptom presentation are likely the result of the interaction of multiple factors within cultural contexts that affect how individuals identify and classify bodily sensations, perceive illness, and seek medical attention for them.

All of these disorders are characterized by the prominent focus on somatic concerns and their initial presentation mainly in medical rather than mental health care settings. Somatic symptom disorder and illness anxiety disorder offer more clinically useful methods of characterizing individuals who may have been considered in the past for a diagnosis of somatization disorder and hypochondriasis. Furthermore, approximately two-thirds to three-fourths of individuals previously diagnosed with hypochondriasis are subsumed under the diagnosis of somatic symptom disorder. However, the

remaining one-quarter to one-third of individuals with previously diagnosed hypochondriasis have high health anxiety in the absence of somatic symptoms, and many such individuals' symptoms would not qualify for an anxiety disorder diagnosis. The DSM-5 diagnosis of illness anxiety disorder is for this latter group of individuals. Illness anxiety disorder can be considered either a somatic symptom and related disorder or an anxiety disorder. Because of the strong focus on somatic concerns, and because illness anxiety disorder is most often encountered in medical settings, for utility it is listed with the somatic symptom and related disorders. In functional neurological symptom disorder, the key to diagnosis is neurological symptoms that can be demonstrated, on the basis of positive clinical examination features, to be incompatible with recognized pathophysiology. This is now a "rule-in" diagnosis, and not a diagnosis of exclusion, and can be made in the presence of a recognized neurological disorder. It no longer requires the presence of a recent psychological stressor, because such stressors are not always present. Psychological factors affecting other medical conditions is also included in this chapter. Its essential feature is the presence of one or more clinically significant psychological or behavioral factors that adversely affect a medical condition by increasing the risk for suffering, death, or disability. Like the other somatic symptom and related disorders, factitious disorder embodies persistent problems related to illness perception and identity. In the great majority of reported cases of factitious disorder, both

imposed on self and imposed on another, individuals present with somatic symptoms and expressed medical disease conviction. Consequently, DSM-5 factitious disorder is included among the somatic symptom and related disorders. Other specified somatic symptom and related disorder and unspecified somatic symptom and related disorder include conditions for which some, but not all, of the criteria for somatic symptom disorder or illness anxiety disorder are met, as well as pseudocyesis.

Somatic Symptom Disorder

Diagnostic Criteria F45.1

A. One or more somatic symptoms that are distressing or result in significant disruption of daily life.

B. Excessive thoughts, feelings, or behaviors related to the somatic symptoms or associated health concerns as manifested by at least one of the following:

   1. Disproportionate and persistent thoughts about the seriousness of one's symptoms.
   2. Persistently high level of anxiety about health or symptoms.
   3. Excessive time and energy devoted to these symptoms or health concerns.

C. Although any one somatic symptom may not be continuously present, the state of being symptomatic is persistent (typically more than 6 months).

Specify if:

With predominant pain (previously pain disorder): This specifier is for individuals whose somatic symptoms predominantly involve pain.

Specify if:

Persistent: A persistent course is characterized by severe symptoms, marked impairment, and long duration (more than 6 months).

Specify current severity:

Mild: Only one of the symptoms specified in Criterion B is fulfilled.

Moderate: Two or more of the symptoms specified in Criterion B are fulfilled.

Severe: Two or more of the symptoms specified in Criterion B are fulfilled, plus there are multiple somatic complaints (or one very severe somatic symptom).

Diagnostic Features

Individuals with somatic symptom disorder typically have multiple, current, somatic symptoms that are distressing or result in significant disruption of daily life (Criterion A), although sometimes only one severe symptom, most commonly pain, is present. Symptoms may be specific (e.g., localized pain) or relatively nonspecific (e.g., fatigue). The symptoms sometimes represent normal bodily sensations or discomfort that does not generally signify serious disease. Somatic symptoms without an evident medical explanation are not sufficient to make this diagnosis. The individual's suffering is authentic, whether or not it is medically explained.

The symptoms may or may not be associated with another medical condition. The diagnoses of somatic symptom disorder and a concurrent medical illness are not mutually exclusive, and these frequently occur together. For example, an individual may become seriously disabled by symptoms of somatic symptom disorder after an uncomplicated myocardial infarction even if the myocardial infarction itself did not result in any disability. If another medical condition or high risk for developing one is present (e.g., strong family history), the thoughts, feelings, and behaviors associated with this condition are excessive (Criterion B).

Individuals with somatic symptom disorder tend to have very high levels of worry about illness (Criterion B). They appraise their bodily symptoms as unduly threatening, harmful, or troublesome and often think the worst about their health. Even when there is evidence to the contrary, some individuals still fear the medical seriousness of their symptoms. In severe somatic symptom disorder, health concerns may assume a central role in the individual's life, becoming a feature of his or her identity and dominating interpersonal relationships.

Individuals typically experience distress that is principally focused on somatic symptoms and their significance. When asked directly about their distress, some individuals describe it in relation to other aspects of their lives, while others deny any source of distress other than the somatic

symptoms. Health-related quality of life is often impaired, both physically and mentally. The diagnosis can further be specified by stating whether complaints predominantly involve pain and/or if complaints are marked by a persistent course.

Additionally, severity of somatic symptom disorder can be specified by the number of fulfilled B criteria. Mild forms of somatic symptom disorder (one symptom as specified in Criterion B is fulfilled) are more prevalent, while moderate (two or more B criteria are present) and severe cases (two or more symptoms as specified in Criterion B are fulfilled in combination with multiple somatic complaints or one very severe somatic symptom) are marked by higher levels of impairment. In severe somatic symptom disorder, the impairment is marked, and when persistent, the disorder can lead to invalidism.

There is often a high level of medical care utilization, which rarely alleviates the individual's concerns. Consequently, the individual may seek care from multiple doctors for the same symptoms. These individuals often seem unresponsive to medical interventions, and new interventions may only exacerbate the presenting symptoms. Some individuals with the disorder seem unusually sensitive to medication side effects. Some feel that their medical assessment and treatment have been inadequate.

The criteria for somatic symptom disorder appear suitable for use in children and adolescents, but they have been less studied in youth than among adults.

Associated Features

Cognitive features include attention focused on somatic symptoms, attribution of normal bodily sensations to physical illness (possibly with catastrophic interpretations), worry about illness, a self-concept of bodily weakness, and intolerance of bodily complaints. Besides health anxiety, emotional features may include negative affectivity, desperation, and demoralization related to somatic symptoms. The relevant associated behavioral features may include repeated bodily checking for abnormalities, repeated seeking of medical help and reassurance, and avoidance of physical activity. These behavioral features are most pronounced in severe, persistent somatic symptom disorder. These features are usually associated

with frequent requests for medical help for different somatic symptoms. This may lead to medical consultations in which individuals are so focused on their concerns about somatic symptom(s) that they cannot be redirected to other matters. Any reassurance by the doctor that the symptoms are not indicative of serious physical illness tends to be short-lived and/or is experienced by the individuals as the doctor not taking their symptoms with due seriousness. As the focus on somatic symptoms is a primary feature of the disorder, individuals with somatic symptom disorder typically present to general medical health services rather than mental health services. The suggestion of referral to a mental health specialist may be met with surprise or even frank refusal by individuals with somatic symptom disorder.

Prevalence

The prevalence of somatic symptom disorder is unclear. Estimates about the prevalence of somatic symptom disorder come from the limited epidemiological literature on

DSM-IV-TR somatoform disorders. However, the prevalence of somatic symptom disorder is expected to be higher than that of the more restrictive DSM-IV-TR somatization disorder (<1%) but lower than that of undifferentiated somatoform disorder (approximately 19%). More recent population-based studies with a questionnaire-based strategy using DSM-5 diagnostic criteria for somatic symptom disorder in adult and adolescent samples report prevalence rates between 6.7% and 17.4%. Based on research conducted in Europe and North America, the prevalence of somatic symptom disorder in the general adult population can be approximated as 4%–6%.

Somatic symptom disorder has a higher frequency in primary care patients than in the general population. Based on reviews and meta-analyses of studies from multiple countries that had still used DSM-IV or ICD-10 criteria, a 12-month prevalence of somatic symptom disorder and related conditions in primary care patients between 10% and 20% appears plausible. Prevalence rates are higher in clinical settings that specialize in

psychosomatic or functional disorders, with reported frequencies of somatic symptom disorder between 40% and 60%.

Women tend to report more somatic symptoms than do men, and the prevalence of somatic symptom disorder is consequently likely to be higher in women.

Development and Course

In a study of Danish children ages 5–7 years, functional somatic symptoms were common health complaints, which for a significant minority (roughly one-fifth) of those with complaints were severe enough to cause distress, impairment, school absences, or medical help-seeking. Age at onset does not seem to affect the duration of untreated illness.

The course of somatic symptom disorder is likely to be chronic and fluctuating and influenced by the number of symptoms, individual's age, level of impairment, and any comorbidity. The course is also influenced by personality traits, with less harm avoidance and greater cooperativeness associated with a shorter time to remission.

In children, the most common symptoms are recurrent abdominal pain, headache, fatigue, and nausea. A single prominent symptom is more common in children than in adults. When the diagnosis is being made in younger individuals, it is important to obtain patient, family, and other assessments (e.g., school) of symptom presentation. Patient and caregiver engagement during evaluation and management is fundamental because parents' interpretation of and response to symptoms may determine the level of associated distress, the demands for medical investigations and interventions, and time away from school.

In older individuals, pain localized in several body regions appears to be the most common symptom. Somatic symptoms and concurrent medical illnesses are common as multimorbidity increases with age. Prevalence rates of somatic symptom disorder seem to be stable until age 65 years and might decrease thereafter. For making the diagnosis in older individuals, a focus on the requirement for excessive thoughts, feelings, or behaviors related to the somatic symptoms or associated health concerns (Criterion B) is crucial. Somatic symptom disorder may be underdiagnosed in older adults either because certain somatic symptoms (e.g., pain, fatigue) are

considered part of normal aging or because illness worry is considered "understandable" in older adults who have more general medical illnesses and medications than do younger people.

Risk and Prognostic Factors

Temperamental. The personality trait of negative affectivity (neuroticism) has been identified as an independent correlate/risk factor of a high number of somatic symptoms. Comorbid anxiety or depression is common and may exacerbate symptoms and impairment.

Environmental. Somatic symptom disorder is more frequent in individuals with few years of education and low socioeconomic status, and in those who have recently experienced stressful or health-related life events. Early lifetime adversity such as childhood sexual abuse is also likely a risk factor for somatic symptom disorder in adults.

Course modifiers. Persistent somatic symptoms are associated with demographic features (women, older age, fewer years of education, lower socioeconomic status, unemployment), a reported history of sexual abuse or other childhood adversity, concurrent chronic physical illness or mental disorder (depression, anxiety, persistent depressive disorder, panic), social stress, and reinforcing social factors such as illness benefits. Total somatic symptom severity is probably associated with female gender, anxiety, depression and general medical illness. Cognitive factors that affect clinical course include sensitization to pain, heightened attention to bodily sensations, and attribution of bodily symptoms to a possible medical illness rather than recognizing them as a normal phenomenon or psychological stress.

Culture-Related Diagnostic Issues

High numbers of somatic symptoms are found in population-based and primary care studies around the world, with a similar pattern of the most commonly reported somatic symptoms, impairment, and treatment seeking. The relationship between number of somatic symptoms and illness worry is

similar in different cultural contexts, and marked illness worry is associated with impairment and greater treatment seeking cross-culturally. In many cultural contexts, individuals with depression commonly present with somatic symptoms.

Despite these similarities, there are differences in somatic symptoms across cultural contexts and ethnoracial groups. Sociocultural factors, particularly stigma related to mental disorders, may explain differences in somatic symptom reporting across cultural contexts. The description of somatic symptoms varies with linguistic and other local cultural factors. These somatic presentations have been described as "idioms of distress" because somatic symptoms may have special meanings and shape patient-clinician interactions in the particular cultural contexts. For example, sensations of heaviness, complaints of "gas," too much heat in the body, or burning in the head are common in some cultures or ethnic groups but rare in others. Cultural explanations also vary, and somatic symptoms may be attributed variously to particular family, work (e.g., burnout), or other environmental stresses; general medical illness; the suppression of feelings of anger and resentment; or certain culturally specific attributions, such as semen loss. Certain somatic symptoms may be part of specific explanatory models in a given cultural context; for example, traditional understandings of *shenjing shuairuo* in China link concepts of "weakness of nerves" (neurasthenia) and hot-cold imbalance with prominent symptoms such as fatigue and low energy. There may also be differences in medical treatment seeking and utilization of nonmedical, traditional, alternative and complementary healing practices among cultural groups, in addition to differences due to variable access to medical care services. Cultural beliefs, previous illnesses, insurance status, health literacy, and health care experiences can influence individuals' perception of somatic symptoms and health care use. Seeking treatment for multiple somatic symptoms in general medical clinics is a worldwide phenomenon.

Sex- and Gender-Related Diagnostic Issues

In population-based studies, women report more somatic symptoms than men, and in one study of primary care patients with chronic pain, women reported more severe somatic symptoms than men. While exposure to

sexual trauma, intimate partner violence, and a childhood trauma history is associated with increased somatic symptom expression in

both women and men, a childhood history of multiple adverse childhood experiences is especially likely to increase somatic symptom expression in women.

In women, gender is associated with an increased likelihood of developing persistent symptoms of somatic symptom disorder. There appears to be no evidence that gender is associated with the duration of untreated illness and response to psychological or pharmacological treatment.

Association With Suicidal Thoughts or Behavior

Somatic symptom disorder is associated with suicidal thoughts and suicide attempts. It is likely that suicidal thoughts and behaviors are partly explained by the diagnostic overlap and frequent comorbidity of somatic symptom disorder and depressive disorders. In addition, dysfunctional illness perceptions and the severity of somatic symptoms appear to be independently associated with an increased risk of suicidal ideation.

Functional Consequences of Somatic Symptom Disorder

The disorder is associated with marked impairment of health status and high psychological distress. Many individuals with severe somatic symptom disorder are likely to have impaired health status scores more than 2 standard deviations below population norms. Health status is particularly impaired in the presence of multiple or severe symptoms.

Differential Diagnosis

If the somatic symptoms are consistent with another mental disorder (e.g., panic disorder), and the diagnostic criteria for that disorder are fulfilled, then that mental disorder should be considered as an alternative or additional diagnosis. If, as commonly occurs, the criteria for both somatic

symptom disorder and another mental disorder diagnosis are fulfilled, then both should be diagnosed, as both may require treatment.

Other medical conditions. The presence of somatic symptoms of unclear etiology is not in itself sufficient to make the diagnosis of somatic symptom disorder. The symptoms of many individuals with disorders like irritable bowel syndrome or fibromyalgia would not satisfy the criterion necessary to diagnose somatic symptom disorder (Criterion B). Conversely, the presence of somatic symptoms of an established medical condition (e.g., diabetes or heart disease) does not exclude the diagnosis of somatic symptom disorder if the criteria are otherwise met. Factors that distinguish individuals with somatic symptom disorder from individuals with general medical conditions alone include the ineffectiveness of analgesics, a history of mental disorders, unclear provocative or palliative factors, persistence without cessation, and stress.

Psychological factors affecting other medical conditions. The diagnosis of somatic symptom disorder requires distressing or impairing somatic symptoms that may or may not be associated with another medical condition but must be accompanied by excessive or disproportionate thoughts, feelings, or behaviors related to the somatic symptoms or associated health concerns. In contrast, the diagnosis of psychological factors affecting other medical conditions requires the presence of a medical condition, as well as psychological factors that adversely affect its course or interfere with its treatment.

Panic disorder. In panic disorder, somatic symptoms and anxiety about health tend to occur in acute episodes, whereas in somatic symptom disorder, anxiety and somatic symptoms are more persistent.

Generalized anxiety disorder. Individuals with generalized anxiety disorder worry about multiple events, situations, or activities, only one of which may involve their health. The main focus is not usually somatic symptoms or fear of illness as it is in somatic symptom disorder.

Depressive disorders. Depressive disorders are commonly accompanied by somatic symptoms such as fatigue, headaches, or joint, abdominal, or other

pains. However, depressive disorders are differentiated from somatic symptom disorder by the requirement of the presence of depressed mood or, in the case of major depressive disorder, either depressed mood or decreased interest or pleasure in activities. In some cultural contexts, these core symptoms of depression may be initially denied or deemphasized by individuals whose presentations would otherwise meet criteria for a depressive disorder. Such individuals might instead emphasize somatic symptoms that may be idiomatic (e.g., heavy heart) and unfamiliar to clinicians.

Illness anxiety disorder. If the individual has extensive worries about health but no or minimal somatic symptoms, it may be more appropriate to consider illness anxiety disorder.

Functional neurological symptom disorder (conversion disorder). In functional neurological symptom disorder, the presenting symptom is loss of function (e.g., of a limb), whereas in somatic symptom disorder, the focus is on the distress that particular symptoms cause. The features listed under Criterion B of somatic symptom disorder may be helpful in differentiating the two disorders.

Delusional disorder. In somatic symptom disorder, the individual's beliefs that somatic symptoms might reflect serious underlying physical illness are not held with delusional intensity. Nonetheless, the individual's beliefs concerning the somatic symptoms can be firmly held. In contrast, in delusional disorder, somatic type, the individual's conviction that the somatic symptoms are indicative of having a serious underlying illness is stronger than that found in somatic symptom disorder.

Body dysmorphic disorder. In body dysmorphic disorder, the individual is excessively concerned about, and preoccupied by, a perceived defect in his or her physical appearance. In contrast, in somatic symptom disorder, the concern about somatic symptoms reflects fear of underlying illness, not of a defect in appearance.

Obsessive-compulsive disorder. In somatic symptom disorder, the recurrent ideas about somatic symptoms or illness are less intrusive, and individuals with this disorder do not exhibit the associated repetitive behaviors aimed at reducing anxiety that occur in obsessive-compulsive disorder.

Factitious disorder and malingering. In factitious disorder and malingering, individuals present themselves as ill or impaired but have falsified presenting physical signs and symptoms with the intent to deceive. In contrast, the symptoms of somatic symptom disorder are not simulated or self-induced, and these individuals suffer authentically and seriously from their somatic complaints.

Comorbidity

Somatic symptom disorder is associated with high rates of comorbidity with other mental disorders as well as general medical conditions. The most relevant co-occurring mental disorders are anxiety and depressive disorders, each of which occurs in up to 50% of cases of somatic symptom disorders and significantly contributes to overall functional impairment and poorer quality of life. Other mental disorders that have been found to co-occur with somatic symptom disorder are posttraumatic stress disorder and obsessive-compulsive disorder. Other evidence indicates an association with sexual dysfunction in men.

Elevated levels of the psychological features (Criterion B) of somatic symptom disorder have been found in several general medical conditions. When a concurrent general medical condition is present, the degree of impairment is more marked than would be expected from the physical illness alone. Moreover, somatization in medical illness has been shown to worsen disease and treatment outcomes, adherence, and quality of life and to increase health care utilization.

Illness Anxiety Disorder

Diagnostic Criteria	F45.21

A. Preoccupation with having or acquiring a serious illness.

B. Somatic symptoms are not present or, if present, are only mild in intensity. If another medical condition is present or there is a high risk for developing a medical condition (e.g., strong family history is present), the preoccupation is clearly excessive or disproportionate.

C. There is a high level of anxiety about health, and the individual is easily alarmed about personal health status.

D. The individual performs excessive health-related behaviors (e.g., repeatedly checks his or her body for signs of illness) or exhibits maladaptive avoidance (e.g., avoids doctor appointments and hospitals).

E. Illness preoccupation has been present for at least 6 months, but the specific illness that is feared may change over that period of time.

F. The illness-related preoccupation is not better explained by another mental disorder, such as somatic symptom disorder, panic disorder, generalized anxiety disorder, body dysmorphic disorder, obsessive-compulsive disorder, or delusional disorder, somatic type.

Specify whether:

Care-seeking type: Medical care, including physician visits or undergoing tests and procedures, is frequently used.

Care-avoidant type: Medical care is rarely used.

Diagnostic Features

Most individuals who previously would have been diagnosed with hypochondriasis in DSM-IV (preoccupation with having a serious disease based on the individual's misinterpretation of bodily symptoms) are now classified as having somatic symptom disorder; however, in one-third of cases, the diagnosis of illness anxiety disorder applies instead.

Illness anxiety disorder entails a preoccupation with having or acquiring a serious, undiagnosed medical illness (Criterion A). Somatic symptoms are not present or, if present, are only mild in intensity (Criterion B). A

thorough evaluation fails to identify a serious medical condition that accounts for the individual's concerns. While the concern may be derived from a nonpathological physical sign or sensation, the individual's distress emanates not primarily from the physical complaint itself but rather from his or her anxiety about the meaning, significance, or cause of the complaint (i.e., the suspected medical diagnosis). If a physical sign or symptom is present, it is often a normal physiological sensation (e.g., orthostatic dizziness), a benign and self-limited dysfunction (e.g., transient tinnitus), or a bodily discomfort not generally considered indicative of disease (e.g., belching). If a diagnosable medical condition is present, the individual's anxiety and preoccupation are clearly excessive and disproportionate to the severity of the condition (Criterion B). Most empirical evidence and existing literature pertain to previously defined DSM hypochondriasis and health anxiety, and it is unclear to what extent and how precisely they apply to the description of this new diagnosis.

The preoccupation with the idea that one is sick is accompanied by substantial anxiety about health and disease (Criterion C). Individuals with illness anxiety disorder are easily alarmed about illness, such as by hearing about someone else falling ill or reading a health-related news story. Their concerns about undiagnosed disease do not respond to appropriate medical reassurance, negative diagnostic tests, or benign course. The physician's attempts at reassurance and symptom palliation generally do not alleviate the individual's

concerns and may heighten them. Illness concerns assume a prominent place in the individual's life, affecting daily activities, and may even result in invalidism. Illness becomes a central feature of the individual's identity and self-image, a frequent topic of social discourse, and a characteristic response to stressful life events. Individuals with the disorder often examine themselves repeatedly (e.g., examining one's throat in the mirror) (Criterion D). They research their suspected disease excessively (e.g., on the Internet) and repeatedly seek reassurance from family, friends, or physicians. This incessant worrying often becomes frustrating for othe rs and may result in considerable strain within the family. In some cases, the anxiety leads to

maladaptive avoidance of situations (e.g., visiting sick family members) or activities (e.g., exercise) that these individuals fear might jeopardize their health.

Associated Features

Because they believe they are medically ill, individuals with illness anxiety disorder are encountered far more frequently in medical than in mental health settings. The majority of individuals with illness anxiety disorder have extensive yet unsatisfactory medical care. They generally have elevated rates of utilization of medical and mental health services compared with the general population. In a minority of cases of illness anxiety disorder, individuals are too anxious to seek medical attention and avoid medical health care.

They often consult multiple physicians for the same problem and obtain repeatedly negative diagnostic test results. At times, medical attention leads to a paradoxical exacerbation of anxiety or to iatrogenic complications from diagnostic tests and procedures. Individuals with the disorder are generally dissatisfied with their medical care and find it unhelpful, often feeling they are not being taken seriously by physicians. At times, these concerns may be justified, since physicians sometimes are dismissive or respond with frustration or hostility. This response can occasionally result in a failure to diagnose a medical condition that is present.

Prevalence

Prevalence estimates of illness anxiety disorder are based on estimates of the DSM-III and DSM-IV diagnosis *hypochondriasis* and health anxiety. The 1- to 2-year prevalence of health anxiety and/or disease conviction in community surveys and population-based samples from high-income countries such as the United States and Germany ranges from 1.3% to 10%. In ambulatory medical populations, the 6-month/1-year prevalence rates are between 2.2% and 8% across a range of countries, with weighted mean prevalence rates of 3%. By contrast, in a study of patients in specialty clinics, about one-fifth of individuals reported illness anxiety. The prevalence of the disorder is similar in men and women.

Development and Course

The development and course of illness anxiety disorder are unclear. Illness anxiety disorder is generally thought to be a chronic, episodic, and relapsing condition with an age at onset in early and middle adulthood. The disorder is thought to be rare in children, although the onset of health-related anxieties can occur in childhood or adolescence. In some population-based samples, health-related anxiety increases with age, but in others, health anxiety peaks in middle age, before declining in older age. The ages of individuals with high health anxiety in medical settings do not appear to differ from those of other individuals in those settings. In older individuals, health-related anxiety often focuses on memory loss and sensory loss.

Risk and Prognostic Factors

Environmental. Illness anxiety disorder may sometimes be precipitated by a major life stress or a serious but ultimately benign threat to the individual's health. A history of

childhood abuse or of a serious childhood illness, serious illness in a parent, or death of an ill parent during childhood may predispose to development of the disorder in adulthood.

Course modifiers. Approximately one-third to one-half of individuals with illness anxiety disorder have a transient form, which is associated with less psychiatric comorbidity, more medical comorbidity, and less severe illness anxiety disorder.

Culture-Related Diagnostic Issues

The diagnosis should be made with caution in individuals whose ideas about disease are congruent with widely held cultural beliefs. The prevalence appears to be similar across different countries, although little is known about the cross-cultural variation in phenomenology.

Functional Consequences of Illness Anxiety Disorder

Illness anxiety disorder causes substantial role impairment and decrements in physical function and health-related quality of life. Health concerns often interfere with interpersonal relationships, disrupt family life, and damage occupational performance.

Differential Diagnosis

Other medical conditions. The first differential diagnostic consideration is an underlying medical condition, including neurological or endocrine conditions, occult malignancies, and other diseases that affect multiple body systems. The presence of a medical condition does not rule out the possibility of coexisting illness anxiety disorder. If a medical condition is present, the health-related anxiety and disease concerns are clearly disproportionate to its seriousness. Transient preoccupations related to a medical condition do not constitute illness anxiety disorder.

Adjustment disorders. Health-related anxiety is a normal response to serious illness and is not a mental disorder. Such nonpathological health anxiety is clearly related to the medical condition and is typically time-limited. If the health anxiety is severe enough to cause clinically significant distress or impairment in one or more important areas of functioning, an adjustment disorder may be diagnosed. However, if disproportionate health-related anxiety persists for longer than 6 months, a diagnosis of illness anxiety disorder may apply.

Somatic symptom disorder. Both somatic symptom disorder and illness anxiety disorder may be characterized by a high level of anxiety about health and excessive health-related behaviors. They are differentiated by the fact that somatic symptom disorder requires the presence of somatic symptoms that are distressing or result in significant disruption of daily life, whereas in illness anxiety disorder, somatic symptoms either are not present or, if present, are only mild in intensity.

Anxiety disorders. In generalized anxiety disorder, individuals worry about multiple events, situations, or activities, only one of which may involve health. In panic disorder, the individual may be concerned that the panic attacks reflect the presence of a medical illness; however, although these individuals may have health anxiety, their anxiety is typically very acute

and episodic. In illness anxiety disorder, the health anxiety and fears are more persistent and enduring. Individuals with illness anxiety disorder may experience panic attacks that are triggered by their illness concerns.

Obsessive-compulsive and related disorders. Individuals with illness anxiety disorder may have intrusive thoughts about having a disease and also may have associated compulsive behaviors (e.g., seeking reassurance). However, in illness anxiety disorder, the preoccupations are usually focused on having a disease, whereas in obsessive-compulsive disorder (OCD), the thoughts are intrusive and are usually focused on fears of getting a disease in the future. Most individuals with OCD have obsessions or compulsions

involving other concerns in addition to fears about contracting disease. In body dysmorphic disorder, concerns are limited to the individual's physical appearance, which is viewed as defective or flawed.

Major depressive disorder. Some individuals with a major depressive episode ruminate about their health and worry excessively about illness. A separate diagnosis of illness anxiety disorder is not made if these concerns occur only during major depressive episodes. However, if excessive illness worry persists after remission of an episode of major depressive disorder, the diagnosis of illness anxiety disorder should be considered.

Psychotic disorders. Individuals with illness anxiety disorder are not delusional and can acknowledge the possibility that the feared disease is not present. Their ideas do not attain the rigidity and intensity seen in the somatic delusions occurring in psychotic disorders (e.g., schizophrenia; delusional disorder, somatic type; major depressive disorder, with psychotic features). True somatic delusions are generally more bizarre (e.g., that an organ is rotting or dead) than the concerns seen in illness anxiety disorder. The concerns seen in illness anxiety disorder, though not founded in reality, are plausible.

Comorbidity

Illness anxiety disorder co-occurs with anxiety disorders (in particular, generalized anxiety disorder and panic disorder), OCD, and depressive

disorders. Approximately two-thirds of individuals with illness anxiety disorder are likely to have at least one other comorbid major mental disorder. Individuals with illness anxiety disorder may have an elevated risk for personality disorders.

Functional Neurological Symptom Disorder (Conversion Disorder)

Diagnostic Criteria

A. One or more symptoms of altered voluntary motor or sensory function.
B. Clinical findings provide evidence of incompatibility between the symptom and recognized neurological or medical conditions.
C. The symptom or deficit is not better explained by another medical or mental disorder.
D. The symptom or deficit causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or warrants medical evaluation.

Coding note: The ICD-10-CM code depends on the symptom type (see below).

Specify symptom type:

F44.4 With weakness or paralysis

F44.4 With abnormal movement (e.g., tremor, dystonia, myoclonus, gait disorder)

F44.4 With swallowing symptoms

F44.4 With speech symptom (e.g., dysphonia, slurred speech)

F44.5 With attacks or seizures

F44.6 With anesthesia or sensory loss

F44.6 With special sensory symptom (e.g., visual, olfactory, or hearing disturbance)

F44.7 With mixed symptoms

Specify if:

Acute episode: Symptoms present for less than 6 months.

Persistent: Symptoms occurring for 6 months or more.

Specify if:

With psychological stressor *(specify stressor)*

Without psychological stressor

Diagnostic Features

In functional neurological symptom disorder (conversion disorder), there may be one or more neurological symptoms of various types. Motor symptoms include weakness or paralysis; abnormal movements, such as tremor, jerks, or dystonic movements; and gait abnormalities. Sensory symptoms include altered, reduced, or absent skin sensation, vision, or hearing. Episodes of apparent unresponsiveness with or without limb movements may resemble epileptic seizures, syncope, or coma (also called *dissociative*, *psychogenic*, or *nonepileptic seizures or attacks*). Other symptoms include reduced or absent speech volume (dysphonia/aphonia); altered speech articulation, prosody, or fluency; a sensation of a lump in the throat (globus); and diplopia. This disorder has been called "conversion disorder" in prior editions of DSM as well as in much of the psychiatric research literature. The term "conversion" originated in psychoanalytic theory, which proposes that unconscious psychic conflict is "converted" into physical symptoms.

The diagnosis rests on clinical findings that show clear evidence of incompatibility with recognized neurological disease. These should usually be elicited and interpreted in the context of the whole clinical picture by a health care professional with expertise in the diagnosis of neurological

conditions. The diagnosis is not one of exclusion and can be made in individuals who also have neurological diseases like epilepsy or multiple sclerosis. The diagnosis should not be made simply because results from investigations are normal or because the symptom is “bizarre.” Internal inconsistency during examination is one way to demonstrate incompatibility (i.e., demonstrating that physical signs elicited through one examination method are no longer present when tested a different way). There are dozens of examples of such “positive” examination findings. Examples of examination findings that indicate incompatibility with recognized neurological disease include the following:

- For functional limb weakness or paralysis: Hoover’s sign, in which weakness of hip extension returns to normal strength with contralateral hip flexion against resistance; the hip abductor sign, in which weakness of thigh abduction returns to normal with contralateral hip abduction against resistance; or a discrepancy between on-the-bed performance (e.g., weakness of ankle plantar flexion) compared with another task (e.g., ability to walk on tiptoes).
- For functional tremor: the tremor entrainment test, in which a tremor changes when the individual is distracted by copying the examiner in making a rhythmical movement with the contralateral hand or foot. The test is positive when the tremor “entrains” the rhythm of the unaffected hand or foot, the tremor is suppressed, or the individual cannot copy simple rhythmical movements. Other features of functional limb tremor include variability in frequency or direction of the tremor.
- For functional dystonia: individuals typically present with fixed inverted position of the ankle, a clenched fist, or unilateral contraction of platysma, often with sudden onset.
- For attacks resembling epileptic seizures or syncope (also called functional or dissociative [nonepileptic] seizures): features suggestive of functional neurological symptom disorder include persistent eye closure sometimes with resistance to opening, bilateral motor movements with preserved awareness, or a duration longer than 5 minutes. Clinical features usually need to be combined and may be supported with a normal simultaneous ictal electroencephalogram (although this alone does not exclude all forms of epilepsy or syncope.

- For functional speech symptoms: internal inconsistencies in speech articulation and phonation.

- For functional visual symptoms: a tubular visual field (i.e., tunnel vision) and tests that indicate internal inconsistency in visual acuity, such as the “fogging test” (i.e., while the individual views the eye chart with both eyes open, the “good” eye is subtly fogged so that any useful binocular vision must be a result of “bad” eye function).

It is important to note that the diagnosis of functional neurological symptom disorder should be based on the overall clinical picture and not on a single clinical finding.

Associated Features

Several associated features can support the diagnosis of functional neurological symptom disorder, although none are specific. There may be a history of other functional somatic symptoms or disorders, especially involving pain and fatigue. Onset may be associated with stress or trauma, either psychological or physical in nature. The potential etiological relevance of this stress or trauma may be suggested by a close temporal relationship. However, while assessment for stress and trauma is important, it may be absent in up to 50% of individuals, and the diagnosis should not be withheld if none is found.

Functional neurological symptom disorder is often associated with dissociative symptoms, such as depersonalization, derealization, and dissociative amnesia, particularly at symptom onset or during attacks.

The phenomenon of *la belle indifférence* (i.e., lack of concern about the nature or implications of the symptom) has been associated with functional neurological symptom disorder, but it is not specific and should not be used to make the diagnosis. Similarly, the concept of *secondary gain* (i.e., when individuals derive external benefits such as money or release from responsibilities) is also not specific to functional neurological symptom disorder.

Prevalence

Transient functional neurological symptoms are common, but the precise prevalence of the disorder is unknown. Based on research in the United States and northern Europe, the incidence of individual persistent functional neurological symptoms is estimated to be 4–12/100,000 per year.

Prevalence in specialty clinics appears to be higher, although data are limited. For example, 5% of outpatients ages 9–17 in a Japanese psychiatric clinic and 6% of adult and adolescent admissions to an inpatient psychiatric hospital in Oman received a diagnosis consistent with functional neurological symptom disorder. In neurology clinics, around 5%–15% of individuals have a diagnosis of functional neurological symptom disorder in studies from Scotland and Australia.

Development and Course

Onset has been reported throughout the life course. The mean onset of nonepileptic attacks peaks at ages 20–29 years, and motor symptoms have their mean onset at ages 30–39 years. The symptoms can be transient or persistent. The prognosis may be better in younger children than in adolescents and adults.

Risk and Prognostic Factors

Temperamental. Maladaptive personality traits, especially emotional instability, are commonly associated with functional neurological symptom disorder.

Environmental. There may be a history of childhood abuse and neglect. Stressful life events including physical injury are common but not universal triggering factors.

Genetic and physiological. The presence of neurological disease that causes similar symptoms is a risk factor (e.g., around one in five individuals with functional [nonepileptic] seizures also have epilepsy).

Course modifiers. Short duration of symptoms and agreement with the diagnosis are positive prognostic factors. Maladaptive personality traits, the presence of comorbid physical disease, and the receipt of disability benefits appear to be negative prognostic factors.

Culture-Related Diagnostic Issues

Episodes of unresponsiveness (including seizures) and motor symptoms are the most common functional neurological symptoms across cultural contexts. High comorbidity between functional neurological and dissociative symptoms is common cross-culturally, especially in individuals with nonepileptic seizures. Changes resembling functional neurological (and dissociative) symptoms are common in certain culturally sanctioned rituals. If the symptoms are fully explained within the particular cultural context and do not result in clinically significant distress or disability, then the diagnosis of functional neurological symptom disorder is not made.

Sex- and Gender-Related Diagnostic Issues

Functional neurological symptom disorder is two to three times more common in women for most symptom presentations. One large clinical study found higher rates of cognitive impairment and weakness in men and increased past sexual and physical trauma in women.

Association With Suicidal Thoughts or Behavior

Cohort studies of functional neurological symptom disorder mostly show higher rates of suicidal thoughts and attempts. Individuals with functional symptoms in a neurology clinic have a higher rate of suicidal thoughts than individuals with recognized neurological disease. A study in Turkey of 100 consecutive psychiatric outpatients with functional neurological symptom disorder found that a history of suicide attempt was associated with risky use of alcohol, a history of childhood maltreatment, and greater severity of dissociative symptoms as compared with those who did not attempt suicide.

Functional Consequences of Functional Neurological Symptom Disorder

Individuals with functional neurological symptom disorder may have substantial physical disability. The severity of disability can be similar to that experienced by individuals with comparable recognized medical conditions.

Differential Diagnosis

Recognized neurological disease. The main differential diagnosis is recognized neurological disease that might better explain the symptoms. After a thorough neurological assessment, an unexpected neurological disease cause for the symptoms is rarely found at follow-up. However, reassessment may be required if the symptoms appear to be progressive. Functional neurological symptom disorder commonly coexists with recognized neurological disease and may be part of the prodromal state of some progressive neurological diseases.

Somatic symptom disorder. Functional neurological symptom disorder may be diagnosed in addition to somatic symptom disorder. Most of the somatic symptoms

encountered in somatic symptom disorder cannot be demonstrated to be clearly incompatible with recognized neurological or medical disease, whereas in functional neurological symptom disorder, such incompatibility is required for the diagnosis.

Factitious disorder and malingering. Functional neurological symptom disorder describes genuinely experienced symptoms that are not intentionally produced (i.e., not feigned). However, definite evidence of feigning (e.g., marked discrepancy between reported and observed activities of daily living) would suggest malingering if the individual's apparent aim is to obtain an obvious external reward, or factitious disorder in the absence of such reward.

Dissociative disorders. Dissociative symptoms are common in individuals with functional neurological symptom disorder. If both functional neurological symptom disorder and a dissociative disorder are present, both diagnoses should be made.

Body dysmorphic disorder. Individuals with body dysmorphic disorder are excessively concerned about a perceived defect in their physical appearance but do not complain of symptoms of sensory or motor functioning in the affected body part.

Depressive disorders. In depressive disorders, individuals may report general heaviness of their limbs, whereas the weakness of functional

neurological symptom disorder is more focal and prominent. Depressive disorders are also differentiated by the presence of core depressive symptoms.

Panic disorder. Episodic neurological symptoms (e.g., tremor and paresthesia) can occur in both functional neurological symptom disorder and panic attacks. In panic attacks, the neurological symptoms are typically associated with characteristic cardiorespiratory symptoms and retained awareness. Loss of awareness with amnesia for the attack occurs in functional seizures but not in panic attacks.

Comorbidity

Anxiety disorders, especially panic disorder, and depressive disorders commonly co-occur with functional neurological symptom disorder. Somatic symptom disorder may co-occur as well. Personality disorders are more common in individuals with functional neurological symptom disorder than in the general population. Neurological or other medical conditions commonly coexist with functional neurological symptom disorder as well.

Psychological Factors Affecting Other Medical Conditions

Diagnostic Criteria F54

A. A medical symptom or condition (other than a mental disorder) is present.

B. Psychological or behavioral factors adversely affect the medical condition in one of the following ways:

   1. The factors have influenced the course of the medical condition as shown by a close temporal association between the psychological factors and the development or

exacerbation of, or delayed recovery from, the medical condition.

2. The factors interfere with the treatment of the medical condition (e.g., poor adherence).
3. The factors constitute additional well-established health risks for the individual.

4. The factors influence the underlying pathophysiology, precipitating or exacerbating symptoms or necessitating medical attention.

C. The psychological and behavioral factors in Criterion B are not better explained by another mental disorder (e.g., panic disorder, major depressive disorder, posttraumatic stress disorder).

Specify current severity:

Mild: Increases medical risk (e.g., inconsistent adherence with antihypertension treatment).

Moderate: Aggravates underlying medical condition (e.g., anxiety aggravating asthma).

Severe: Results in medical hospitalization or emergency room visit.

Extreme: Results in severe, life-threatening risk (e.g., ignoring heart attack symptoms).

Diagnostic Features

The essential feature of psychological factors affecting other medical conditions is the presence of one or more clinically significant psychological or behavioral factors that adversely affect a medical condition by increasing the risk for suffering, death, or disability (Criterion B). These factors can adversely affect the medical condition by influencing its course or treatment, by constituting an additional well-established health

risk factor, or by influencing the underlying pathophysiology to precipitate or exacerbate symptoms or to necessitate medical attention.

Psychological or behavioral factors include psychological distress, patterns of interpersonal interaction, coping styles, and maladaptive health behaviors, such as denial of symptoms or poor adherence to medical recommendations. Common clinical examples are anxiety-exacerbating asthma, denial of need for treatment for acute chest pain, and manipulation of insulin by an individual with diabetes wishing to lose weight. Many different psychological factors have been demonstrated to adversely influence medical conditions—for example, symptoms of depression or anxiety, stressful life events, relationship style, personality traits, and coping styles. The adverse effects can range from acute, with immediate medical consequences (e.g., Takotsubo cardiomyopathy), to chronic, occurring over a long period of time (e.g., chronic occupational stress increasing risk for hypertension). Affected medical conditions can be those with clear pathophysiology (e.g., diabetes, cancer, coronary disease), functional syndromes (e.g., migraine, irritable bowel syndrome, fibromyalgia), or idiopathic medical symptoms (e.g., pain, fatigue, dizziness).

This diagnosis should be reserved for situations in which the effect of the psychological factor on the medical condition is evident and the psychological factor has clinically significant effects on the course or outcome of the medical condition. Abnormal psychological or behavioral symptoms that develop in response to a medical condition are more properly coded as an adjustment disorder (a clinically significant psychological response to an identifiable stressor). There must be reasonable evidence to suggest an association between the psychological factors and the medical condition, although it may often not be possible to demonstrate direct causality or the mechanisms underlying the relationship.

Prevalence

The prevalence of psychological factors affecting other medical conditions is unclear. In U.S. private insurance billing data, it was a more common diagnosis than DSM-IV somatic symptom disorders.

Development and Course

Psychological factors affecting other medical conditions can occur across the lifespan. Particularly with young children, corroborative history from parents or school can assist the

diagnostic evaluation. Some conditions are characteristic of particular life stages (e.g., in older individuals, the stress associated with acting as a caregiver for an ill spouse or partner).

Culture-Related Diagnostic Issues

Many differences between cultural contexts may influence psychological factors and their effects on medical conditions, such as those in language and communication style, idioms of distress, explanatory models of illness, patterns of seeking health care, service availability and organization, doctor-patient relationships and other healing practices, family and gender roles, and attitudes toward pain and death. Psychological factors affecting other medical conditions must be differentiated from culturally specific coping behaviors such as accessing faith, spiritual, or traditional healers or other variations in illness management that are acceptable within cultural contexts and represent an attempt to help heal the medical condition. These local practices may complement rather than obstruct evidence-based interventions. Use of alternative healing practices may delay use of medical services and affect outcomes, but when the intent of the healing practice is to address the problem in a culturally sanctioned way, these practices should not be pathologized as psychological factors affecting other medical conditions.

Functional Consequences of Psychological Factors Affecting Other Medical Conditions

Psychological and behavioral factors have been demonstrated to affect the course of many medical diseases.

Differential Diagnosis

Mental disorder due to another medical condition. A temporal association between symptoms of a mental disorder and those of a medical condition is also characteristic of a mental disorder due to another medical condition, but the presumed causality is in the opposite direction. In a mental disorder due to another medical condition, the medical condition is judged to be causing the mental disorder through a direct physiological mechanism. In psychological factors affecting other medical conditions, the psychological or behavioral factors are judged to affect the course of the medical condition.

Adjustment disorders. Abnormal psychological or behavioral symptoms that develop in response to a medical condition are more properly coded as an adjustment disorder (a clinically significant psychological response to an identifiable stressor). For example, an individual with angina that is precipitated whenever he becomes enraged would be diagnosed as having psychological factors affecting other medical conditions, whereas an individual with angina who developed maladaptive anticipatory anxiety would be diagnosed as having an adjustment disorder with anxiety. In clinical practice, however, psychological factors and a medical condition are often mutually exacerbating (e.g., anxiety as both a precipitant and a consequence of angina), in which case the distinction is arbitrary. Other mental disorders frequently result in medical complications, most notably substance use disorders (e.g., alcohol use disorder, tobacco use disorder). If an individual has a coexisting major mental disorder that adversely affects or causes another medical condition, diagnoses of the mental disorder and the medical condition are usually sufficient. Psychological factors affecting other medical conditions is diagnosed when the psychological traits or behaviors do not meet criteria for a mental diagnosis.

Somatic symptom disorder. Somatic symptom disorder is characterized by a combination of distressing somatic symptoms and excessive or maladaptive thoughts, feelings, and behavior in response to these symptoms or associated health concerns. The individual may or may not have a diagnosable medical condition. In contrast, in psychological factors

affecting other medical conditions, the psychological factors adversely affect a medical condition; the individual's thoughts, feelings, and behavior are not necessarily excessive. The difference is one of emphasis, rather than a clear-cut distinction. In psychological factors affecting other medical conditions, the emphasis is on the exacerbation of the medical condition (e.g., an individual with angina that is precipitated whenever he becomes anxious). In somatic symptom disorder, the emphasis is on maladaptive thoughts, feelings, and behavior (e.g., an individual with angina who worries constantly that she will have a heart attack, takes her blood pressure multiple times per day, and restricts her activities).

Illness anxiety disorder. Illness anxiety disorder is characterized by high illness anxiety that is distressing and/or disruptive to daily life with minimal somatic symptoms. The focus of clinical concern is the individual's worry about having a disease; in most cases, no serious disease is present. In psychological factors affecting other medical conditions, anxiety may be a relevant psychological factor affecting a medical condition, but the clinical concern is the adverse effects on the medical condition.

Comorbidity

By definition, the diagnosis of psychological factors affecting other medical conditions entails a relevant psychological or behavioral syndrome or trait and a comorbid medical condition.

Factitious Disorder

Diagnostic Criteria

Factitious Disorder Imposed on Self **F68.10**

A. Falsification of physical or psychological signs or symptoms, or induction of injury or disease, associated with identified deception.

B. The individual presents himself or herself to others as ill, impaired, or injured.

C. The deceptive behavior is evident even in the absence of obvious external rewards.

D. The behavior is not better explained by another mental disorder, such as delusional disorder or another psychotic disorder.

Specify:

Single episode

Recurrent episodes (two or more events of falsification of illness and/or induction of injury)

Factitious Disorder Imposed on Another (Previously Factitious Disorder by Proxy) F68.A

A. Falsification of physical or psychological signs or symptoms, or induction of injury or disease, in another, associated with identified deception.

B. The individual presents another individual (victim) to others as ill, impaired, or injured.

C. The deceptive behavior is evident even in the absence of obvious external rewards.

D. The behavior is not better explained by another mental disorder, such as delusional disorder or another psychotic disorder.

Note: The perpetrator, not the victim, receives this diagnosis.

Specify:

Single episode

Recurrent episodes (two or more events of falsification of illness and/or induction of injury)

Recording Procedures

When an individual falsifies illness in another (e.g., children, adults, pets), the diagnosis is factitious disorder imposed on another. The perpetrator, not the victim, is given the diagnosis. The victim may be given an abuse diagnosis (e.g., T74.12X; see the chapter “Other Conditions That May Be a Focus of Clinical Attention”). If an individual with factitious disorder imposed on another has also deceptively represented his or her own illness or injury, both factitious disorder imposed on self and on another can be diagnosed.

Diagnostic Features

The essential feature of factitious disorder is the falsification of medical or psychological signs and symptoms in the individual or others that are associated with the identified deception. Individuals with factitious disorder can also seek treatment for themselves or another following induction of injury or disease. The diagnosis requires demonstrating that the individual is taking surreptitious actions to misrepresent, simulate, or cause signs or symptoms of illness or injury even in the absence of obvious external rewards. The diagnosis of factitious disorder emphasizes the objective identification of falsification of signs and symptoms of illness and not the individual motivations of the falsifier. Methods of illness falsification can include exaggeration, fabrication, simulation, and induction. While a preexisting medical condition may be present, the deceptive behavior or induction of injury associated with deception causes others to view such individuals (or, in the case of factitious disorder imposed on another, the victim) as more ill or impaired, and this can lead to excessive clinical intervention. Individuals with factitious disorder might, for example, report feelings of depression and suicidal thoughts or behavior following the death of a spouse despite the death not being true or the individual’s not having a spouse; deceptively report episodes of neurological symptoms (e.g., seizures, dizziness, or blacking out); manipulate a laboratory test (e.g., by adding blood to urine) to falsely indicate an abnormality; falsify medical records to indicate an illness; ingest a substance (e.g., insulin or warfarin) to induce an abnormal laboratory result or illness; or physically injure themselves or induce illness in themselves or another (e.g., by injecting

fecal material to produce an abscess or to induce sepsis). Although individuals with factitious disorder most often present to health care professionals for treatment of their factitious symptoms, some individuals with factitious disorder choose to mislead community members in person or online about illness or injury without necessarily engaging health care professionals.

Associated Features

Individuals with factitious disorder imposed on self or factitious disorder imposed on another are at risk for experiencing great psychological distress or functional impairment by causing harm to themselves and others. Family, friends, faith leaders, and health care professionals are also often adversely affected by their behavior (e.g., devoted time, attention, and resources to provide medical care and emotional support to the falsifier). Individuals with factitious disorder imposed on another sometimes falsely allege the presence of educational deficits or disabilities in their children for which they demand special attention, often at considerable inconvenience to education professionals.

Whereas some aspects of factitious disorders might represent criminal behavior (e.g., factitious disorder imposed on another, in which the parent's actions represent abuse and maltreatment of a child), such criminal behavior and mental illness are not mutually exclusive. Moreover, such behaviors, including the induction of injury or disease, are associated with deception.

Prevalence

The prevalence of factitious disorder is unknown, likely because of the role of deception in this population. Further complicating efforts at determining prevalence is the fact that health care professionals infrequently record the diagnosis, even in recognized cases.

Based on a study of general hospital inpatients in the United States referred for psychiatric consultation, it is estimated that almost 1% have presentations that meet the criteria for factitious disorder. Factitious

disorder imposed on self or another appears to be encountered more frequently in tertiary care settings than at primary care sites.

Development and Course

The course of factitious disorder is usually one of intermittent episodes. Single episodes and episodes that are characterized as persistent and unremitting are both less common. Onset is usually in early adulthood, often after hospitalization for a medical condition or a mental disorder. When imposed on another, the disorder may begin after hospitalization of the individual's child or other dependent. In individuals with recurrent episodes of falsification of signs and symptoms of illness and/or induction of injury, this pattern of successive deceptive contact with medical personnel, including hospitalizations, may become lifelong.

Sex- and Gender-Related Diagnostic Issues

While the prevalence is not known, a pooled analysis of all case series and studies finds that two-thirds of individuals with factitious disorder are women and one-third are men.

Differential Diagnosis

Deception to avoid legal liability. Caregivers who lie about abuse injuries in dependents solely to protect themselves from liability are not diagnosed with factitious disorder imposed on another because protection from liability is an external reward (Criterion C, the deceptive behavior is evident even in the absence of obvious external rewards). Such caregivers who, upon observation, analysis of medical records, and/or interviews with others, are found to lie more extensively than needed for immediate self-protection are diagnosed with factitious disorder imposed on another.

Somatic symptom and related disorders. In somatic symptom disorder and the care-seeking type of illness anxiety disorder, there may be excessive attention and treatment seeking for perceived medical concerns, but there is no evidence that the individual is providing false information or behaving deceptively.

Malingering.

Malingering is differentiated from factitious disorder by the intentional reporting of symptoms for personal gain (e.g., money, time off work). In contrast, the diagnosis of factitious disorder requires that the illness falsification is not fully accounted for by external rewards. Factitious disorder and malingering are not mutually exclusive, however. The motives in any single case might be multiple and shifting depending on the circumstances and reactions of others.

Functional neurological symptom disorder (conversion disorder). Functional neurological symptom disorder is characterized by neurological symptoms that are inconsistent with neurological pathophysiology. Factitious disorder with neurological symptoms is distinguished from functional neurological symptom disorder by evidence of deceptive falsification of symptoms.

Borderline personality disorder. Deliberate physical self-harm in the absence of suicidal intent can also occur in association with other mental disorders such as borderline personality disorder. Factitious disorder requires that the induction of injury occur in association with deception.

Medical condition or mental disorder not associated with intentional symptom falsification.
Presentation of signs and symptoms of illness that do not conform to an identifiable medical condition or mental disorder increases the likelihood of the presence of a factitious disorder. However, the diagnosis of factitious disorder does not exclude the

presence of a true medical condition or mental disorder, as comorbid illness often occurs in the individual along with factitious disorder. For example, individuals who might manipulate blood sugar levels to produce symptoms may also have diabetes.

Other Specified Somatic Symptom and Related Disorder

F45.8

This category applies to presentations in which symptoms characteristic of a somatic symptom and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the somatic symptom and related disorders diagnostic class.

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Brief somatic symptom disorder:** Duration of symptoms is less than 6 months.
2. **Brief illness anxiety disorder:** Duration of symptoms is less than 6 months.
3. **Illness anxiety disorder without excessive health-related behaviors or maladaptive avoidance:** Criterion D for illness anxiety disorder is not met.
4. **Pseudocyesis:** A false belief of being pregnant that is associated with objective signs and reported symptoms of pregnancy.

Unspecified Somatic Symptom and Related Disorder

F45.9

This category applies to presentations in which symptoms characteristic of a somatic symptom and related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the somatic symptom and related disorders diagnostic class. The unspecified somatic symptom and related disorder category should not be used unless there are decidedly unusual situations

where there is insufficient information to make a more specific diagnosis.

Feeding and Eating Disorders

Feeding and eating disorders are characterized by a persistent disturbance of eating or eating-related behavior that results in the altered consumption or absorption of food and that significantly impairs physical health or psychosocial functioning. Diagnostic criteria are provided for pica, rumination disorder, avoidant/restrictive food intake disorder, anorexia nervosa, bulimia nervosa, and binge-eating disorder.

The diagnostic criteria for anorexia nervosa, bulimia nervosa, and binge-eating disorder result in a classification scheme that is mutually exclusive, so that during a single episode, only one of these diagnoses can be assigned. The rationale for this approach is that, despite a number of common psychological and behavioral features, the disorders differ substantially in clinical course, outcome, and treatment needs.

Some individuals with disorders described in this chapter report eating-related symptoms resembling those typically endorsed by individuals with substance use disorders, such as craving and patterns of compulsive use. This resemblance may reflect the involvement of the same neural systems, including those implicated in regulatory self-control and reward, in both groups of disorders. However, the relative contributions of shared and distinct factors in the development and perpetuation of eating and substance use disorders remain insufficiently understood.

Finally, obesity is not included in DSM-5 as a mental disorder. Obesity (excess body fat) results from the long-term excess of energy intake relative to energy expenditure. A range of genetic, physiological, behavioral, and environmental factors that vary across individuals contributes to the development of obesity; thus, obesity is not considered a mental disorder. However, there are robust associations between obesity and a number of mental disorders (e.g., binge-eating disorder, depressive and bipolar disorders, schizophrenia). The side effects of some psychotropic

medications contribute importantly to the development of obesity, and obesity may be a risk factor for the development of some mental disorders (e.g., depressive disorders).

Pica

Diagnostic Criteria

A. Persistent eating of nonnutritive, nonfood substances over a period of at least 1 month.
B. The eating of nonnutritive, nonfood substances is inappropriate to the developmental level of the individual.
C. The eating behavior is not part of a culturally supported or socially normative practice.
D. If the eating behavior occurs in the context of another mental disorder (e.g., intellectual developmental disorder [intellectual disability], autism spectrum disorder, schizophrenia) or medical condition (including pregnancy), it is sufficiently severe to warrant additional clinical attention.

Coding note: The ICD-10-CM codes for pica are **F98.3** in children and **F50.89** in adults.

Specify if:

In remission: After full criteria for pica were previously met, the criteria have not been met for a sustained period of time.

Diagnostic Features

The essential feature of pica is the eating of one or more nonnutritive, nonfood substances on a persistent basis over a period of at least 1 month (Criterion A) that is severe enough to warrant clinical attention. Typical

substances ingested tend to vary with age and availability and might include paper, soap, cloth, hair, string, wool, soil, chalk, talcum powder, paint, gum, metal, pebbles, charcoal or coal, ash, clay, starch, or ice. The term *nonfood* is included because the diagnosis of pica does not apply to ingestion of diet products that have minimal nutritional content. There is typically no aversion to food in general. The eating of nonnutritive, nonfood substances must be developmentally inappropriate (Criterion B) and not part of a culturally supported or socially normative practice (Criterion C). A minimum age of 2 years is suggested for a pica diagnosis to exclude developmentally normal mouthing of objects by infants that results in ingestion. The eating of nonnutritive, nonfood substances can be an associated feature of other mental disorders (e.g., intellectual developmental disorder [intellectual disability], autism spectrum disorder, schizophrenia). If the eating behavior occurs exclusively in the context of another mental disorder, a separate diagnosis of pica should be made only if the eating behavior is sufficiently severe to warrant additional clinical attention (Criterion D).

Associated Features

Although deficiencies in vitamins or minerals (e.g., zinc, iron) have been reported, often no specific biological abnormalities are found. In some cases, pica comes to clinical attention only following general medical complications (e.g., mechanical bowel problems; intestinal obstruction, such as that resulting from a bezoar; intestinal perforation; infections such as toxoplasmosis and toxocariasis as a result of ingesting feces or dirt; poisoning, such as by ingestion of lead-based paint).

Prevalence

Limited data suggest that the prevalence of pica is approximately 5% among school-age children. Roughly one-third of pregnant women, especially those with food insecurity (i.e., without reliable access to affordable and nutritious food), engage in pica. Conditions associated with pica include lack of available food and vitamin deficiency.

Development and Course

Onset of pica can occur in childhood, adolescence, or adulthood, although childhood onset is most commonly reported. Pica can occur in otherwise normally developing children, whereas in adults it appears more likely to occur in the context of intellectual developmental disorder or other mental disorders. The course of the disorder can be protracted and can result in medical emergencies (e.g., intestinal obstruction, acute weight loss, poisoning). The disorder can potentially be fatal depending on substances ingested.

Risk and Prognostic Factors

Environmental. Neglect, lack of supervision, and developmental delay can increase the risk for this condition.

Culture-Related Diagnostic Issues

In some populations, the eating of earth or other seemingly nonnutritive substances is believed to be of spiritual, medicinal, or other social value, or may be a culturally supported

or socially normative practice. Such behavior does not warrant a diagnosis of pica (Criterion C). Pica behavior may be prevalent in some cultural groups, but it should not be assumed to be socially normative without further evaluation.

Sex- and Gender-Related Diagnostic Issues

Pica occurs in both genders. The eating of nonnutritive, nonfood substances may manifest in pregnancy, when specific cravings (e.g., chalk or ice) may occur. The diagnosis of pica during pregnancy is appropriate only if such cravings lead to the ingestion of nonnutritive, nonfood substances to the extent that the eating of these substances poses potential medical risks. A worldwide meta-analysis showed the prevalence rate of pica to be 28% during pregnancy and/or the postpartum period.

Diagnostic Markers

Abdominal flat plate radiography, ultrasound, and other scanning methods may reveal obstructions related to pica. Blood tests and other laboratory tests can be used to ascertain levels of poisoning or the nature of infection.

Functional Consequences of Pica

Pica can significantly impair physical functioning, but it is rarely the sole cause of impairment in social functioning. Pica often occurs with other disorders associated with impaired social functioning.

Differential Diagnosis

Eating of nonnutritive, nonfood substances may occur during the course of other mental disorders (e.g., autism spectrum disorder, schizophrenia) and in Kleine-Levin syndrome. In any such instance, an additional diagnosis of pica should be given only if the eating behavior is sufficiently persistent and severe to warrant additional clinical attention.

Anorexia nervosa. Pica can usually be distinguished from the other feeding and eating disorders by the consumption of nonnutritive, nonfood substances. It is important to note, however, that some presentations of anorexia nervosa include ingestion of nonnutritive, nonfood substances, such as paper tissues, as a means of attempting to control appetite. In such cases, when the eating of nonnutritive, nonfood substances is primarily used as a means of weight control, anorexia nervosa should be the primary diagnosis.

Factitious disorder. Some individuals with factitious disorder may intentionally ingest foreign objects as part of the pattern of falsification of physical symptoms. In such instances, there is an element of deception that is consistent with deliberate induction of injury or disease.

Nonsuicidal self-injury and nonsuicidal self-injury behaviors in personality disorders. Some individuals may swallow potentially harmful items (e.g., pins, needles, knives) in the context of maladaptive behavior patterns associated with personality disorders or nonsuicidal self-injury.

Comorbidity

Disorders most commonly comorbid with pica are autism spectrum disorder and intellectual developmental disorder (intellectual disability) and, to a lesser degree, schizophrenia and obsessive-compulsive disorder. Pica can be associated with trichotillomania (hair-pulling disorder) and excoriation (skin-picking) disorder. In comorbid presentations, the hair or skin is typically ingested. Pica can also be associated with avoidant/restrictive food intake disorder, particularly in individuals with a strong sensory component to their presentation. When an individual is known to have pica, assessment should include consideration of the possibility of gastrointestinal complications, poisoning, infection, and nutritional deficiency.

Rumination Disorder

Diagnostic Criteria F98.21

A. Repeated regurgitation of food over a period of at least 1 month. Regurgitated food may be re-chewed, re-swallowed, or spit out.
B. The repeated regurgitation is not attributable to an associated gastrointestinal or other medical condition (e.g., gastroesophageal reflux, pyloric stenosis).
C. The eating disturbance does not occur exclusively during the course of anorexia nervosa, bulimia nervosa, binge-eating disorder, or avoidant/restrictive food intake disorder.
D. If the symptoms occur in the context of another mental disorder (e.g., intellectual developmental disorder [intellectual disability] or another neurodevelopmental disorder), they are sufficiently severe to warrant additional clinical attention.

Specify if:

In remission: After full criteria for rumination disorder were previously met, the criteria have not been met for a sustained

period of time.

Diagnostic Features

The essential feature of rumination disorder is the repeated regurgitation of food occurring after feeding or eating over a period of at least 1 month (Criterion A). Previously swallowed food that may be partially digested is brought up into the mouth without apparent nausea, involuntary retching, or disgust. The food may be re-chewed and then ejected from the mouth or re-swallowed. Regurgitation in rumination disorder should be frequent, occurring at least several times per week, typically daily. The behavior is not better explained by an associated gastrointestinal or other medical condition (e.g., gastroesophageal reflux, pyloric stenosis) (Criterion B) and does not occur exclusively during the course of anorexia nervosa, bulimia nervosa, binge-eating disorder, or avoidant/restrictive food intake disorder (Criterion C). If the symptoms occur in the context of another mental disorder (e.g., intellectual developmental disorder [intellectual disability]), they must be sufficiently severe to warrant additional clinical attention (Criterion D) and should represent a primary aspect of the individual's presentation requiring intervention. The disorder may be diagnosed across the life span, particularly in individuals who also have intellectual developmental disorder. Many individuals with rumination disorder can be directly observed engaging in the behavior by the clinician. In other instances diagnosis can be made on the basis of self-report or corroborative information from parents or caregivers. Individuals may describe the behavior as habitual or outside of their control.

Associated Features

Infants with rumination disorder display a characteristic position of straining and arching the back with the head held back, making sucking movements with their tongue. They may give the impression of gaining satisfaction from the activity. They may be irritable and hungry between episodes of regurgitation. Weight loss and failure to make expected weight gains are common features in infants with rumination disorder. Malnutrition may occur despite the infant's apparent hunger and the ingestion of

relatively large amounts of food, particularly in severe cases, when regurgitation immediately follows each feeding episode and regurgitated food is expelled. Malnutrition might also occur in older children and adults, particularly when the regurgitation is accompanied by restriction of intake. Adolescents and adults may attempt to disguise the regurgitation behavior by placing a hand over the mouth or coughing. Some will avoid eating with others because of the acknowledged social undesirability of the

behavior. This may extend to an avoidance of eating prior to social situations, such as work or school (e.g., avoiding breakfast because it may be followed by regurgitation).

Prevalence

Although rumination disorder was historically described primarily among individuals with intellectual disability, the limited European data available on prevalence suggest that the disorder may occur in approximately 1%–2% of grade-school-age children.

Development and Course

Onset of rumination disorder can occur in infancy, childhood, adolescence, or adulthood. The age at onset in infants is usually between ages 3 and 12 months. In infants, the disorder frequently remits spontaneously, but its course can be protracted and can result in medical emergencies (e.g., severe malnutrition). It can potentially be fatal, particularly in infancy. Rumination disorder can have an episodic course or occur continuously until treated. In infants, as well as in older individuals with intellectual developmental disorder or other neurodevelopmental disorders, the regurgitation and rumination behavior appears to have a self-soothing or self-stimulating function, similar to that of other repetitive motor behaviors such as head banging.

Risk and Prognostic Factors

Environmental.

Psychosocial problems such as lack of stimulation, neglect, stressful life situations, and problems in the parent-child relationship may be predisposing factors in infants and young children.

Functional Consequences of Rumination Disorder

Malnutrition secondary to repeated regurgitation may be associated with growth delay and have a negative effect on development and learning potential. Some older individuals with rumination disorder deliberately restrict their food intake because of the social undesirability of regurgitation. They may therefore present with weight loss or low weight. In older children, adolescents, and adults, social functioning is more likely to be adversely affected.

Differential Diagnosis

Gastrointestinal conditions. It is important to differentiate regurgitation in rumination disorder from other conditions characterized by gastroesophageal reflux or vomiting, such as gastroparesis, pyloric stenosis, hiatal hernia, and Sandifer syndrome in infants. These other medical conditions can usually be ruled out on the basis of history and clinical observation.

Anorexia nervosa and bulimia nervosa. Individuals with anorexia nervosa and bulimia nervosa may also engage in regurgitation with subsequent spitting out of food as a means of disposing of ingested calories because of concerns about weight gain.

Comorbidity

Regurgitation with associated rumination can occur in the context of a concurrent medical condition or another mental disorder (e.g., generalized anxiety disorder). When the regurgitation occurs in this context, a diagnosis of rumination disorder is appropriate only when the severity of the disturbance exceeds that routinely associated with such conditions or disorders and warrants additional clinical attention.

Avoidant/Restrictive Food Intake Disorder

Diagnostic Criteria F50.82

A. An eating or feeding disturbance (e.g., apparent lack of interest in eating or food; avoidance based on the sensory characteristics of food; concern about aversive consequences of eating) associated with one (or more) of the following:

   1. Significant weight loss (or failure to achieve expected weight gain or faltering growth in children).
   2. Significant nutritional deficiency.
   3. Dependence on enteral feeding or oral nutritional supplements.
   4. Marked interference with psychosocial functioning.

B. The disturbance is not better explained by lack of available food or by an associated culturally sanctioned practice.

C. The eating disturbance does not occur exclusively during the course of anorexia nervosa or bulimia nervosa, and there is no evidence of a disturbance in the way in which one's body weight or shape is experienced.

D. The eating disturbance is not attributable to a concurrent medical condition or not better explained by another mental disorder. When the eating disturbance occurs in the context of another condition or disorder, the severity of the eating disturbance exceeds that routinely associated with the condition or disorder and warrants additional clinical attention.

Specify if:

In remission: After full criteria for avoidant/restrictive food intake disorder were previously met, the criteria have not been met for a sustained period of time.

Diagnostic Features

Avoidant/restrictive food intake disorder replaces and extends the DSM-IV diagnosis of feeding disorder of infancy or early childhood to include older children, adolescents, and adults. The main diagnostic feature of avoidant/restrictive food intake disorder is avoidance or restriction of food intake that is associated with one or more of the following consequences: significant weight loss, significant nutritional deficiency (or related health impact), dependence on enteral feeding or oral nutritional supplements, or marked interference with psychosocial functioning (Criterion A).

In some individuals, food avoidance or restriction is based on the sensory characteristics of qualities of food, such as extreme sensitivity to appearance, color, smell, texture, temperature, or taste. Such behavior has been described as “restrictive eating,” “selective eating,” “choosy eating,” “perseverant eating,” “chronic food refusal,” and “food neophobia” and may manifest as refusal to eat particular brands of foods or to tolerate the smell of food being eaten by others. Individuals with heightened sensory sensitivities associated with autism may show similar behaviors.

In other individuals, food avoidance or restriction represents a conditioned negative response associated with food intake following, or in anticipation of, an aversive experience, such as choking; a traumatic procedure, usually involving the gastrointestinal tract (e.g., esophagoscopy); or repeated vomiting. The terms *functional dysphagia* and *globus hystericus* have also been used for such conditions.

In yet other individuals, food avoidance or restriction manifests as a lack of interest in eating or food.

The determination of whether weight loss is significant (Criterion A1) is a clinical judgment; instead of losing weight, children and adolescents who have not completed growth may not maintain weight or height increases along their developmental trajectory.

Determination of significant nutritional deficiency (Criterion A2) is also based on clinical assessment (e.g., assessment of dietary intake, physical

examination, and laboratory testing), and related impact on physical health can be of a similar severity to that seen in anorexia nervosa (e.g., hypothermia, bradycardia, anemia). In severe cases, particularly in infants, malnutrition can be life-threatening. “Dependence” on enteral feeding or oral nutritional supplements (Criterion A3) means that supplementary feeding is required to sustain adequate intake. Examples of individuals requiring supplementary feeding include infants with failure to thrive who require nasogastric tube feeding, children with neurodevelopmental disorders who are dependent on nutritionally complete supplements, and individuals who rely on gastrostomy tube feeding or complete oral nutrition supplements in the absence of an underlying medical condition. Inability to participate in normal social activities, such as eating with others, attending school or work, or sustaining relationships as a result of the disturbance would indicate marked interference with psychosocial functioning (Criterion A4). Substantial disruption of family functioning (e.g., marked restriction of foods permitted in the home, inordinate accommodations to provide foods from specific grocery stores or restaurants) may also satisfy Criterion A4.

Avoidant/restrictive food intake disorder does not include avoidance or restriction of food intake related to lack of availability of food (e.g., food insecurity) or to cultural practices (e.g., religious fasting or normal dieting) (Criterion B). The disturbance is not better explained by excessive concern about body weight or shape (Criterion C) or by concurrent medical factors or mental disorders (Criterion D).

Associated Features

Several features may be associated with food avoidance or reduced food intake, and these features may differ across ages. Very young infants may present with food refusal, gagging, or vomiting. Infants and young children may not engage with a primary caregiver during feeding or communicate hunger in favor of other activities. In older children and adolescents, food avoidance or restriction may be associated with more generalized emotional difficulties that do not meet diagnostic criteria for an anxiety, depressive, or bipolar disorder, sometimes called “food avoidance emotional disorder.”

Prevalence

Little information is available on the prevalence of avoidant/restrictive food intake disorder. A study in Australia reported a frequency of 0.3% among individuals age 15 years or older.

Development and Course

Food avoidance or restriction associated with insufficient intake or lack of interest in eating most commonly develops in infancy or early childhood and may persist in adulthood. Likewise, avoidance based on sensory characteristics of food tends to arise in the first decade of life but may persist into adulthood. Avoidance related to aversive consequences can arise at any age. The scant literature regarding long-term outcomes suggests that food avoidance or restriction based on sensory aspects is relatively stable and long-standing, but when persisting into adulthood, such avoidance/restriction can be associated with relatively normal functioning. There is currently insufficient evidence directly linking avoidant/restrictive food intake disorder and subsequent onset of an eating disorder.

Infants with avoidant/restrictive food intake disorder may be irritable and difficult to console during feeding, or may appear apathetic and withdrawn. In some instances, caregiver-child interaction may contribute to the infant's feeding problem (e.g., presenting food inappropriately, or interpreting the infant's behavior as an act of aggression or rejection). Inadequate nutritional intake may exacerbate the associated features (e.g., irritability, developmental lags) and further contribute to feeding difficulties. Associated factors

include infant temperament or developmental impairments that reduce an infant's responsiveness to feeding. Coexisting parental psychopathology, or child abuse or neglect, is suggested if feeding and weight improve in response to changing caregivers. In infants, children, and prepubertal adolescents, avoidant/restrictive food intake disorder may be associated with growth delay, and the resulting malnutrition negatively affects development and learning potential. In older children, adolescents, and

adults, social functioning tends to be adversely affected. Regardless of the age, family function may be affected, with heightened stress at mealtimes and in other feeding or eating contexts involving friends and relatives.

Avoidant/restrictive food intake disorder manifests more commonly in children and adolescents than in adults, and there may be a long delay between onset and clinical presentation. Triggers for presentation vary considerably and include physical, social, and emotional difficulties.

Risk and Prognostic Factors

Temperamental. Anxiety disorders, autism spectrum disorder, obsessive-compulsive disorder, and attention-deficit/hyperactivity disorder may increase risk for avoidant or restrictive feeding or eating behavior characteristic of the disorder.

Environmental. Environmental risk factors for avoidant/restrictive food intake disorder include familial anxiety. Higher rates of feeding disturbances may occur in children of mothers with eating disorders.

Genetic and physiological. History of gastrointestinal conditions, gastroesophageal reflux disease, vomiting, and a range of other medical problems has been associated with feeding and eating behaviors characteristic of avoidant/restrictive food intake disorder.

Culture-Related Diagnostic Issues

Presentations similar to avoidant/restrictive food intake disorder occur in various populations, including in the United States, Canada, Australia, Europe, Japan, and China. Avoidant/restrictive food intake disorder should not be diagnosed when avoidance of food intake is solely related to specific religious or cultural practices.

Sex- and Gender-Related Diagnostic Issues

Avoidant/restrictive food intake disorder appears to be approximately equally common in boys and girls, but avoidant/restrictive food intake disorder comorbid with autism spectrum disorder has a male predominance. Food avoidance or restriction related to altered sensory sensitivities can

occur in some physiological conditions, most notably pregnancy, but is not usually extreme and does not meet full criteria for the disorder.

Functional Consequences of Avoidant/Restrictive Food Intake Disorder

Associated developmental and functional limitations include impairment of physical development and social difficulties that can have a significant negative impact on family function.

Differential Diagnosis

Restriction of food intake is a nonspecific symptom that can accompany a number of mental disorders and medical conditions and that can also be developmentally appropriate. Avoidant/restrictive food intake disorder can be diagnosed concurrently with the disorders below if all criteria are met, and the eating disturbance requires specific clinical attention.

Other medical conditions (e.g., gastrointestinal disease, food allergies and intolerances, occult malignancies).

Restriction of food intake may occur in other medical conditions, especially those with ongoing symptoms such as vomiting, loss of appetite, nausea, abdominal pain, or diarrhea. A diagnosis of avoidant/restrictive food intake disorder requires that the disturbance of intake is beyond that directly accounted for by physical symptoms consistent with a medical condition; the eating disturbance may also persist after being triggered by a medical condition and following resolution of the medical condition.

Underlying medical or comorbid mental conditions may complicate feeding and eating. Because older individuals, postsurgical patients, and individuals receiving chemotherapy often lose their appetite, an additional diagnosis of avoidant/restrictive food intake disorder requires that the eating disturbance is a primary focus for intervention.

Obsessive-compulsive and related disorder due to pediatric acute-onset neuropsychiatric syndrome.

Acute-onset symptoms, late age at onset, or atypical symptoms suggest the need for a thorough assessment to rule out the diagnosis of obsessive-

compulsive and related disorder due to pediatric acute-onset neuropsychiatric syndrome (PANS). PANS is characterized by abrupt, dramatic onset of obsessive-compulsive symptoms or severely restricted food intake, together with a range of additional neuropsychiatric symptoms.

Specific neurological/neuromuscular, structural, or congenital disorders and conditions associated with feeding difficulties. Feeding difficulties are common in a number of congenital and neurological conditions often related to problems with oral/esophageal/pharyngeal structure and function, such as hypotonia of musculature, tongue protrusion, and unsafe swallowing. Avoidant/restrictive food intake disorder can be diagnosed in individuals with such presentations as long as all diagnostic criteria are met.

Reactive attachment disorder. Some degree of withdrawal from caregivers is characteristic of reactive attachment disorder and can lead to a disturbance in the caregiver-child relationship that can affect feeding and the child's intake. Avoidant/restrictive food intake disorder should be diagnosed concurrently only if all criteria are met for both disorders and the feeding disturbance is a primary focus for intervention.

Autism spectrum disorder. Individuals with autism spectrum disorder often present with rigid eating behaviors and heightened sensory sensitivities. However, these features do not always result in the level of impairment that would be required for a diagnosis of avoidant/restrictive food intake disorder. Avoidant/restrictive food intake disorder should be diagnosed concurrently only if all criteria are met for both disorders and when the eating disturbance requires specific treatment.

Specific phobia, social anxiety disorder, and other anxiety disorders. Specific phobia, other type, includes as an example "situations that may lead to choking or vomiting" and can represent the primary trigger for the fear, anxiety, or avoidance required for diagnosis. Distinguishing specific phobia from avoidant/restrictive food intake disorder can be difficult when a fear of choking or vomiting has resulted in food avoidance. Although avoidance or restriction of food intake secondary to a pronounced fear of choking or vomiting can be conceptualized as specific phobia, in situations when the eating problem becomes the primary focus of clinical attention, avoidant/restrictive food intake disorder becomes the appropriate diagnosis.

In social anxiety disorder, the individual may present with a fear of being observed by others while eating, which can also occur in avoidant/restrictive food intake disorder.

Anorexia nervosa. Restriction of energy intake relative to requirements leading to significantly low body weight is a core feature of anorexia nervosa. However, individuals with anorexia nervosa also display a fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain, as well as specific disturbances in

relation to perception and experience of their own body weight and shape. These features are not present in avoidant/restrictive food intake disorder, and the two disorders should not be diagnosed concurrently. Differential diagnosis between avoidant/restrictive food intake disorder and anorexia nervosa may be difficult, especially in late childhood and early adolescence, because these disorders may share a number of common symptoms (e.g., food avoidance, low weight). Differential diagnosis is also potentially difficult in individuals with anorexia nervosa who deny any fear of fatness but nonetheless engage in persistent behaviors that prevent weight gain and who do not recognize the medical seriousness of their low weight—a presentation sometimes termed “non–fat phobic anorexia nervosa.” Full consideration of symptoms, course, and family history is advised, and diagnosis may be best made in the context of a clinical relationship over time. In some individuals, avoidant/restrictive food intake disorder might precede the onset of anorexia nervosa.

Obsessive-compulsive disorder. Individuals with obsessive-compulsive disorder may present with avoidance or restriction of intake in relation to preoccupations with food or ritualized eating behavior. Avoidant/restrictive food intake disorder should be diagnosed concurrently only if all criteria are met for both disorders and when the aberrant eating is a major aspect of the clinical presentation requiring specific intervention.

Major depressive disorder. In major depressive disorder, appetite might be affected to such an extent that individuals present with significantly restricted food intake, usually in relation to overall energy intake and often

associated with weight loss. Usually appetite loss and related reduction of intake abate with resolution of mood problems. Avoidant/restrictive food intake disorder should only be used concurrently if full criteria are met for both disorders and when the eating disturbance requires specific treatment.

Schizophrenia spectrum disorders. Individuals with schizophrenia, delusional disorder, or other psychotic disorders may exhibit odd eating behaviors, avoidance of specific foods because of delusional beliefs, or other manifestations of avoidant or restrictive intake. In some cases, delusional beliefs may contribute to a concern about negative consequences of ingesting certain foods. Avoidant/restrictive food intake disorder should be used concurrently only if all criteria are met for both disorders and when the eating disturbance requires specific treatment.

Factitious disorder or factitious disorder imposed on another. Avoidant/restrictive food intake disorder should be differentiated from factitious disorder or factitious disorder imposed on another. In order to assume the sick role, some individuals with factitious disorder may intentionally describe diets that are much more restrictive than those they are actually able to consume, as well as complications of such behavior, such as a need for enteral feedings or nutritional supplements, an inability to tolerate a normal range of foods, and/or an inability to participate normally in age-appropriate situations involving food. The presentation may be impressively dramatic and engaging, and the symptoms reported inconsistently. In factitious disorder imposed on another, the caregiver describes symptoms consistent with avoidant/restrictive food intake disorder and may induce physical symptoms such as failure to gain weight. As with any diagnosis of factitious disorder imposed on another, the caregiver receives the diagnosis rather than the affected individual, and diagnosis should be made only on the basis of a careful, comprehensive assessment of the affected individual, the caregiver, and their interaction.

Developmentally normal behavior. During normal development, some toddlers and children transiently narrow the variety of foods they are willing to eat. This phenomenon, sometimes referred to as “picky eating,” usually resolves spontaneously without intervention. Avoidant/restrictive food intake disorder does not include such developmentally normal behaviors unless they become sufficiently severe to lead to failure to meet appropriate

nutritional needs or produce significant impairment in functioning (Criterion A).

Comorbidity

The most commonly observed disorders comorbid with avoidant/restrictive food intake disorder are anxiety disorders, obsessive-compulsive disorder, and neurodevelopmental disorders (specifically autism spectrum disorder, attention-deficit/hyperactivity disorder, and intellectual developmental disorder [intellectual disability]).

Anorexia Nervosa

Diagnostic Criteria

A. Restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health. *Significantly low weight* is defined as a weight that is less than minimally normal or, for children and adolescents, less than that minimally expected.

B. Intense fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight.

C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.

Coding note: The ICD-10-CM code depends on the subtype (see below).

Specify whether:

F50.01 Restricting type: During the last 3 months, the individual has not engaged in recurrent episodes of binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas). This subtype describes presentations in which weight loss is accomplished primarily through dieting, fasting, and/or excessive exercise.

F50.02 Binge-eating/purging type: During the last 3 months, the individual has engaged in recurrent episodes of binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas).

Specify if:

In partial remission: After full criteria for anorexia nervosa were previously met, Criterion A (low body weight) has not been met for a sustained period, but either Criterion B (intense fear of gaining weight or becoming fat or behavior that interferes with weight gain) or Criterion C (disturbances in self-perception of weight and shape) is still met.

In full remission: After full criteria for anorexia nervosa were previously met, none of the criteria have been met for a sustained period of time.

Specify current severity:

The minimum level of severity is based, for adults, on current body mass index (BMI) (see below) or, for children and adolescents, on BMI percentile. The ranges below are derived from World Health Organization categories for thinness in adults; for children and adolescents, corresponding BMI percentiles should be used. The level of severity may be increased to reflect clinical symptoms, the degree of functional disability, and the need for supervision.

Mild: BMI ≥ 17 kg/m^2.

Moderate: BMI 16–16.99 kg/m^2.

Severe: BMI 15–15.99 kg/m^2.

Extreme: BMI < 15 kg/m^2.

Subtypes

Most individuals with the binge-eating/purging type of anorexia nervosa who binge eat also purge through self-induced vomiting or the misuse of laxatives, diuretics, or enemas. Some individuals with this subtype of anorexia nervosa do not binge eat but do regularly purge after the consumption of small amounts of food.

Crossover between the subtypes over the course of the disorder is not uncommon; therefore, subtype description should be used to describe current symptoms rather than longitudinal course.

Diagnostic Features

There are three essential features of anorexia nervosa: persistent energy intake restriction; intense fear of gaining weight or of becoming fat, or persistent behavior that interferes with weight gain; and a disturbance in self-perceived weight or shape. The individual maintains a body weight that is below a minimally normal level for age, sex, developmental trajectory, and physical health (Criterion A). Individuals' body weights frequently meet this criterion following a significant weight loss, but among children and adolescents, there may alternatively be failure to make expected weight gain or to maintain a normal developmental trajectory (i.e., while growing in height) instead of weight loss.

Criterion A requires that the individual's weight be significantly low (i.e., less than minimally normal or, for children and adolescents, less than that minimally expected). Weight assessment can be challenging because normal weight range differs among individuals, and different thresholds have been published defining thinness or underweight status. Body mass index (BMI; calculated as weight in kilograms/height in meters^2) is a useful measure to assess body weight for height. For adults, a BMI of 18.5 kg/m^2 has been employed by the Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO) as the lower limit of normal body weight. Therefore, most adults with a BMI greater than or

equal to 18.5 kg/m^2 would not be considered to have a significantly low body weight. On the other hand, a BMI of lower than 17.0 kg/m^2 has been considered by the WHO to indicate moderate or severe thinness; therefore, an individual with a BMI less than 17.0 kg/m^2 would likely be considered to have a significantly low weight. An adult with a BMI between 17.0 and 18.5 kg/m^2, or even slightly above 18.5 kg/m^2, might be considered to have a significantly low weight if clinical history or other physiological information supports this judgment. Adults who are not underweight by population-based standards—for example, adults with BMIs of 19.0 kg/m^2 or greater—should not be assigned a diagnosis of anorexia nervosa; a diagnosis of other specified feeding or eating disorder (atypical anorexia nervosa) may be considered for such individuals.

For children and adolescents, determining a BMI-for-age percentile is useful (see, e.g., the CDC BMI percentile calculator for children and teenagers; https://www.cdc.gov/healthyweight/bmi/calculator.html). As for adults, it is not possible to provide definitive standards for judging whether a child's or an adolescent's weight is significantly low, and variations in developmental trajectories among youth limit the utility of simple numerical guidelines. The CDC has used a BMI-for-age below the 5th percentile as suggesting underweight; children and adolescents with a BMI above this benchmark may be judged to be significantly underweight in light of failure to maintain their expected growth trajectory. However, such individuals whose BMI nonetheless remains greater than the median BMI for age should not be assigned a diagnosis of anorexia nervosa; a diagnosis of other specified feeding or eating disorder (atypical anorexia nervosa) may be considered for such individuals.

Individuals with this disorder typically display an intense fear of gaining weight or of becoming fat (Criterion B). This intense fear of becoming fat is usually not alleviated by weight loss. In fact, concern about weight gain may increase even as weight falls. Younger individuals with anorexia nervosa, as well as some adults, may not recognize or

acknowledge a fear of weight gain. In the absence of another explanation for the significantly low weight, clinician inference drawn from collateral history, observational data, physical and laboratory findings, or longitudinal course either indicating a fear of weight gain or supporting persistent behaviors that prevent it may be used to establish Criterion B.

The experience and significance of body weight and shape are distorted in these individuals (Criterion C). Some individuals feel globally overweight. Others realize that they are thin but are still concerned that certain body parts, particularly the abdomen, buttocks, and thighs, are “too fat.” They may employ a variety of techniques to evaluate their body size or weight, including frequent weighing, obsessive measuring of body parts, and persistent use of a mirror to check for perceived areas of “fat.” The self-esteem of individuals with anorexia nervosa is highly dependent on their perceptions of body shape and weight. Weight loss is often viewed as an impressive achievement and a sign of extraordinary self-discipline, whereas weight gain is perceived as an unacceptable failure of self-control. Although some individuals with this disorder may acknowledge being thin, they often do not recognize the serious medical implications of their malnourished state.

Often, the individual is brought to professional attention by family members after marked weight loss (or failure to make expected weight gains) has occurred. If individuals seek help on their own, it is usually because of distress over the somatic and psychological sequelae of starvation. It is rare for an individual with anorexia nervosa to complain of weight loss per se. In fact, individuals with anorexia nervosa frequently either lack insight into or deny the problem. It is therefore often important to obtain information from family members or other sources to evaluate the history of weight loss and other features of the illness.

Associated Features

The semistarvation of anorexia nervosa, and the purging behaviors sometimes associated with it, can result in significant and potentially life-threatening medical conditions. The nutritional compromise associated with this disorder affects most major organ systems and can produce a variety of disturbances. Physiological disturbances, including amenorrhea and vital

sign abnormalities, are common. While most of the physiological disturbances associated with malnutrition are reversible with nutritional rehabilitation, some, including loss of bone mineral density, are often not completely reversible. Behaviors such as self-induced vomiting and misuse of laxatives, diuretics, and enemas may cause a number of disturbances that lead to abnormal laboratory findings; however, some individuals with anorexia nervosa exhibit no laboratory abnormalities.

When seriously underweight, many individuals with anorexia nervosa have depressive signs and symptoms such as depressed mood, social withdrawal, irritability, insomnia, and diminished interest in sex. Because these features are also observed in individuals without anorexia nervosa who are significantly undernourished, many of the depressive features may be secondary to the physiological sequelae of semistarvation, although they may also be sufficiently severe to warrant an additional diagnosis of major depressive disorder.

Obsessive-compulsive features, both related and unrelated to food, are often prominent. Most individuals with anorexia nervosa are preoccupied with thoughts of food. Some collect recipes or hoard food. Observations of behaviors associated with other forms of starvation suggest that obsessions and compulsions related to food may be exacerbated by undernutrition. When individuals with anorexia nervosa exhibit obsessions and compulsions that are not related to food, body shape, or weight, an additional diagnosis of obsessive-compulsive disorder (OCD) may be warranted.

Other features sometimes associated with anorexia nervosa include concerns about eating in public, feelings of ineffectiveness, a strong desire to control one’s environment, inflexible thinking, limited social spontaneity, and overly restrained emotional expression.

Compared with individuals with anorexia nervosa, restricting type, those with binge-eating/purging type have higher rates of impulsivity and are more likely to abuse alcohol and other drugs.

Some individuals with anorexia nervosa show excessive levels of physical activity. Increases in physical activity often precede onset of the

disorder, and over the course of the disorder increased activity accelerates weight loss. During treatment, excessive activity may be difficult to control, thereby jeopardizing weight recovery.

Individuals with anorexia nervosa may misuse medications, such as by manipulating dosage, in order to achieve weight loss or avoid weight gain. Individuals with diabetes mellitus may omit or reduce insulin doses in order to minimize carbohydrate utilization.

Prevalence

According to two U.S. epidemiological studies conducted in community samples, the 12-month prevalence of anorexia nervosa ranges from 0.0% to 0.05% with much higher rates in women than in men (0% to 0.08% in women; 0% to 0.01% in men), and the lifetime prevalence ranges from 0.60% to 0.80% (0.9% to 1.42% in women; 0.12% to 0.3% in men). By contrast, one study of adolescents found similar rates in both genders.

Anorexia nervosa appears to be most prevalent in postindustrialized, high-income countries such as in the United States, many European countries, Australia, New Zealand, and Japan. Although the prevalence of anorexia nervosa is uncertain in most low- and middle-income countries, it appears to be increasing in many countries in the Global South, including in Asia and the Middle East. Anorexia nervosa occurs across U.S. ethnoracial groups; however, its prevalence seems to be lower among Latinx and non-Latinx Black Americans than among non-Latinx Whites.

Development and Course

Anorexia nervosa commonly begins during adolescence or young adulthood. It rarely begins before puberty or after age 40, but cases of both early and late onset have been described. The onset of this disorder is often associated with a stressful life event, such as leaving home for college. The course and outcome of anorexia nervosa are highly variable. Younger individuals may manifest atypical features, including denying “fear of fat.” Older individuals more likely have a longer duration of illness, and their clinical presentation may include more signs and symptoms of long-standing disorder. Clinicians should not exclude anorexia nervosa from the differential diagnosis solely on the basis of older age.

Many individuals have a period of changed eating behavior prior to full criteria for the disorder being met. Some individuals with anorexia nervosa recover fully after a single episode, with some exhibiting a fluctuating pattern of weight gain followed by relapse, and others experiencing a chronic course over many years. Hospitalization may be required to restore weight and to address medical complications. Most individuals with anorexia nervosa experience remission within 5 years of presentation. Among individuals admitted to hospitals, overall remission rates may be lower. The crude mortality rate for anorexia nervosa is approximately 5% per decade. Death most commonly results from medical complications associated with the disorder itself or from suicide.

Risk and Prognostic Factors

Temperamental. Individuals who develop anxiety disorders or display obsessional traits in childhood are at increased risk for developing anorexia nervosa.

Environmental. Historical and cross-cultural variability in the prevalence of anorexia nervosa supports its association with cultures and settings in which thinness is valued. Occupations and avocations that encourage thinness, such as modeling and elite athletics, are also associated with increased risk.

Genetic and physiological. There is an increased risk for anorexia nervosa and for other eating and psychiatric disorders among biological relatives of individuals with anorexia nervosa. Genome-wide association studies have begun to identify specific risk loci, including loci associated with other psychiatric disorders and with metabolic traits such as insulin resistance and lipid profile. A range of brain abnormalities, many suggesting abnormal processing of reward, has been described in anorexia nervosa using functional imaging technologies such as functional magnetic resonance imaging and positron emission tomography. The degree to which these findings reflect changes associated with malnutrition versus primary abnormalities associated with the disorder is unclear.

Culture-Related Diagnostic Issues

Anorexia nervosa occurs across culturally and socially diverse populations, although available evidence suggests cross-cultural variation in its occurrence and presentation. The presentation of weight concerns among individuals with feeding and eating disorders varies substantially across cultural contexts. The absence of an expressed intense fear of weight gain, sometimes referred to as "fat phobia," appears to be relatively more common in populations in Asia, where the rationale for dietary restriction is commonly related to a more culturally sanctioned complaint such as gastrointestinal discomfort. Mental health service utilization in the United States among individuals with an eating disorder is significantly lower among underserved ethnic and racialized groups.

Diagnostic Markers

The following laboratory abnormalities may be observed in anorexia nervosa; their presence may serve to increase diagnostic confidence.

Hematology. Leukopenia is common, with the loss of all cell types but usually with apparent lymphocytosis. Mild anemia can occur, as well as thrombocytopenia and, rarely, bleeding problems.

Serum chemistry. Dehydration may be reflected by an elevated blood urea nitrogen level. Hypercholesterolemia is common. Hepatic enzyme levels may be elevated. Hypomagnesemia, hypozincemia, hypophosphatemia, and hyperamylasemia are occasionally observed. Self-induced vomiting may lead to metabolic alkalosis (elevated serum bicarbonate), hypochloremia, and hypokalemia; laxative abuse may cause a mild metabolic acidosis.

Endocrine. Serum thyroxine (T_4) levels are usually in the low-normal range; triiodothyronine (T_3) levels are decreased, while reverse T_3 levels are elevated. Females have low serum estrogen levels, whereas males have low levels of serum testosterone.

Electrocardiography. Sinus bradycardia is common, and, rarely, arrhythmias are noted. Significant prolongation of the QTc interval is observed in some individuals.

Bone mass.

Low bone mineral density, with specific areas of osteopenia or osteoporosis, is often seen. The risk of fracture is significantly elevated.

Electroencephalography. Diffuse abnormalities, reflecting a metabolic encephalopathy, may result from significant fluid and electrolyte disturbances.

Resting energy expenditure. There is often a significant reduction in resting energy expenditure.

Physical signs and symptoms. Many of the physical signs and symptoms of anorexia nervosa are attributable to starvation. Amenorrhea is commonly present and appears to be an indicator of physiological dysfunction. If present, amenorrhea is usually a consequence of the weight loss, but in a minority of individuals it may actually precede the weight loss. In prepubertal females, menarche may be delayed. In addition to amenorrhea, there may be complaints of constipation, abdominal pain, cold intolerance, lethargy, and excess energy.

The most remarkable finding on physical examination is emaciation. Commonly, there is also significant hypotension, hypothermia, and bradycardia. Some individuals develop lanugo, a fine downy body hair. Some develop peripheral edema, especially during weight restoration or upon cessation of laxative and diuretic abuse. Rarely, petechiae or ecchymoses, usually on the extremities, may indicate a bleeding diathesis. Some individuals evidence a yellowing of the skin associated with hypercarotenemia. As may be seen in individuals with bulimia nervosa, individuals with anorexia nervosa who self-induce vomiting may have hypertrophy of the salivary glands, particularly the parotid glands, as well as dental enamel erosion. Some individuals may have scars or calluses on the dorsal surface of the hand from repeated contact with the teeth while inducing vomiting.

Association With Suicidal Thoughts or Behavior

Suicide risk is elevated in anorexia nervosa, with rates reported to be 18 times greater than in an age- and gender-matched comparison group. A

systematic review found that suicide is the second leading cause of death in anorexia nervosa. Another review found that one-quarter to one-third of individuals with anorexia nervosa have suicidal ideation, and approximately 9%–25% of individuals with anorexia nervosa have attempted suicide. Likely contributors to the increased risk for suicide in those with eating disorders include greater exposure to sexual abuse; impaired decision-making; high rates of nonsuicidal self-injury, a known risk factor for suicide attempts; and comorbidity with mood disorders.

Functional Consequences of Anorexia Nervosa

Individuals with anorexia nervosa may exhibit a range of functional limitations associated with the disorder. While some individuals remain active in social and professional functioning, others demonstrate significant social isolation and/or failure to fulfill academic or career potential.

Differential Diagnosis

In addition to the general consideration of differential diagnosis for all cases, it is especially important to consider other possible causes of either significantly low body weight or significant weight loss when the presenting features are atypical (e.g., onset after age 40 years).

Medical conditions (e.g., gastrointestinal disease, hyperthyroidism, occult malignancies, and acquired immunodeficiency syndrome [AIDS]). Serious weight loss may occur in medical conditions, but individuals with these disorders usually do not also manifest a disturbance in the way their body weight or shape is experienced or an intense fear of weight gain or persist in behaviors that interfere with appropriate weight gain. Acute weight loss associated with a medical condition can occasionally be followed by the onset or recurrence of anorexia nervosa, which can initially be masked by the comorbid medical condition. Rarely, anorexia nervosa develops after bariatric surgery.

Major depressive disorder. In major depressive disorder, severe weight loss may occur, but most individuals with major depressive disorder do not have either a desire for excessive weight loss or an intense fear of gaining weight.

Schizophrenia.

Individuals with schizophrenia may exhibit odd eating behavior and occasionally experience significant weight loss, but they rarely show the fear of gaining weight and the body image disturbance required for a diagnosis of anorexia nervosa.

Substance use disorders. Individuals with substance use disorders may experience low weight because of poor nutritional intake but generally do not fear gaining weight and do not manifest body image disturbance. Individuals who abuse substances that reduce appetite (e.g., cocaine, stimulants) and who also endorse fear of weight gain should be carefully evaluated for the possibility of comorbid anorexia nervosa, given that the substance use may represent a persistent behavior that interferes with weight gain (Criterion B).

Social anxiety disorder, obsessive-compulsive disorder, and body dysmorphic disorder.
Some of the features of anorexia nervosa overlap with the criteria for social anxiety disorder, OCD, and body dysmorphic disorder. Specifically, individuals may feel humiliated or embarrassed to be seen eating in public, as in social anxiety disorder; may exhibit obsessions and compulsions related to food, as in OCD; or may be preoccupied with an imagined defect in bodily appearance, as in body dysmorphic disorder. If the individual with anorexia nervosa has social fears that are limited to eating behavior alone, the diagnosis of social anxiety disorder should not be made, but social fears unrelated to eating behavior (e.g., excessive fear of speaking in public) may warrant an additional diagnosis of social anxiety disorder. Similarly, an additional diagnosis of OCD should be considered only if the individual exhibits obsessions and compulsions unrelated to food (e.g., an excessive fear of contamination), and an additional diagnosis of body dysmorphic disorder should be considered only if the distortion is unrelated to body shape and size (e.g., preoccupation that one’s nose is too big).

Bulimia nervosa. Individuals with bulimia nervosa exhibit recurrent episodes of binge eating, engage in inappropriate behavior to avoid weight gain (e.g., self-induced vomiting), and are overly concerned with body shape and weight. However, unlike individuals with anorexia nervosa, binge-

eating/purging type, individuals with bulimia nervosa maintain body weight at or above a minimally normal level.

Avoidant/restrictive food intake disorder. Individuals with this disorder may exhibit significant weight loss or significant nutritional deficiency, but they do not have a fear of gaining weight or of becoming fat, nor do they have a disturbance in the way they experience their body shape and weight.

Comorbidity

Bipolar, depressive, and anxiety disorders commonly co-occur with anorexia nervosa. Many individuals with anorexia nervosa report the presence of either an anxiety disorder or symptoms of anxiety prior to onset of their eating disorder. OCD is described in some individuals with anorexia nervosa, especially those with the restricting type. Alcohol use disorder and other substance use disorders may also be comorbid with anorexia nervosa, especially among those with the binge-eating/purging type.

Bulimia Nervosa

Diagnostic Criteria F50.2

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
   1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances.
   2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. Recurrent inappropriate compensatory behaviors in order to prevent weight gain, such as self-induced vomiting; misuse of

laxatives, diuretics, or other medications; fasting; or excessive exercise.

C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least once a week for 3 months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. The disturbance does not occur exclusively during episodes of anorexia nervosa.

Specify if:

In partial remission: After full criteria for bulimia nervosa were previously met, some, but not all, of the criteria have been met for a sustained period of time.

In full remission: After full criteria for bulimia nervosa were previously met, none of the criteria have been met for a sustained period of time.

Specify current severity:

The minimum level of severity is based on the frequency of inappropriate compensatory behaviors (see below). The level of severity may be increased to reflect other symptoms and the degree of functional disability.

Mild: An average of 1–3 episodes of inappropriate compensatory behaviors per week.

Moderate: An average of 4–7 episodes of inappropriate compensatory behaviors per week.

Severe: An average of 8–13 episodes of inappropriate compensatory behaviors per week.

Extreme: An average of 14 or more episodes of inappropriate compensatory behaviors per week.

Diagnostic Features

There are three essential features of bulimia nervosa: recurrent episodes of binge eating (Criterion A), recurrent inappropriate compensatory behaviors to prevent weight gain (Criterion B), and self-evaluation that is unduly influenced by body shape and weight (Criterion D). To qualify for the diagnosis, the binge eating and inappropriate compensatory behaviors must occur, on average, at least once per week for 3 months (Criterion C).

An “episode of binge eating” is defined as eating, in a discrete period of time, an amount of food that is definitely larger than most individuals would eat in a similar period of time under similar circumstances (Criterion A1). The context in which the eating occurs may affect the clinician’s estimation of whether the intake is excessive. For example, a quantity of food that might be regarded as excessive for a typical meal might be considered normal during a celebration or holiday meal. A “discrete period of time” refers to a limited period, usually less than 2 hours. A single episode of binge eating need not be restricted to one setting. For example, an individual may begin a binge in a restaurant and then continue to eat on returning home. Continual snacking on small amounts of food throughout the day would not be considered an eating binge.

An occurrence of excessive food consumption must be accompanied by a sense of lack of control (Criterion A2) to be considered an episode of binge eating. An indicator of loss of control is the inability to refrain from eating or to stop eating once started. Some individuals describe a dissociative quality during, or following, the binge-eating episodes. The impairment in control associated with binge eating may not be absolute; for example, an individual may continue binge eating while the telephone is ringing but will cease if a roommate or spouse unexpectedly enters the room. Some individuals report that their binge-eating episodes are no longer characterized by an acute feeling of loss of control but rather by a more generalized pattern of uncontrolled eating. If individuals report that they have abandoned efforts to control their eating, loss of control should be considered as present. Binge eating can also be planned in some instances.

The type of food consumed during binges varies both across individuals and for a given individual. Binge eating appears to be characterized more by an abnormality in the amount of food consumed than by a craving for a specific nutrient. However, during binges, individuals tend to eat foods they would otherwise avoid.

Individuals with bulimia nervosa are typically ashamed of their eating problems and attempt to conceal their symptoms. Binge eating usually occurs in secrecy or as inconspicuously as possible. The binge eating often continues until the individual is uncomfortably, or even painfully, full. The most common antecedent of binge eating is negative affect.

Other triggers include interpersonal stressors; dietary restraint; negative feelings related to body weight, body shape, and food; and boredom. Binge eating may minimize or mitigate factors that precipitated the episode in the short-term, but negative self-evaluation and dysphoria often are the delayed consequences.

Another essential feature of bulimia nervosa is the recurrent use of inappropriate compensatory behaviors to prevent weight gain (Criterion B). Many individuals with bulimia nervosa employ several methods to compensate for binge eating. Self-induced vomiting, a type of purging behavior, is the most common inappropriate compensatory behavior. The immediate effects of vomiting include relief from physical discomfort and reduction of fear of gaining weight. In some cases, vomiting becomes a goal in itself, and the individual will binge eat in order to vomit or will vomit after eating a small amount of food. Individuals with bulimia nervosa may use a variety of methods to induce vomiting, including the use of fingers or instruments to stimulate the gag reflex. Individuals generally become adept at inducing vomiting and are eventually able to vomit at will. Rarely, individuals consume syrup of ipecac to induce vomiting. Other purging behaviors include the misuse of laxatives and diuretics and, in rare cases, the misuse of enemas following episodes of binge eating, although this is seldom the sole compensatory method employed. A number of compensatory methods other than purging may also be used in rare cases. Some individuals may take thyroid hormone in an attempt to avoid weight gain. Individuals with diabetes mellitus and bulimia nervosa may omit or reduce insulin doses in order to reduce the metabolism of food consumed during eating binges. Individuals with bulimia nervosa may fast for a day or more or exercise excessively in an attempt to prevent weight gain. Exercise may be considered excessive when it significantly interferes with important

activities, when it occurs at inappropriate times or in inappropriate settings, or when the individual continues to exercise despite injury or other medical complications.

Individuals with bulimia nervosa place an excessive emphasis on body shape or weight in their self-evaluation, and these factors are typically extremely important in determining self-esteem (Criterion D). Individuals with this disorder may closely resemble those with anorexia nervosa in their fear of gaining weight, in their desire to lose weight, and in the level of dissatisfaction with their bodies. However, a diagnosis of bulimia nervosa should not be given when the disturbance occurs only during episodes of anorexia nervosa (Criterion E).

Associated Features

Individuals with bulimia nervosa typically are within the normal weight or overweight range (body mass index [BMI] ≥ 18.5 and < 30 in adults). The disorder occurs but is uncommon among obese individuals. Between eating binges, individuals with bulimia nervosa typically restrict their total caloric consumption and preferentially select low-calorie ("diet") foods while avoiding foods that they perceive to be fattening or likely to trigger a binge.

Menstrual irregularity or amenorrhea often occurs among females with bulimia nervosa; it is uncertain whether such disturbances are related to weight fluctuations, to nutritional deficiencies, or to emotional distress. The fluid and electrolyte disturbances resulting from the purging behavior are sometimes sufficiently severe to constitute medically serious problems. Rare but potentially fatal complications include esophageal tears, gastric rupture, and cardiac arrhythmias. Serious cardiac and skeletal myopathies have been reported among individuals following repeated use of syrup of ipecac to induce vomiting. Individuals who chronically abuse laxatives may become dependent on their use to stimulate bowel movements. Gastrointestinal symptoms are commonly associated with bulimia nervosa, and rectal prolapse has also been reported among individuals with this disorder.

Prevalence

According to two U.S. epidemiological studies conducted in adult community samples, the 12-month prevalence of bulimia nervosa ranges from 0.14% to 0.3%, with much higher

rates in women than in men (0.22% to 0.5% in women; 0.05% to 0.1% in men), and the lifetime prevalence ranges from 0.28% to 1.0% (0.46% to 1.5% in women; 0.05% to 0.08% in men). In one study of adolescents ages 13–18, lifetime prevalence rates were 1.3% and 0.5% in girls and boys, respectively.

In the United States, the prevalence of bulimia nervosa is similar across U.S. ethnoracial groups. The reported prevalence of bulimia nervosa is highest in populations residing in high-income industrialized countries, such as the United States, Canada, Australia, New Zealand, and many European countries; in most of these countries the prevalence of bulimia nervosa is roughly comparable.

The prevalence of bulimia nervosa in some regions of Latin America and the Middle East is similar to the prevalence in most high-income countries. The prevalence of bulimia nervosa appears to be gradually increasing in many low- and middle-income countries.

Development and Course

Bulimia nervosa commonly begins in adolescence or young adulthood. Onset before puberty or after age 40 is uncommon. The binge eating frequently begins during or after an episode of dieting to lose weight. Experiencing multiple stressful life events also can precipitate onset of bulimia nervosa.

Disturbed eating behavior persists for at least several years in a high percentage of clinical samples. The course may be chronic or intermittent, with periods of remission alternating with recurrences of binge eating. However, over longer-term follow-up, the symptoms of many individuals appear to diminish with or without treatment, although treatment clearly impacts outcome. Periods of remission longer than 1 year are associated with better long-term outcome.

Significantly elevated risk for mortality (all-cause and suicide) has been reported for individuals with bulimia nervosa. The crude mortality rate (ratio of the number of deaths during the year to the average population in that year) for bulimia nervosa is nearly 2% per decade.

Diagnostic crossover from initial bulimia nervosa to anorexia nervosa occurs in a minority of cases (10%–15%). Individuals who do experience crossover to anorexia nervosa commonly will revert back to bulimia nervosa or have multiple occurrences of crossovers between these disorders. A subset of individuals with bulimia nervosa continue to binge eat but no longer engage in inappropriate compensatory behaviors, and therefore their symptoms meet criteria for binge-eating disorder or other specified eating disorder. Diagnosis should be based on the current (i.e., past 3 months) clinical presentation.

Risk and Prognostic Factors

Temperamental. Weight concerns, low self-esteem, depressive symptoms, social anxiety disorder, and childhood generalized anxiety disorder are associated with increased risk for the development of bulimia nervosa.

Environmental. Internalization of a thin body ideal has been found to increase risk for developing weight concerns, which in turn increases risk for the development of bulimia nervosa. Individuals who experienced childhood sexual or physical abuse are at increased risk for developing bulimia nervosa.

Genetic and physiological. Childhood obesity and early pubertal maturation increase risk for bulimia nervosa. Familial transmission of bulimia nervosa may be present, as well as genetic vulnerabilities for the disorder.

Course modifiers. Severity of psychiatric comorbidity predicts worse long-term outcome of bulimia nervosa.

Culture-Related Diagnostic Issues

Although data show that community-based prevalence of bulimia nervosa does not differ significantly across U.S. ethnoracial groups, treatment

utilization for bulimia nervosa is lower among underserved U.S. ethnic and racialized groups than among the non-Latinx White population.

Sex- and Gender-Related Diagnostic Issues

Bulimia nervosa is much more common in girls and women than in boys and men. Boys and men are especially underrepresented in treatment-seeking samples, for reasons that have not yet been systematically examined.

Diagnostic Markers

No specific diagnostic test for bulimia nervosa currently exists. However, several laboratory abnormalities may occur as a consequence of purging and may increase diagnostic certainty. These include fluid and electrolyte abnormalities, such as hypokalemia (which can provoke cardiac arrhythmias), hypochloremia, and hyponatremia. The loss of gastric acid through vomiting may produce a metabolic alkalosis (elevated serum bicarbonate), and the frequent induction of diarrhea or dehydration through laxative and diuretic abuse can cause metabolic acidosis. Some individuals with bulimia nervosa exhibit mildly elevated levels of serum amylase, probably reflecting an increase in the salivary isoenzyme.

Physical examination usually yields no physical findings. However, inspection of the mouth may reveal significant and permanent loss of dental enamel, especially from lingual surfaces of the front teeth because of recurrent vomiting. These teeth may become chipped and appear ragged and “moth-eaten.” There may also be an increased frequency of dental caries. In some individuals, the salivary glands, particularly the parotid glands, may become notably enlarged. Individuals who induce vomiting by manually stimulating the gag reflex may develop calluses or scars on the dorsal surface of the hand from repeated contact with the teeth. Serious cardiac and skeletal myopathies have been reported among individuals following repeated use of syrup of ipecac to induce vomiting.

Association With Suicidal Thoughts or Behavior

Suicide risk is elevated in bulimia nervosa. A review found that approximately one-quarter to one-third of individuals with bulimia nervosa have had suicidal ideation, and a similar proportion have attempted suicide.

Functional Consequences of Bulimia Nervosa

Individuals with bulimia nervosa may exhibit a range of functional limitations associated with the disorder and have reduced health-related quality of life. A minority of individuals report severe role impairment, with the social-life domain most likely to be adversely affected by bulimia nervosa.

Differential Diagnosis

Anorexia nervosa, binge-eating/purging type. Individuals whose binge-eating behavior occurs only during episodes of anorexia nervosa are given the diagnosis anorexia nervosa, binge-eating/purging type, and should not be given the additional diagnosis of bulimia nervosa. For individuals with an initial diagnosis of anorexia nervosa who binge and purge but whose presentation no longer meets the full criteria for anorexia nervosa, binge-eating/purging type (e.g., when weight is normal), a diagnosis of bulimia nervosa should be given only when all criteria for bulimia nervosa have been met for at least 3 months.

Binge-eating disorder. Some individuals binge eat but do not engage in regular inappropriate compensatory behaviors. In these cases, the diagnosis of binge-eating disorder should be considered.

Kleine-Levin syndrome. In certain neurological or other medical conditions, such as Kleine-Levin syndrome, there is disturbed eating behavior, but the characteristic psychological features of bulimia nervosa, such as overconcern with body shape and weight, are not present.

Major depressive disorder, with atypical features. Overeating is common in major depressive disorder, with atypical features, but individuals with this disorder do not engage in inappropriate compensatory behaviors and do not exhibit the excessive concern with body shape and weight characteristic of

bulimia nervosa. If criteria for both disorders are met, both diagnoses should be given.

Borderline personality disorder. Binge-eating behavior is included in the impulsive behavior criterion that is part of the definition of borderline personality disorder. If the criteria for both borderline personality disorder and bulimia nervosa are met, both diagnoses should be given.

Comorbidity

Comorbidity with mental disorders is common in individuals with bulimia nervosa, with most experiencing at least one other mental disorder and many experiencing multiple comorbidities. Comorbidity is not limited to any particular subset but rather occurs across a wide range of mental disorders. There is an increased frequency of depressive symptoms (e.g., low self-esteem) and bipolar and depressive disorders (particularly depressive disorders) in individuals with bulimia nervosa. In many individuals, the mood disturbance begins at the same time as or following the development of bulimia nervosa, and individuals often ascribe their mood disturbances to the bulimia nervosa. However, in some individuals, the mood disturbance clearly precedes the development of bulimia nervosa. There may also be an increased frequency of anxiety symptoms (e.g., fear of social situations) or anxiety disorders. These mood and anxiety disturbances frequently remit following effective treatment of the bulimia nervosa. The lifetime prevalence of substance use disorder, particularly alcohol use disorder or stimulant use disorder, is at least 30% among individuals with bulimia nervosa. Stimulant use often begins in an attempt to control appetite and weight. A substantial percentage of individuals with bulimia nervosa also have personality features that meet criteria for one or more personality disorders, most frequently borderline personality disorder.

Binge-Eating Disorder

Diagnostic Criteria	**F50.81**

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

   1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances.
   2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. The binge-eating episodes are associated with three (or more) of the following:

   1. Eating much more rapidly than normal.
   2. Eating until feeling uncomfortably full.

   3. Eating large amounts of food when not feeling physically hungry.
   4. Eating alone because of feeling embarrassed by how much one is eating.
   5. Feeling disgusted with oneself, depressed, or very guilty afterward.

C. Marked distress regarding binge eating is present.

D. The binge eating occurs, on average, at least once a week for 3 months.

E. The binge eating is not associated with the recurrent use of inappropriate compensatory behavior as in bulimia nervosa and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.

Specify if:

In partial remission: After full criteria for binge-eating disorder were previously met, binge eating occurs at an

average frequency of less than one episode per week for a sustained period of time.

In full remission: After full criteria for binge-eating disorder were previously met, none of the criteria have been met for a sustained period of time.

Specify current severity:

The minimum level of severity is based on the frequency of episodes of binge eating (see below). The level of severity may be increased to reflect other symptoms and the degree of functional disability.

Mild: 1–3 binge-eating episodes per week.

Moderate: 4–7 binge-eating episodes per week.

Severe: 8–13 binge-eating episodes per week.

Extreme: 14 or more binge-eating episodes per week.

Diagnostic Features

The essential feature of binge-eating disorder is recurrent episodes of binge eating that must occur, on average, at least once per week for 3 months (Criterion D). An "episode of binge eating" is defined as eating, in a discrete period of time, an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances (Criterion A1). The context in which the eating occurs may affect the clinician's estimation of whether the intake is excessive. For example, a quantity of food that might be regarded as excessive for a typical meal might be considered normal during a celebration or holiday meal. A "discrete period of time" refers to a limited period, usually less than 2 hours. A single episode of binge eating need not be restricted to one setting. For example, an individual may begin a binge in a restaurant and then continue to eat on returning home. Continual snacking on small amounts of food throughout the day would not be considered an eating binge.

An occurrence of excessive food consumption must be accompanied by a sense of lack of control (Criterion A2) to be considered an episode of binge eating. An indicator of loss of control is the inability to refrain from

eating or to stop eating once started. Some individuals describe a dissociative quality during, or following, the binge-eating episodes. The impairment in control associated with binge eating may not be absolute; for example, an individual may continue binge eating while the telephone is ringing but will cease if a roommate or spouse unexpectedly enters the room. Some individuals report that their binge-eating episodes are no longer characterized by an acute feeling of loss of control but rather by a more generalized pattern of uncontrolled eating. If individuals report that they have abandoned efforts to control their eating, loss of control may still be considered as present. Binge eating can also be planned in some instances.

The type of food consumed during binges varies both across individuals and for a given individual. Binge eating appears to be characterized more by an abnormality in the amount of food consumed than by a craving for a specific nutrient.

Binge eating must be characterized by marked distress (Criterion C) and at least three of the following features: eating much more rapidly than normal; eating until feeling uncomfortably full; eating large amounts of food when not feeling physically hungry; eating alone because of feeling embarrassed by how much one is eating; and feeling disgusted with oneself, depressed, or very guilty afterward (Criterion B).

Individuals with binge-eating disorder are typically ashamed of their eating problems and attempt to conceal their symptoms. Binge eating usually occurs in secrecy or as inconspicuously as possible. The most common antecedent of binge eating is negative affect. Other triggers include interpersonal stressors; dietary restraint; negative feelings related to body weight, body shape, and food; and boredom. Binge eating may minimize or mitigate factors that precipitated the episode in the short-term, but negative self-evaluation and dysphoria often are the delayed consequences.

Associated Features

Binge-eating disorder occurs in normal-weight/overweight and obese individuals. It is reliably associated with overweight and obesity in treatment-seeking individuals. Nevertheless, binge-eating disorder is distinct from obesity. Most obese individuals do not engage in recurrent binge eating. In addition, compared with weight-matched obese individuals without binge-eating disorder, those with the disorder consume more calories in laboratory studies of eating behavior and have greater functional impairment, lower quality of life, more subjective distress, and greater psychiatric comorbidity.

Prevalence

According to two U.S. epidemiological studies conducted in community samples, the 12-month prevalence of binge-eating disorder ranges from 0.44% to 1.2%, with rates two to three times higher in women than in men (0.6% to 1.6% in women; 0.26% to 0.8% in men), and the lifetime prevalence ranges from 0.85% to 2.8% (1.25% to 3.5% in women; 0.42% to 2.0% in men).

In the United States, the prevalence of binge-eating disorder appears comparable across ethnoracial groups.

Binge-eating disorder has a roughly similar prevalence in most high-income industrialized countries, including the United States, Canada, many European countries, Australia, and New Zealand, with 12-month prevalence in high-income countries ranging from 0.1% to 1.2%. Although fewer data are available from populations in low- and middle-income countries, binge-eating disorder prevalence in some regions of Latin America appears to be at least as high as in the United States and Europe. Mexican Americans in the United States have a higher prevalence of binge-eating disorder than their counterparts in Mexico.

Development and Course

Little is known about the development of binge-eating disorder. Both binge eating and loss-of-control eating without objectively excessive consumption occur in children and are associated with increased body fat, weight gain, and increases in psychological symptoms. Binge eating is common in adolescent and college-age samples. Loss-of-control eating or episodic

binge eating may represent a prodromal phase of eating disorders for some individuals.

Dieting follows the development of binge eating in many individuals with binge-eating disorder. (This is in contrast to bulimia nervosa, in which dysfunctional dieting usually precedes the onset of binge eating.) Binge-eating disorder typically begins in adolescence or young adulthood but can begin in later adulthood. Individuals with binge-eating disorder who seek treatment usually are older than individuals with either bulimia nervosa or anorexia nervosa who seek treatment.

Remission rates in both natural course and treatment outcome studies are higher for binge-eating disorder than for bulimia nervosa or anorexia nervosa. The course of binge-eating disorder is variable and as yet incompletely understood, with at least some affected individuals showing a relatively persistent, at times relapsing and remitting, symptom trajectory comparable to that of bulimia nervosa in terms of severity and duration. Crossover from binge-eating disorder to other eating disorders is uncommon.

Risk and Prognostic Factors

Genetic and physiological. Binge-eating disorder appears to run in families, which may reflect additive genetic influences.

Culture-Related Diagnostic Issues

Clinical presentations of binge-eating disorder differ across ethnoracial groups in the United States. Black individuals may report fewer symptoms of distress associated with binge eating and present for treatment with higher frequency of binge eating compared with White individuals.

Association With Suicidal Thoughts or Behavior

Suicidal ideation has been reported to occur in approximately 25% of individuals with binge-eating disorder.

Functional Consequences of Binge-Eating Disorder

Binge-eating disorder is associated with a range of functional consequences, including social role adjustment problems, impaired health-related quality of life and life satisfaction, increased medical morbidity and mortality, and associated increased health care utilization compared with body mass index (BMI)–matched control subjects. It may also be associated with an increased risk for weight gain and the development of obesity.

Differential Diagnosis

Bulimia nervosa. Binge-eating disorder has recurrent binge eating in common with bulimia nervosa but differs from the latter disorder in some fundamental respects. In terms of clinical presentation, the recurrent inappropriate compensatory behavior (e.g., purging, driven exercise) seen in bulimia nervosa is absent in binge-eating disorder. Unlike individuals with bulimia nervosa, individuals with binge-eating disorder typically do not show marked or sustained dietary restriction designed to influence body weight and shape between binge-eating episodes. They may, however, report frequent attempts at dieting. Binge-eating disorder also differs from bulimia nervosa in terms of response to treatment. Rates of improvement are consistently higher among individuals with binge-eating disorder than among those with bulimia nervosa.

Obesity. Binge-eating disorder is associated with overweight and obesity but has several key features that are distinct from obesity. First, levels of overvaluation of body weight and shape are higher in obese individuals with the disorder than in those without the disorder. Second, rates of psychiatric comorbidity are significantly higher among obese individuals with the disorder compared with those without the disorder. Third, the outcome of evidence-based psychological treatments for binge-eating disorder is more often successful than the treatment of obesity in individuals with comorbid obesity and binge-eating disorder.

Bipolar and depressive disorders. Increases in appetite and weight gain are included in the criteria for major depressive episode and in the atypical features specifiers for

depressive and bipolar disorders. Increased eating in the context of a major depressive episode may or may not be associated with loss of control. If the full criteria for both disorders are met, both diagnoses can be given. Binge eating and other symptoms of disordered eating are seen in association with bipolar disorder. If the full criteria for both disorders are met, both diagnoses should be given.

Borderline personality disorder. Binge eating is included in the impulsive behavior criterion that is part of the definition of borderline personality disorder. If the full criteria for both disorders are met, both diagnoses should be given.

Comorbidity

Binge-eating disorder is associated with significant psychiatric comorbidity that is comparable to that of bulimia nervosa and anorexia nervosa. The most common comorbid disorders are major depressive disorder and alcohol use disorder. The psychiatric comorbidity is linked to the severity of binge eating and not to the degree of obesity.

Other Specified Feeding or Eating Disorder

F50.89

This category applies to presentations in which symptoms characteristic of a feeding and eating disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the feeding and eating disorders diagnostic class. The other specified feeding or eating disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific feeding and eating disorder. This is done by recording “other specified

feeding or eating disorder" followed by the specific reason (e.g., "bulimia nervosa of low frequency").

Examples of presentations that can be specified using the "other specified" designation include the following:

1. **Atypical anorexia nervosa:** All of the criteria for anorexia nervosa are met, except that despite significant weight loss, the individual's weight is within or above the normal range. Individuals with atypical anorexia nervosa may experience many of the physiological complications associated with anorexia nervosa.
2. **Bulimia nervosa (of low frequency and/or limited duration):** All of the criteria for bulimia nervosa are met, except that the binge eating and inappropriate compensatory behaviors occur, on average, less than once a week and/or for less than 3 months.
3. **Binge-eating disorder (of low frequency and/or limited duration):** All of the criteria for binge-eating disorder are met, except that the binge eating occurs, on average, less than once a week and/or for less than 3 months.
4. **Purging disorder:** Recurrent purging behavior to influence weight or shape (e.g., self-induced vomiting; misuse of laxatives, diuretics, or other medications) in the absence of binge eating.
5. **Night eating syndrome:** Recurrent episodes of night eating, as manifested by eating after awakening from sleep or by excessive food consumption after the evening meal. There is awareness and recall of the eating. The night eating is not better explained by external influences such as changes in the individual's sleep-wake cycle or by local social norms. The night eating causes significant distress and/or impairment in functioning. The disordered pattern of eating is not better explained by binge-eating disorder or another mental disorder, including substance use, and is not attributable to another medical condition or to an effect of medication.

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Unspecified Feeding or Eating Disorder

F50.9

This category applies to presentations in which symptoms characteristic of a feeding and eating disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the feeding and eating disorders diagnostic class. The unspecified feeding or eating disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific feeding and eating disorder and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

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Elimination Disorders

Elimination disorders all involve the inappropriate elimination of urine or feces and are usually first diagnosed in childhood or adolescence. This group of disorders includes *enuresis,* the repeated voiding of urine into inappropriate places, and *encopresis,* the repeated passage of feces into inappropriate places. Subtypes are provided to differentiate nocturnal from diurnal (i.e., during waking hours) voiding for enuresis and the presence or absence of constipation and overflow incontinence for encopresis. Although there are minimum age requirements for diagnosis of both disorders, these are based on developmental age and not solely on chronological age. Both disorders may be voluntary or involuntary. Although these disorders typically occur separately, co-occurrence may also be observed.

Enuresis

Diagnostic Criteria F98.0

A. Repeated voiding of urine into bed or clothes, whether involuntary or intentional.
B. The behavior is clinically significant as manifested by either a frequency of at least twice a week for at least 3 consecutive months or the presence of clinically significant distress or impairment in social, academic (occupational), or other important areas of functioning.
C. Chronological age is at least 5 years (or equivalent developmental level).

D. The behavior is not attributable to the physiological effects of a substance (e.g., a diuretic, an antipsychotic medication) or another medical condition (e.g., diabetes, spina bifida, a seizure disorder).

Specify whether:

Nocturnal only: Passage of urine only during nighttime sleep.

Diurnal only: Passage of urine during waking hours.

Nocturnal and diurnal: A combination of the two subtypes above.

Subtypes

The nocturnal-only subtype of enuresis, sometimes referred to as *monosymptomatic enuresis,* is the most common subtype and involves incontinence only during nighttime sleep, typically during the first one-third of the night. The diurnal-only subtype occurs in the absence of nocturnal enuresis and may be referred to simply as *urinary incontinence.* Individuals with this subtype can be divided into two groups. Individuals with "urge incontinence" have sudden urge symptoms and detrusor instability, whereas individuals with "voiding postponement" consciously defer micturition urges until incontinence results. The nocturnal-and-diurnal subtype is also known as *nonmonosymptomatic enuresis.*

Diagnostic Features

The essential feature of enuresis is repeated voiding of urine during the day or at night into bed or clothes (Criterion A). Most often the voiding is involuntary, but occasionally it may be intentional. To qualify for a diagnosis of enuresis, the voiding of urine must occur at least twice a week for at least 3 consecutive months or must cause clinically significant distress or impairment in social, academic (occupational), or other important areas of functioning (Criterion B). The individual must have

reached an age at which continence is expected (i.e., a chronological age of at least 5 years or, for children with developmental delays, a mental age of at least 5 years) (Criterion C). The urinary incontinence is not attributable to the physiological effects of a substance (e.g., a diuretic, an antipsychotic medication) or another medical condition (e.g., diabetes, spina bifida, ectopic ureter in a female, posterior urethral valves in a male, tethered cord, a seizure disorder) (Criterion D).

Associated Features

During nocturnal enuresis, occasionally the voiding takes place during rapid eye movement (REM) sleep, and the child may recall a dream that involved the act of urinating. During daytime (diurnal) enuresis, the child defers voiding until incontinence occurs, sometimes because of a reluctance to use the toilet as a result of social anxiety or a preoccupation with school or play activity. The enuretic events most commonly occur in the early afternoon on school days or after returning from school. Children with executive functioning problems and other neurological problems that may be associated with symptoms of disruptive behavior may be at high risk for urinary incontinence without sensory awareness. It is not uncommon for children with daytime urinary incontinence and the nocturnal-and-diurnal subtype of enuresis to have persistence of incontinence after appropriate treatment of an associated infection.

Prevalence

The prevalence of daytime incontinence ranges from 3.2% to 9.0% in children age 7 years, from 1.1% to 4.2% in youth ages 11–13 years, and from 1.2% to 3.0% in adolescents ages 15–17 years.

The prevalence of nocturnal enuresis in the community decreases with age; in several geographical settings, including the United States, the Netherlands, and Hong Kong, the range is around 5%–10% among 5-year-olds, 3%–5% among 10-year-olds, and around 1% among individuals 15 years or older. Boys and members of socially oppressed groups may have higher prevalence as found in African American children in the United States and Turkish or Moroccan children in the Netherlands. The disorder

may also have higher prevalence in youth with learning disabilities or attention-deficit/hyperactivity disorder.

Development and Course

Enuresis can follow two courses: a “primary” type, in which the individual has never established urinary continence, and a “secondary” type, in which the disturbance develops after a period of established urinary continence. There are no differences in prevalence of comorbid mental disorders between the two types. By definition, primary enuresis begins at age 5 years. The most common time for the onset of secondary enuresis is between ages 5 and 8 years, but it may occur at any time. After age 5 years, the rate of spontaneous remission is 5%–10% per year. Most children with the disorder become continent by adolescence, but in approximately 1% of cases the disorder continues into adulthood. Diurnal enuresis is uncommon after age 9 years. While occasional diurnal incontinence is not uncommon in middle childhood, it is substantially more common in those who also have other co-occurring mental health problems, including cognitive and behavioral problems. When enuresis persists into late childhood or adolescence, the incontinence may resolve, but urinary frequency generally persists and incontinence can recur later in adulthood in women.

Risk and Prognostic Factors

A number of predisposing factors for bladder dysfunction have been suggested, including developmental delays and neuropsychiatric problems.

Environmental. Factors recognized to be associated with bladder dysfunction include delayed toileting and psychosocial stress.

Genetic and physiological. Nocturnal enuresis has been associated with a mismatch between nocturnal urine production, nocturnal bladder storage capacity, and the ability to arouse from sleep. Underlying these mechanisms are possibly disorders of central nervous system signal processing and the default mode network. The increased arousal thresholds do not, however, mean that these children sleep well; in fact, sleep quality of enuretic

children is often poor. Nocturnal enuresis is a genetically heterogeneous disorder. Heritability has been shown in family, twin, and segregation analyses. Risk for childhood nocturnal enuresis is approximately 3.6 times higher in offspring of enuretic mothers and 10.1 times higher in the presence of paternal urinary incontinence. The risk magnitudes for nocturnal enuresis and diurnal incontinence are similar.

Culture-Related Diagnostic Issues

Enuresis has been reported in a variety of European, African, and Asian countries as well as in the United States. At a national level, prevalence rates are remarkably similar, and there is great similarity in the developmental trajectories found in different countries. Local school-based surveys, however, show wide prevalence variation of nocturnal enuresis across settings in Africa, South Asia, Europe, and the Caribbean (4%–50%), at least in part due to methodological variation. The very high rates of enuresis in orphanages and other residential institutions are not explained by the mode or early timing of toilet training.

Cultural contexts affect both the diagnosis and the perceived etiology of enuresis. For example, traditional Chinese medicine attributes enuresis to long-term kidney yang (masculine energy) deficiency. Heightened impact on parents of children's enuresis has been reported in societies with economic limitations in obtaining care for the child or in the context of social policies that restrict the number of children (e.g., China's one-child policy), possibly leading to higher risk of parental emotional disorders.

Sex- and Gender-Related Diagnostic Issues

Nocturnal enuresis is more common in males than in females (almost 2:1). This male preponderance is particularly true in younger age groups, cases with milder severity, and cases involving enuresis occurring only at night. Urinary tract infections are frequently associated with daytime wetting, especially in females. Diurnal incontinence is more common in females than in males, and the ratio increases with age. The relative risk of having a child who develops enuresis is greater for previously enuretic fathers than for previously enuretic mothers.

Functional Consequences of Enuresis

The amount of impairment associated with enuresis is a function of the limitation on the child's social activities (e.g., ineligibility for sleep-away camp) or its effect on the child's self-esteem, the degree of social ostracism by peers, and the anger, punishment, and rejection on the part of caregivers.

Differential Diagnosis

Neurogenic bladder or another medical condition. The diagnosis of enuresis is not made in the presence of a neurogenic bladder or any other structural condition (such as a posterior urethral valve or ectopic ureter) or another medical condition that causes polyuria or

urgency (e.g., untreated diabetes mellitus or diabetes insipidus) or during an acute urinary tract infection. However, a diagnosis is compatible with such medical conditions if urinary incontinence was regularly present prior to the development of another medical condition or if it persists after the institution of appropriate treatment of the medical condition.

Medication side effects. Enuresis may occur during treatment with antipsychotic medications, diuretics, or other medications that may induce constipation, polyuria, or alterations in executive functioning, all of which may lead to incontinence. In this case, the diagnosis should not be made in isolation but may be noted as a medication side effect. However, a diagnosis of enuresis may be made if urinary incontinence was regularly present prior to treatment with the medication.

Comorbidity

Although most children with enuresis do not have a comorbid mental disorder, the prevalence of comorbid behavioral and developmental symptoms is higher in children with both diurnal and nocturnal enuresis than in children without incontinence. Developmental delays, including speech, language, learning, and motor skills delays, are also present in a portion of children with enuresis. Encopresis and constipation are present in

both day and night incontinence. Restless legs syndrome and parasomnias such as non–rapid eye movement sleep arousal disorders (sleepwalking and sleep terror types) are associated with nocturnal enuresis. Additionally, there is a link between nocturnal enuresis and heavy snoring or sleep apneas. Approximately 50% of enuretic children with proven sleep-disordered breathing will become dry by undergoing adenotonsillectomy. Urinary tract infections are more common in children with daytime urinary incontinence and nonmonosymptomatic nocturnal enuresis, especially the diurnal subtype, than in those who are continent.

Encopresis

Diagnostic Criteria F98.1

A. Repeated passage of feces into inappropriate places (e.g., clothing, floor), whether involuntary or intentional.
B. At least one such event occurs each month for at least 3 months.
C. Chronological age is at least 4 years (or equivalent developmental level).
D. The behavior is not attributable to the physiological effects of a substance (e.g., laxatives) or another medical condition except through a mechanism involving constipation.

Specify whether:

With constipation and overflow incontinence: There is evidence of constipation on physical examination or by history.

Without constipation and overflow incontinence: There is no evidence of constipation on physical examination or by history.

Subtypes

Feces in encopresis, “with constipation and overflow incontinence” subtype, are characteristically (but not invariably) poorly formed, and leakage can be infrequent to continuous, occurring throughout the day and at times during sleep. Only part of the feces is passed during toileting, and the incontinence resolves after treatment of the constipation.

In the “without constipation and overflow incontinence” subtype, feces are likely to be of normal form and consistency, and soiling is intermittent. Feces may be deposited in a

prominent location. This is usually associated with the presence of oppositional defiant disorder or conduct disorder or may be the consequence of anal masturbation. Soiling without constipation is less common than soiling with constipation.

Diagnostic Features

The essential feature of encopresis is repeated passage of feces into inappropriate places (e.g., clothing or floor) (Criterion A). Most often the passage is involuntary but occasionally may be intentional. The event must occur at least once a month for at least 3 months (Criterion B), and the chronological age of the child must be at least 4 years (or for children with developmental delays, the mental age must be at least 4 years) (Criterion C). The fecal incontinence must not be exclusively attributable to the physiological effects of a substance (e.g., laxatives) or another medical condition except through a mechanism involving constipation (Criterion D).

When the passage of feces is involuntary rather than intentional, it is often related to constipation, impaction, and retention with subsequent overflow. The constipation may develop for psychological reasons (e.g., anxiety about defecating in a particular place, a more general pattern of anxious or oppositional behavior), leading to avoidance of defecation and excessive volitional stool retention. Physiological predispositions to constipation include ineffectual straining or paradoxical defecation dynamics, with contraction rather than relaxation of the external sphincter or pelvic floor during straining for defecation. Dietary habits (such as

insufficient fluid intake), celiac disease, hypothyroidism, or a medication side effect can also induce constipation. Once constipation has developed, it may be complicated by an anal fissure, painful defecation, and further fecal retention. The consistency of the stool may vary. In some individuals the stool may be of normal or near-normal consistency. In other individuals—such as those with overflow incontinence secondary to fecal retention—it may be liquid.

Associated Features

The child with encopresis often feels ashamed and may wish to avoid situations (e.g., camp, school) that might lead to embarrassment. The amount of impairment is a function of the effect on the child's self-esteem, the degree of social ostracism by peers, and the anger, punishment, and rejection on the part of caregivers. Smearing feces may be deliberate or accidental, resulting from the child’s attempt to clean or hide feces that were passed involuntarily. When the incontinence is clearly deliberate, features of oppositional defiant disorder or conduct disorder may also be present. Many children with encopresis and chronic constipation also have enuresis symptoms and may have associated urinary reflux in the bladder or ureters that may lead to chronic urinary infections, the symptoms of which may remit with treatment of the constipation.

Prevalence

It is estimated that the majority of children older than 4 years with encopresis have the subtype “with constipation and overflow incontinence.” Encopresis affects 1%–4% of children in high-income countries, while in some Asian countries (Iran, South Korea, Sri Lanka) a prevalence of 2%–8% has been reported. Encopresis is more prevalent among children ages 4–6 years (> 4%) than among children ages 10–12 years (< 2%); prevalence is also higher among children who experience early abuse or neglect and low-income youth.

Development and Course

Encopresis is not diagnosed until a child has reached a chronological age of at least 4 years (or for children with developmental delays, a mental age of at least 4 years). Inadequate,

inconsistent toilet training and psychosocial stress (e.g., entering school, the birth of a sibling) may be predisposing factors. Two types of course have been described: a “primary” type, in which the individual has never established fecal continence, and a “secondary” type, in which the disturbance develops after a period of established fecal continence. Encopresis can persist, with intermittent exacerbations, for years.

Risk and Prognostic Factors

Painful defecation can lead to constipation and a cycle of withholding behaviors that make encopresis more likely. Male gender and age prior to adolescence are risk factors for encopresis. A number of factors are thought to contribute to the development of fecal incontinence, including anxiety, depression, behavioral disorders, psychological stressors (e.g., bullying, poor school performance), and lower socioeconomic status.

Culture-Related Diagnostic Issues

Differences in food and beverage intake in different cultures, hot climatic conditions in tropical countries, and psychosocial adversity may influence the incidence of constipation, unexplained abdominal pain, and fecal retention that lead to encopresis. Parents in some societies may not seek health services for encopresis because of sociocultural reasons. For example, Turkish and Moroccan parents in the Netherlands may not report encopresis because of religious concerns about the impurity of urine and feces.

Sex- and Gender-Related Diagnostic Issues

In children younger than 5 years, the gender ratio appears to be equal, but encopresis tends to be more common in boys than in girls among older

children, with a ratio that varies globally (in community and hospital-based studies) from 2:1 (in the United States) to 6:1.

Diagnostic Markers

The diagnosis of encopresis is a clinical diagnosis based on history and physical examination and generally does not require any diagnostic testing. Detection of a rectal fecal impaction by digital rectal examination would support the diagnosis of encopresis, with constipation and overflow incontinence. Although not indicated for the diagnosis of encopresis, an abdominal radiograph demonstrating a fecal impaction would also support the diagnosis of encopresis, with constipation and overflow incontinence. Colonic transit testing, which typically involves ingestion of radiopaque markers followed by abdominal imaging to evaluate colonic transit time, can help differentiate between encopresis with or without constipation and overflow incontinence. Abdominal imaging demonstrating retention of radiopaque markers would suggest encopresis, with constipation and overflow incontinence, while prompt evacuation of radiopaque markers would support the diagnosis of encopresis, without constipation and overflow incontinence. In certain children, anorectal manometry testing may be helpful for better understanding physiological factors that may be contributing to encopresis. Anorectal manometry allows for evaluation of anorectal function and sensation. In the child with refractory symptoms or signs suggesting the presence of an underlying medical condition leading to fecal incontinence, further evaluation may be indicated. Such evaluation is designed to exclude other medical conditions.

Functional Consequences of Encopresis

Encopresis is associated with a significant decrease in health-related quality of life and family functioning, particularly in older children.

Differential Diagnosis

A diagnosis of encopresis in the presence of another medical condition is appropriate only if the mechanism involves constipation that cannot be

explained by other medical conditions. Fecal incontinence related to other medical conditions (e.g., chronic diarrhea, spina bifida, anal stenosis) would not warrant a DSM-5 diagnosis of encopresis.

Comorbidity

Enuresis is often present in children with encopresis, particularly in children with encopresis, without constipation and overflow incontinence.

Other Specified Elimination Disorder

This category applies to presentations in which symptoms characteristic of an elimination disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the elimination disorders diagnostic class. The other specified elimination disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific elimination disorder. This is done by recording “other specified elimination disorder” followed by the specific reason (e.g., “low-frequency enuresis”).

Coding note: Code **N39.498** for other specified elimination disorder with urinary symptoms; **R15.9** for other specified elimination disorder with fecal symptoms.

Unspecified Elimination Disorder

This category applies to presentations in which symptoms characteristic of an elimination disorder that cause clinically

significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the elimination disorders diagnostic class. The unspecified elimination disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific elimination disorder and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Coding note: Code **R32** for unspecified elimination disorder with urinary symptoms; **R15.9** for unspecified elimination disorder with fecal symptoms.

Sleep-Wake Disorders

The DSM-5 classification of sleep-wake disorders is intended for use by mental health and general medical clinicians (those caring for adult, geriatric, and pediatric individuals). Sleep-wake disorders encompass 10 disorders or disorder groups: insomnia disorder, hypersomnolence disorder, narcolepsy, breathing-related sleep disorders, circadian rhythm sleep-wake disorders, non–rapid eye movement (NREM) sleep arousal disorders, nightmare disorder, rapid eye movement (REM) sleep behavior disorder, restless legs syndrome, and substance/medication-induced sleep disorder. Individuals with these disorders typically present with sleep-wake complaints of dissatisfaction regarding the quality, timing, and amount of sleep. Resulting daytime distress and impairment are core features shared by all of these sleep-wake disorders.

The organization of this chapter is designed to facilitate differential diagnosis of sleep-wake complaints and to clarify when referral to a sleep specialist is appropriate for further assessment and treatment planning. The DSM-5 sleep disorders nosology uses a simple, clinically useful approach, while also reflecting scientific advances in epidemiology, genetics, pathophysiology, assessment, and interventions research since DSM-IV. The approach taken to the classification of sleep-wake disorders in DSM-5 can be understood within the context of “lumping versus splitting.” For example, in some categories (e.g., insomnia disorder), a “lumping” approach has been adopted (i.e., three categories that were separate in DSM-IV—insomnia with other mental disorders, insomnia with other medical conditions, and insomnia with other sleep disorders—are all included in the single insomnia category as specifiers), whereas in other categories (e.g., narcolepsy), a “splitting” approach has been taken (i.e., there are four separately coded subtypes of narcolepsy, such as type 1 with cataplexy or hypocretin deficiency, and type 2 without cataplexy and either

without hypocretin deficiency or hypocretin unmeasured), reflecting the availability of validators derived from epidemiological, neurobiological, and interventions research.

Because DSM-5 is intended for use by mental health and general medical clinicians who are not experts in sleep medicine, DSM-5 presents an effort to simplify sleep-wake disorders classification and thus aggregates diagnoses under broader, less differentiated labels. In contrast, the *International Classification of Sleep Disorders*, 3rd Edition (ICSD-3), elaborates numerous diagnostic subtypes, reflects the science and opinions of the sleep specialist community, and has been prepared by and for sleep specialists.

The simpler, less-differentiated approach to the diagnosis of sleep-wake disorders in DSM-5 shows superior interrater reliability, as well as convergent, discriminant, and face validity. The text accompanying each set of diagnostic criteria provides linkages to the corresponding disorders included in ICSD-3.

The field of sleep disorders medicine has progressed in this direction since the publication of DSM-IV. The use of biological validators is now embodied in the DSM-5 classification of sleep-wake disorders, particularly for disorders of excessive sleepiness, such as narcolepsy, for which cerebrospinal fluid hypocretin-1 immunoreactivity values can be diagnostic; for breathing-related sleep disorders, for which formal sleep studies (i.e., polysomnography) are indicated; and for restless legs syndrome, which can often coexist with periodic limb movements during sleep, detectable via polysomnography.

Co-Occurring Disorders and Differential Diagnosis

Sleep disorders are often accompanied by depression, anxiety, and cognitive changes that must be addressed in treatment planning and management. Furthermore, persistent sleep disturbances (both insomnia and excessive sleepiness) are established risk factors for the subsequent development of

mental illnesses (including substance use and non–substance use disorders) and other medical conditions. They may also represent a prodromal expression of an episode of mental illness, allowing the possibility of early intervention to preempt or to attenuate a full-blown episode.

The differential diagnosis of sleep-wake complaints necessitates a multidimensional approach, with consideration of possibly coexisting clinical conditions, which are the rule and not the exception. Sleep disturbances furnish a clinically useful indicator of clinical conditions that often coexist with depression and other common mental disorders. Prominent among these comorbidities are breathing-related sleep disorders, cardiac and pulmonary conditions (e.g., congestive heart failure, chronic obstructive pulmonary disease), neurodegenerative disorders (e.g., Alzheimer's disease), and disorders of the musculoskeletal system (e.g., osteoarthritis). These disorders not only may disturb sleep but also may themselves be worsened during sleep (e.g., prolonged apneas or electrocardiographic arrhythmias during REM sleep; confusional arousals in individuals with major neurocognitive disorder; seizures in individuals with complex partial seizures). REM sleep behavior disorder is often an early indicator of neurodegenerative disorders (alpha synucleinopathies) like Parkinson's disease. For all of these reasons—related to differential diagnosis, clinical comorbidity, and facilitation of treatment planning—sleep disorders are included in DSM-5.

Key Concepts and Terms

Four distinct sleep stages can be measured by polysomnography: REM sleep and three stages of NREM sleep (N1, N2, and N3).

- REM sleep, during which the majority of typical story-like dreams occur, occupies about 20%–25% of total sleep.
- NREM sleep stage 1 (N1) is a transition from wakefulness to sleep and occupies about 5% of time spent asleep in healthy adults.
- NREM sleep stage 2 (N2), which is characterized by specific electroencephalographic waveforms (sleep spindles and K complexes), occupies about 50% of time spent asleep.

- NREM sleep stage 3 (N3) (also known as slow-wave sleep) is the deepest level of sleep and occupies about 20% of sleep time in healthy, younger adults.

These sleep stages have a characteristic temporal organization across the night. N3 tends to occur in the first one-third to one-half of the night and increases in duration in response to sleep deprivation. REM sleep occurs cyclically throughout the night, alternating with NREM sleep about every 80–100 minutes. REM sleep periods increase in duration toward the morning.

Human sleep also varies characteristically across the life span. After relative stability with large amounts of slow-wave sleep in childhood and early adolescence, sleep continuity and depth deteriorate across the adult age range. This deterioration is reflected by increased wakefulness and N1 sleep and decreased N3 sleep. Because of this, age must be considered in the diagnosis of a sleep disorder in any individual.

Polysomnography is the monitoring of multiple electrophysiological parameters during sleep. Most polysomnographic studies are conducted during the individual's usual sleeping hours—that is, at night. However, daytime polysomnographic studies also are used to quantify daytime sleepiness. The most common daytime procedure is the multiple sleep

latency test, in which the individual is instructed to lie down in a dark room and not resist falling asleep; this protocol is repeated five times during the day. The amount of time required to fall asleep (sleep latency) is measured on each trial and is used as an index of physiological sleepiness.

The following standard terminology for polysomnographic measures is used throughout the text in this chapter, and other terms provide context for the chapter discussion:

- *Sleep continuity* refers to the overall balance of sleep and wakefulness during a night of sleep. "Better" sleep continuity indicates consolidated sleep with little wakefulness or fragmentation; "worse" sleep continuity indicates disrupted sleep with more wakefulness and fragmentation.

  Specific sleep continuity measures include *sleep latency*—the amount of time required to fall asleep (expressed in minutes); *wake*

after sleep onset—the amount of awake time between initial sleep onset and final awakening (expressed in minutes); the *number of awakenings*; and *sleep efficiency*—the ratio of actual time spent asleep to time spent in bed (expressed as a percentage, with higher numbers indicating better sleep continuity).

- *Sleep architecture* refers to the amount and distribution of specific sleep stages. Sleep architecture measures include absolute amounts of REM sleep and each NREM sleep stage (in minutes), relative amount of REM sleep and NREM sleep stages (expressed as a percentage of total sleep time), and latency between sleep onset and the first REM period (*REM latency*). When the latency to onset of REM sleep is < 15 minutes, the terms *sleep-onset REM* and *sleep-onset REM period* are employed.

Association With Suicidal Thoughts or Behavior

A review of multiple studies found that the symptom of insomnia may increase the risk for suicidal thoughts, suicidal behavior, and death, even after adjustment for depression, and that nightmares increase risk for suicidal thoughts and behavior. In one study of college students, 31.3% of those with sleep problems had suicidal thoughts, and conversely, nearly all (82.7%) individuals with suicidal thoughts had sleep problems. A review and consensus statement of the American Academy of Sleep Medicine concluded that in teenagers, < 8 hours of sleep is associated with increased risk of self-harm, suicidal thoughts, and suicidal behavior.

Insomnia Disorder

Diagnostic Criteria — F51.01

A. A predominant complaint of dissatisfaction with sleep quantity or quality, associated with one (or more) of the following symptoms:

   1. Difficulty initiating sleep. (In children, this may manifest as difficulty initiating sleep without caregiver intervention.)

2. Difficulty maintaining sleep, characterized by frequent awakenings or problems returning to sleep after awakenings. (In children, this may manifest as difficulty returning to sleep without caregiver intervention.)
3. Early-morning awakening with inability to return to sleep.

B. The sleep disturbance causes clinically significant distress or impairment in social, occupational, educational, academic, behavioral, or other important areas of functioning.
C. The sleep difficulty occurs at least 3 nights per week.

D. The sleep difficulty is present for at least 3 months.
E. The sleep difficulty occurs despite adequate opportunity for sleep.
F. The insomnia is not better explained by and does not occur exclusively during the course of another sleep-wake disorder (e.g., narcolepsy, a breathing-related sleep disorder, a circadian rhythm sleep-wake disorder, a parasomnia).
G. The insomnia is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).
H. Coexisting mental disorders and medical conditions do not adequately explain the predominant complaint of insomnia.

Specify if:

With mental disorder, including substance use disorders

With medical condition

With another sleep disorder

Coding note: The code F51.01 applies to all three specifiers. Code also the relevant associated mental disorder, medical condition, or other sleep disorder immediately after the code for insomnia disorder in order to indicate the association.

Specify if:

Episodic: Symptoms last at least 1 month but less than 3 months.

Persistent: Symptoms last 3 months or longer.

Recurrent: Two (or more) episodes within the space of 1 year.

Note: Acute and short-term insomnia (i.e., symptoms lasting less than 3 months but otherwise meeting all criteria with regard to frequency, intensity, distress, and/or impairment) should be coded as an other specified insomnia disorder.

Note: The diagnosis of insomnia disorder is given whether it occurs as an independent condition or is comorbid with another mental disorder (e.g., major depressive disorder), medical condition (e.g., pain), or another sleep disorder (e.g., a breathing-related sleep disorder). For instance, insomnia may develop its own course with some anxiety and depressive features without those features meeting criteria for any one mental disorder. Insomnia may also manifest as a clinical feature of a more predominant mental disorder. Persistent insomnia is a risk factor for depression, anxiety disorders, and alcohol use disorder and is a common residual symptom after treatment for these conditions. When insomnia is comorbid with a mental disorder, treatment may need to target both conditions. Given these different courses, it is often impossible to establish the precise nature of the relationship between these clinical entities, and this relationship may change over time. Therefore, in the presence of insomnia and a comorbid disorder, it is not necessary to make a causal attribution between the two conditions. Rather, the diagnosis of insomnia disorder is made with concurrent specification of the comorbid conditions. A concurrent insomnia diagnosis should only be considered when the insomnia is sufficiently severe to warrant independent clinical attention; otherwise, no separate diagnosis is necessary.

Recording Procedures

The specifiers “with mental disorder, including substance use disorders”; “with medical condition”; and “with another sleep disorder” are available to allow the clinician to note clinically relevant comorbidities. In such cases, record F51.01 insomnia disorder, with [name of comorbid condition(s) or disorder(s)] followed by the diagnostic code(s) for the comorbid conditions or disorders (e.g., F51.01 insomnia disorder, with moderate cocaine use disorder and trigeminal neuralagia; F14.20 moderate cocaine use disorder; G50.0 trigeminal neuralgia).

Diagnostic Features

The essential feature of insomnia disorder is dissatisfaction with sleep quantity or quality with complaints of difficulty initiating or maintaining sleep. The sleep complaints are accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning. The sleep disturbance may occur during the course of another mental disorder or medical condition, or it may occur independently.

Different manifestations of insomnia can occur at different times of the sleep period. *Sleep-onset insomnia* (or *initial insomnia*) involves difficulty initiating sleep at bedtime. *Sleep maintenance insomnia* (or *middle insomnia*) involves frequent or prolonged awakenings throughout the night. *Late insomnia* involves early-morning awakening with an inability to return to sleep. Difficulty maintaining sleep is the most common single symptom of insomnia, affecting about 60% of those with insomnia, followed by early awakening and difficulty falling asleep, according to a U.S. national sample of health care plan members. A combination of these symptoms is the most common presentation overall. The specific type of sleep complaint often varies over time. Individuals who complain of difficulty falling asleep at one time may later complain of difficulty maintaining sleep, and vice versa. Symptoms of difficulty falling asleep and difficulty maintaining sleep can be quantified by the individual’s retrospective self-report, sleep diaries in which information is collected prospectively, or other methods, such as actigraphy or polysomnography. However, the diagnosis of insomnia disorder is based on the individual’s subjective perception of sleep or a

caretaker’s report. Subjective reports from individuals with insomnia disorder frequently indicate longer sleep latencies, greater time awake during the night, and less total sleep time than objective (e.g. polysomnographic) data demonstrate. The reasons for this discrepancy are not well understood, but disturbances in the underlying neurophysiology reflective of hyperarousal or cortical activation are believed to play a role.

Nonrestorative sleep, a complaint of poor sleep quality that does not leave the individual rested upon awakening despite adequate duration, is a common sleep complaint usually occurring in association with difficulty initiating or maintaining sleep, or less frequently in isolation. The precise relationship of isolated nonrestorative sleep to insomnia disorder remains unclear. The prevalence of isolated nonrestorative sleep has been estimated at about 5% and, unlike insomnia complaints, is reported more commonly in younger individuals. This complaint can also be reported in association with another sleep disorder (e.g., breathing-related sleep disorder). When a complaint of nonrestorative sleep occurs in isolation (i.e., in the absence of difficulty initiating and/or maintaining sleep or other sleep-wake disorders), a diagnosis of other specified sleep-wake disorder is made.

Aside from the frequency and duration criteria required to make the diagnosis, additional guidelines are useful to quantify insomnia severity. These quantitative guidelines, while arbitrary, are provided for illustrative purpose only. For instance, difficulty initiating sleep is defined by a subjective sleep latency > 20–30 minutes, and difficulty maintaining sleep is defined by a subjective time awake after sleep onset > 20–30 minutes. Although there is no standard definition of early-morning awakening, this symptom involves awakening at least 1 hour before the scheduled time and before total sleep time reaches 6½ hours. It is essential to take into account not only the final awakening time but also the bedtime on the previous evening. Awakening at 4:00 A.M. does not have the same clinical significance in those who go to bed at 9:00 P.M. as in those who go to bed at 11:00 P.M. Such a symptom may also reflect an age-dependent decrease in the ability to sustain sleep or an age-dependent shift in the timing of the main sleep period. Although these quantitative criteria are frequently employed in research designs, they do not in their own right reliably distinguish individuals with insomnia from normal sleepers. Moreover,

individuals whose presentations no longer meet subjective diagnostic criteria for insomnia disorder may continue to show objective disturbance by these parameters, as well as daytime impairment.

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Insomnia disorder involves daytime impairments as well as nighttime sleep difficulties. These include fatigue or, less commonly, daytime sleepiness; the latter is more common among older individuals and when insomnia is comorbid with another medical condition (e.g., chronic pain) or sleep disorder (e.g., sleep apnea). Impairment in cognitive performance may include difficulties with attention, concentration and memory, and performing complex manual skills. Associated mood disturbances are typically described as irritability or mood lability and less commonly as depressive or anxiety symptoms. Not all individuals with nighttime sleep disturbances are distressed or have functional impairment. For example, sleep continuity is often interrupted in healthy older adults who nevertheless identify themselves as good sleepers. A diagnosis of insomnia disorder should be reserved for those individuals with significant daytime distress or impairment related to their nighttime sleep difficulties.

Associated Features

Insomnia is often associated with physiological and cognitive arousal and conditioning factors that interfere with sleep. A preoccupation with sleep and distress attributable to the inability to sleep may lead to a vicious cycle: the more the individual strives to sleep, the more frustration builds and further impairs sleep. Thus, excessive attention and efforts to sleep, which override normal sleep-onset mechanisms, may contribute to the development of insomnia. Individuals with persistent insomnia may also acquire maladaptive sleep habits (e.g., spending excessive time in bed; following an erratic sleep schedule; napping) and cognitions (e.g., fear of sleeplessness; apprehensions of daytime impairments; clock monitoring) during the course of the disorder. Engaging in such activities in an environment in which the individual has frequently spent sleepless nights may further compound the conditioned arousal and perpetuate sleep

difficulties. Conversely, the individual may fall asleep more easily when not trying to do so. Some individuals also report better sleep when away from their own bedrooms and their usual routines.

Insomnia may be accompanied by a variety of daytime complaints and symptoms, including fatigue, decreased energy, and mood disturbances. Individuals with insomnia disorder may appear either fatigued or haggard or, conversely, overaroused and "wired." There may be an increased incidence of stress-related psychophysiological symptoms (e.g., tension headache, muscle tension or pain, gastrointestinal symptoms); however, there are no consistent or characteristic abnormalities on physical examination. Symptoms of anxiety or depression that do not meet criteria for a specific mental disorder may be present, as well as an excessive focus on the perceived effects of sleep loss on daytime functioning.

Individuals with insomnia may have elevated scores on self-report psychological or personality inventories with profiles indicating mild depression and anxiety, a worrisome cognitive style, an emotion-focused and internalizing style of conflict resolution, and a somatic focus. Patterns of neurocognitive impairment among individuals with insomnia disorder are inconsistent, although there may be impairments in performing tasks of higher complexity and those requiring frequent changes in performance strategy. Individuals with insomnia often require more effort to maintain cognitive performance.

Prevalence

Population-based estimates vary, depending on the sample and the criteria employed, but indicate that, across multiple countries, about one-third of adults report insomnia symptoms, 10%–15% experience associated daytime impairments, and 4%–22% have symptoms that meet criteria for insomnia disorder, with an average of about 10%. Insomnia disorder is the most prevalent of all sleep disorders. In primary care settings cross-nationally, approximately 20%–40% of individuals complain of significant insomnia symptoms. Prevalence rates for medical and psychiatric populations are significantly higher than those in the general population,

especially among individuals with mood, anxiety, and substance use disorders. Forty to fifty percent of individuals with an insomnia disorder have a comorbid mental disorder. Insomnia is a more prevalent complaint among women than among men, with a gender ratio of about 1.3:1 in multinational samples. The gender ratio rises to 1.7:1 after age 45. The prevalence in Norway among older adolescents (16–18 years) is nearly double in girls compared with boys. Although insomnia can be a symptom or an independent disorder, it is most frequently observed as a comorbid condition with another medical condition or mental disorder.

Development and Course

The onset of insomnia symptoms can occur at any time during life, but the first episode is more common in young adulthood. Less frequently, insomnia begins in childhood or adolescence. In women, the incidence of new-onset insomnia increases with menopause and may persist even after other symptoms (e.g., hot flashes) have resolved. Insomnia may have a late-life onset, which is often associated with the onset of other health-related conditions.

Insomnia can be situational, persistent, or recurrent. Situational or acute insomnia usually lasts a few days or a few weeks and is often associated with life events or rapid changes in sleep schedules or environment. It usually resolves once the initial precipitating event subsides. For some individuals, perhaps those more vulnerable to sleep disturbances, insomnia may persist long after the initial triggering event, possibly because of conditioning factors and heightened arousal. The factors that precipitate insomnia may differ from those that perpetuate it. For example, an individual who is bedridden with a painful injury and has difficulty sleeping may then develop negative associations for sleep. Conditioned arousal may then persist and lead to persistent insomnia. A similar course may develop in the context of an acute psychological stress or a mental disorder. For instance, insomnia that occurs during an episode of major depressive disorder can become a focus of attention, with consequent negative conditioning, and persist even after resolution of the depressive episode in at least 40%–50% of individuals. In some cases, insomnia may also have an insidious onset without any identifiable precipitating factor.

The course of insomnia may also be episodic, with recurrent episodes of sleep difficulties associated with the occurrence of stressful events. Chronicity rates range from 45% to 75% for follow-ups of 1–7 years. Even when the course of the insomnia has become chronic, there is night-to-night variability in sleep patterns, with an occasional restful night's sleep interspersed with several nights of poor sleep. The characteristics of insomnia may also change over time. Many individuals with insomnia have a history of "light" or easily disturbed sleep prior to onset of more persistent sleep problems.

Insomnia complaints are more prevalent among middle-age and older adults. The type of insomnia symptom changes as a function of age, with difficulties initiating sleep being more common among young adults and problems maintaining sleep occurring more frequently among middle-age and older individuals.

Difficulties initiating and maintaining sleep can also occur in children and adolescents, but there are more limited data on prevalence, risk factors, and comorbidity during these developmental phases of the life span. Sleep difficulties in childhood can result from conditioning factors (e.g., a child who does not learn to fall asleep or return to sleep without the presence of a parent) or from the absence of consistent sleep schedules and bedtime routines. Insomnia in adolescence is often triggered or exacerbated by irregular sleep schedules, especially phase delay. In both children and adolescents, psychological and medical factors can contribute to insomnia.

The increased prevalence of insomnia in older adults is partly explained by the higher incidence of physical health problems with aging. Changes in sleep patterns associated with the normal developmental process must be differentiated from those exceeding age-related changes. Older individuals may experience significant delays in sleep onset or frequent awakenings that are not associated with complaints or daytime consequences. Although

polysomnography is of limited value in the routine evaluation of insomnia, it may be more useful in the differential diagnosis among older adults because comorbid conditions associated with insomnia (e.g., sleep apnea) are more common in older individuals.

Risk and Prognostic Factors

While the risk and prognostic factors discussed in this section increase vulnerability to insomnia, sleep disturbances are more likely to occur when predisposed individuals are exposed to precipitating events, such as major life events (e.g., illness, separation) or less severe but more chronic daily stress. Most individuals resume normal sleep patterns after the initial triggering event has disappeared, but others—perhaps those more vulnerable to insomnia—continue experiencing persistent sleep difficulties. Perpetuating factors such as poor sleep habits, irregular sleep scheduling, and the fear of not sleeping feed into the insomnia problem and may contribute to a vicious cycle that may induce persistent insomnia.

Temperamental. Anxiety or worry-prone personality or cognitive styles, increased arousal predisposition, higher stress reactivity, and tendency to repress emotions can increase vulnerability to insomnia.

Environmental. Noise, light, or uncomfortably high or low temperature may increase vulnerability to insomnia. High altitude may also predispose to insomnia attributable to periodic breathing difficulties during sleep.

Genetic and physiological. Female sex and advancing age are associated with increased vulnerability to insomnia. Disrupted sleep and insomnia display a familial disposition. Thirty-five percent to seventy percent of those with insomnia disorder report one or more first-degree relatives (most commonly, the mother) with a history of insomnia. Heritability may be highest for insomnia disorder without comorbidities. The prevalence of insomnia is higher among monozygotic twins relative to dizygotic twins; it is also higher in first-degree family members compared with the general population. The extent to which this link is inherited through a genetic predisposition, learned by observations of parental models, or established as a by-product of another psychopathology remains undetermined, although sleep reactivity to stress appears to play some role.

Course modifiers. Deleterious course modifiers include poor sleep hygiene practices (e.g., excessive caffeine use, irregular sleep schedules).

Culture-Related Diagnostic Issues

Insomnia is a universal human experience. Identification of insomnia as a problem, the explanatory models for the condition, and associated help-seeking choices are affected by culture. Insomnia may be understood as a normal part of aging or of stress response, leading to low help-seeking or to coping via social support and traditional activities such as prayer. Explanatory models of insomnia vary greatly, including attributions to the effect of the environment (e.g., humidity) and bodily processes (e.g., poor blood circulation, internal heat) among others, and may be associated with nonbiomedical treatment-seeking.

Sex- and Gender-Related Diagnostic Issues

First onset in females is often associated with the birth of a new child or with menopause. Despite higher prevalence among perimenopausal and postmenopausal females, polysomnographic studies suggest better preservation of sleep continuity and slow-wave sleep in older females than in older males.

Diagnostic Markers

Polysomnography usually shows impairments of sleep continuity (e.g., increased sleep latency and time awake after sleep onset and decreased sleep efficiency [percentage of time

in bed asleep]) and may show increased stage 1 sleep and decreased stage 3 sleep. The severity of these sleep impairments does not always match the individual's clinical presentation or subjective complaint of poor sleep, as individuals with insomnia often underestimate sleep duration and overestimate wakefulness relative to polysomnography. Quantitative electroencephalographic analyses may indicate that individuals with insomnia have greater high-frequency electroencephalography power relative to good sleepers both around the sleep-onset period and during non–rapid eye movement sleep, although findings vary according to age and gender. This feature is consistent with increased cortical arousal. Neuroimaging studies have suggested altered regional brain function

consistent with hyperarousal in insomnia, although interpretation of these findings is complex. Individuals with insomnia disorder may have a lower sleep propensity and typically do not show increased daytime sleepiness on objective sleep laboratory measures compared with individuals without sleep disorders.

Other laboratory measures show evidence, although not consistently, of increased arousal and a generalized activation of the hypothalamic-pituitary-adrenal axis (e.g., increased cortisol levels, heart rate variability, reactivity to stress, increased metabolic rate). In general, findings are consistent with the hypothesis that increased physiological and cognitive arousal plays a significant role in insomnia disorder.

Association With Suicidal Thoughts or Behavior

The symptom of insomnia has been identified as an independent risk factor for suicidal thoughts and behavior.

Functional Consequences of Insomnia Disorder

Interpersonal, social, and occupational problems may develop as a result of insomnia or excessive concern with sleep, increased daytime irritability, and poor concentration. Decreased attention and concentration are common and may be related to higher rates of accidents observed in individuals with insomnia. Persistent insomnia is also associated with long-term consequences, including twofold or greater increased risk of new-onset major depressive disorder, anxiety disorders, and substance use disorders. Insomnia symptoms may also be a risk factor for relapse of major depressive disorder. Insomnia disorder, especially with objectively demonstrated short-sleep duration (< 6 hours), is a significant risk factor for numerous cardiovascular diseases, including hypertension, coronary artery disease/myocardial infarction, congestive heart failure, and cerebrovascular disease. Increased absenteeism and reduced productivity at work, reduced quality of life, and increased economic burden are also significant functional consequences of insomnia disorder.

Differential Diagnosis

Normal sleep variations.

Normal sleep duration varies considerably across persons. Some individuals who require little sleep (“short sleepers”) may be concerned about their sleep duration. Short sleepers differ from individuals with insomnia disorder by the lack of difficulty falling or staying asleep and by the absence of characteristic daytime symptoms (e.g., fatigue, concentration problems, irritability). However, some short sleepers may desire or attempt to sleep for a longer period of time and, by prolonging time in bed, may create an insomnia-like sleep pattern. Clinical insomnia also should be distinguished from normal, age-related sleep changes. Insomnia must also be distinguished from sleep deprivation attributable to inadequate opportunity or circumstance for sleep resulting, for example, from an emergency or from professional or family obligations forcing the individual to stay awake.

Situational/acute insomnia. *Situational/acute insomnia* is a condition lasting a few days to several weeks, often associated with acute stress due to life events or with changes in

sleep schedules. These acute or short-term insomnia symptoms may also produce significant distress and interfere with social, personal, and occupational functioning. When such symptoms are frequent enough and meet all other criteria except for the 3-month duration, a diagnosis of other specified insomnia disorder or unspecified insomnia disorder is made. While the disorder often remits with subsidence of the stress or adjustment to the change in sleep schedule, some individuals will develop maladaptive patterns of thought and behavior that result in the development of a chronic insomnia disorder.

Delayed sleep phase and shift work types of circadian rhythm sleep-wake disorder. Individuals with the delayed sleep phase type of circadian rhythm sleep-wake disorder report sleep-onset insomnia only when they try to sleep at socially normal times, but they do not report difficulty falling asleep or staying asleep when their bed and rising times are delayed and coincide with their endogenous circadian rhythm. This pattern is observed particularly among adolescents and younger adults. Shift work type differs from insomnia disorder by the history of recent shift work.

Restless legs syndrome. Restless legs syndrome often produces difficulties initiating and maintaining sleep. However, an urge to move the legs and any accompanying unpleasant leg sensations are features that differentiate this disorder from insomnia disorder.

Breathing-related sleep disorders. Most individuals with a breathing-related sleep disorder have a history of loud snoring, breathing pauses during sleep, and excessive daytime sleepiness. Nonetheless, as many as 50% of individuals with sleep apnea may also report insomnia symptoms, a feature that is more common among women and older adults.

Narcolepsy. Narcolepsy may cause insomnia complaints but is distinguished from insomnia disorder by the predominance of symptoms of excessive daytime sleepiness, cataplexy, sleep paralysis, and sleep-related hallucinations.

Parasomnias. Parasomnias are characterized by a complaint of unusual behavior or events during sleep that may lead to intermittent awakenings and difficulty resuming sleep. However, it is these behavioral events, rather than the insomnia per se, that dominate the clinical picture.

Substance/medication-induced sleep disorder, insomnia type. Substance/medication-induced sleep disorder, insomnia type, is distinguished from insomnia disorder by the fact that a substance (i.e., a drug of abuse, a medication, or exposure to a toxin) is judged to be etiologically related to the insomnia (see “Substance/Medication-Induced Sleep Disorder” later in this chapter). For example, insomnia occurring only in the context of heavy coffee consumption would be diagnosed as caffeine-induced sleep disorder, insomnia type, with onset during intoxication.

Comorbidity

Insomnia is a common comorbidity of many medical conditions, including but not limited to cancer, diabetes, coronary heart disease, chronic obstructive pulmonary disease, arthritis, fibromyalgia, other chronic pain conditions, degenerative brain diseases, and traumatic brain injury. The risk relationship appears to be bidirectional: insomnia increases the risk of many of these medical conditions, and medical problems increase the risk of insomnia. The direction of the relationship is not always clear and may

change over time; for this reason, comorbid insomnia is the preferred terminology when insomnia coexists with another medical condition (or mental disorder). Insomnia disorder also coexists with numerous other sleep disorders. Approximately one in seven individuals with insomnia disorder has moderate to severe obstructive sleep apnea. Rates of insomnia complaints among individuals with narcolepsy are estimated to be about 50%.

Individuals with insomnia disorder frequently have a comorbid mental disorder, particularly bipolar, depressive, and anxiety disorders. Persistent insomnia represents a risk factor or an early symptom of subsequent bipolar, depressive, anxiety, and substance use disorders. Individuals with insomnia may misuse medications or alcohol to help with nighttime sleep, anxiolytics to combat tension or anxiety, and caffeine or other stimulants to combat excessive daytime fatigue. In addition to worsening the insomnia, this type of substance use may in some cases progress to a substance use disorder.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders,* 3rd Edition (ICSD-3), recognizes three insomnia diagnoses: *chronic insomnia disorder*, *short-term insomnia disorder*, and *other insomnia disorder*. DSM-5 insomnia disorder and ICSD-3 chronic insomnia disorder closely parallel each other with respect to symptom, duration, and frequency criteria; however, unlike DSM-5, ICSD-3 does not include a separate designation for substance/medication-induced sleep disorder, insomnia type.

Hypersomnolence Disorder

Diagnostic Criteria	F51.11

A. Self-reported excessive sleepiness (hypersomnolence) despite a main sleep period lasting at least 7 hours, with at least one of the following symptoms:
    1. Recurrent periods of sleep or lapses into sleep within the same day.
    2. A prolonged main sleep episode of more than 9 hours per day that is nonrestorative (i.e., unrefreshing).
    3. Difficulty being fully awake after abrupt awakening.

B. The hypersomnolence occurs at least three times per week, for at least 3 months.

C. The hypersomnolence is accompanied by significant distress or impairment in cognitive, social, occupational, or other important areas of functioning.

D. The hypersomnolence is not better explained by and does not occur exclusively during the course of another sleep disorder (e.g., narcolepsy, breathing-related sleep disorder, circadian rhythm sleep-wake disorder, or a parasomnia).

E. The hypersomnolence is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).

F. Coexisting mental and medical disorders do not adequately explain the predominant complaint of hypersomnolence.

Specify if:

With mental disorder, including substance use disorders

With medical condition

With another sleep disorder

Coding note: The code F51.11 applies to all three specifiers. Code also the relevant associated mental disorder, medical condition, or other sleep disorder immediately after the code for hypersomnolence disorder in order to indicate the association.

Specify if:

Acute: Duration of less than 1 month.

Subacute: Duration of 1–3 months.

Persistent: Duration of more than 3 months.

Specify current severity:

Specify severity based on degree of difficulty maintaining daytime alertness as manifested by the occurrence of multiple attacks of irresistible sleepiness within any given day occurring, for example, while sedentary, driving, visiting with friends, or working.

Mild: Difficulty maintaining daytime alertness 1–2 days/week.

Moderate: Difficulty maintaining daytime alertness 3–4 days/week.

Severe: Difficulty maintaining daytime alertness 5–7 days/week.

Recording Procedures

The specifiers "with mental disorder, including substance use disorders"; "with medical condition"; and "with another sleep disorder" are available to allow the clinician to note clinically relevant comorbidities. In such cases, record F51.11 hypersomnolence disorder, with [name of comorbid condition(s) or disorder(s)] followed by the diagnostic code(s) for the comorbid conditions or disorders (e.g., F51.11 hypersomnolence disorder, with major depressive disorder; F33.1 major depressive disorder, recurrent, moderate).

Diagnostic Features

Hypersomnolence disorder includes symptoms of excessive quantity of sleep (e.g., extended nocturnal sleep or long naps), sleepiness, and *sleep inertia* (i.e., a period of impaired performance and reduced vigilance following awakening from the regular sleep episode or from a nap) (Criterion A). Individuals with this disorder generally fall asleep quickly

and have a good sleep efficiency (> 90%). Individuals typically feel sleepiness developing over a period of time, rather than experiencing a sudden sleep "attack." Unintentional sleep episodes typically occur in sedentary situations (e.g., while attending lectures, reading, watching television, or driving long distances), but in more severe cases they can manifest in high-attention situations such as at work, in meetings, or at social gatherings. The persistent need for sleep can lead to automatic behavior (usually of a very routine, low-complexity type) that the individual carries out with little or no subsequent recall. For example, individuals may find themselves having driven several miles from where they thought they were, unaware of the "automatic" driving they did in the preceding minutes.

About 40% of individuals with hypersomnolence disorder may have sleep inertia (also referred to as "sleep drunkenness"), and this symptom may help differentiate hypersomnolence disorder from other causes of sleepiness. They may have difficulty waking up in the morning, sometimes appearing confused, combative, or ataxic. Individuals may set multiple alarm clocks or rely on others to help get them out of bed. Sleep inertia can also occur upon awakening from a daytime nap. During that period, the individual appears awake, but motor coordination is impaired, behavior may be inappropriate, and memory deficits, disorientation in time and space, and feelings of grogginess may occur. This period may last some minutes to hours.

For some individuals with hypersomnolence disorder, the major sleep episode (for most individuals, nocturnal sleep) has a duration of 9 hours or more. In the most extreme cases, sleep episodes can last up to 20 hours. However, the sleep is often nonrestorative and is followed by difficulty awakening in the morning. For other individuals with hypersomnolence disorder, the major sleep episode is of normal nocturnal sleep duration (7–9 hours), and they take relatively long daytime naps (> 1 hour) that do not improve alertness. Most individuals with hypersomnolence disorder take daytime naps nearly every day regardless of the nocturnal sleep duration. While many individuals with hypersomnolence are able to reduce their sleep time during working days, weekend and holiday sleep is greatly increased (by up to 3 hours).

Associated Features

Approximately 80% of individuals with hypersomnolence disorder report that their sleep is nonrestorative, but this symptom is nonspecific and can occur with disorders that disrupt sleep, such as obstructive sleep apnea. Naps are often long (> 1 hour) and unrefreshing. Short naps (i.e., duration of < 30 minutes) are often unrefreshing. Individuals with hypersomnolence often appear sleepy and may even fall asleep in the clinician's waiting area.

A subset of individuals with hypersomnolence disorder have a family history of hypersomnolence and also have symptoms of autonomic nervous system dysfunction, including recurrent vascular-type headaches, reactivity of the peripheral vascular system (Raynaud's phenomenon), and fainting.

Prevalence

Approximately 5%–10% of individuals in the United States who consult in sleep disorder clinics with complaints of daytime sleepiness are diagnosed as having hypersomnolence disorder. It is estimated that about 1% of the European and U.S. general population has episodes of sleep inertia. Hypersomnolence occurs with relatively equal frequency in men and women.

Development and Course

Hypersomnolence disorder usually begins in late adolescence or early adulthood, with a mean age at onset of 17–24 years and a gradual progression over weeks to months. Little is known of the natural history, but for most individuals, the symptoms are persistent and stable, unless treatment is initiated. Spontaneous remission occurs in about 11%–25% of individuals after 5–7 years. Individuals with hypersomnolence disorder are diagnosed, on average, 10–15 years after the appearance of the first symptoms. Pediatric cases are rare. The development of other sleep disorders (e.g., breathing-related sleep disorder) may worsen the degree of sleepiness.

Risk and Prognostic Factors

Environmental.

Hypersomnolence can be increased temporarily by psychological stress and alcohol use, but they have not been documented as environmental precipitating factors. Viral infections have been reported to have preceded or accompanied hypersomnolence in about 10% of cases. Hypersomnolence is common in the months after traumatic brain injury.

Genetic and physiological. Hypersomnolence may be familial, with an autosomal-dominant mode of inheritance.

Diagnostic Markers

Nocturnal polysomnography demonstrates a normal to prolonged sleep duration, short sleep latency, and normal to increased sleep continuity. The nocturnal distribution of rapid eye movement (REM) sleep is also normal. Sleep efficiency is typically > 90%. The multiple sleep latency test documents sleep tendency, typically indicated by mean sleep latency values of < 8 minutes. In hypersomnolence disorder, the mean sleep latency is typically < 10 minutes and frequently 8 minutes or less. Sleep-onset REM periods (i.e., the occurrence of REM sleep within 20 minutes of sleep onset) may be present but occur infrequently. Unfortunately, the multiple sleep latency test has poor test-retest reliability, and it does not distinguish well between hypersomnolence disorder and narcolepsy type 2.

A 2-week sleep diary can help document amounts and timing of sleep, and actigraphy provides more accurate data on habitual sleep patterns. In a 32-hour bed rest protocol in which subjects were encouraged to sleep ad lib, individuals with hypersomnolence disorder slept > 4 hours more than control subjects.

Functional Consequences of Hypersomnolence Disorder

The low level of alertness that occurs while an individual fights the need for sleep can lead to reduced efficiency, diminished concentration, and poor memory during daytime activities. Hypersomnolence can lead to significant distress and dysfunction in work and social relationships. Prolonged nocturnal sleep and difficulty awakening can result in difficulty in meeting

morning obligations, such as arriving at work on time. Unintentional daytime sleep episodes can be embarrassing and even dangerous, if, for instance, the individual is driving or operating machinery when the episode occurs.

Differential Diagnosis

Normative variation in sleep. "Normal" sleep duration varies considerably in the general population. "Long sleepers" (i.e., persons who require a greater than average amount of sleep) do not have excessive sleepiness, sleep inertia, or automatic behavior when they obtain their required amount of nocturnal sleep. Sleep is reported to be refreshing. If social or occupational demands lead to shorter nocturnal sleep, daytime symptoms may appear. In hypersomnolence disorder, by contrast, symptoms of excessive sleepiness occur regardless of nocturnal sleep duration.

An inadequate amount of nocturnal sleep, or *behaviorally induced insufficient sleep syndrome,* can produce symptoms of daytime sleepiness very similar to those of hypersomnolence disorder. An average sleep duration of fewer than 7 hours per night strongly suggests inadequate nocturnal sleep, yet in the United States, the average adult obtains only 6.75 hours of sleep on typical weeknights. Individuals with inadequate nocturnal sleep typically "catch up" with longer sleep durations on days when they are free from social or occupational demands or on vacations. A diagnosis of hypersomnolence disorder should not be made if there is a question regarding the adequacy of nocturnal sleep duration. A diagnostic and therapeutic trial of sleep extension for 10–14 days can often clarify the diagnosis.

Narcolepsy. As in hypersomnolence disorder, individuals with narcolepsy have chronic sleepiness, but several clinical and laboratory findings help distinguish the disorders. In contrast to those with hypersomnolence disorder, individuals with narcolepsy tend to sleep 7–8 hours each day and generally feel refreshed on waking in the morning. Individuals with narcolepsy generally feel more alert after a 15- to 20-minute nap, whereas those with hypersomnolence disorder tend to take longer naps, have trouble waking from naps, and do not feel alert afterward. Individuals with narcolepsy also have varying amounts of cataplexy, hypnagogic

hallucinations, sleep paralysis, and fragmented nocturnal sleep, whereas cataplexy never occurs in hypersomnolence disorder and the other symptoms are uncommon. The multiple sleep latency test typically shows more than two sleep-onset REM periods in narcolepsy.

Fatigue as a symptom of another mental disorder or medical condition. Hypersomnolence disorder should be distinguished from tiredness related to fatigue that may be a symptom of another mental disorder (e.g., generalized anxiety disorder) or medical condition (e.g., chronic fatigue syndrome). Unlike hypersomnolence, tiredness is not necessarily relieved by increased sleep and is unrelated to sleep quantity or quality.

Breathing-related sleep disorders. Chronic sleepiness is common in breathing-related sleep disorders. Individuals with hypersomnolence and breathing-related sleep disorders may have similar patterns of excessive sleepiness. Breathing-related sleep disorders are suggested by a history of loud snoring, pauses in breathing during sleep, and nonrefreshing sleep. Examination often reveals obesity, a small airway, and large neck diameter. Hypertension is common, and some individuals may demonstrate signs of heart failure. Polysomnographic studies can confirm the presence of apneic events in breathing-related sleep disorder (and their absence in hypersomnolence disorder).

Circadian rhythm sleep-wake disorders. In contrast to individuals with hypersomnolence disorder, individuals with specific subtypes of circadian rhythm sleep-wake disorder show specific temporal patterns of symptoms. For example, individuals with delayed sleep phase type often have sleep inertia and sleepiness in the morning and feel most alert in the evening and night, with habitually late bedtimes. In contrast, those with advanced sleep phase type become sleepy and go to bed early in the evening but are alert and wake easily in the early morning.

Parasomnias. Parasomnias such as non–REM sleep arousal disorders (sleepwalking/sleep terrors) or REM sleep behavior disorder rarely produce the prolonged, undisturbed nocturnal sleep or daytime sleepiness characteristic of hypersomnolence disorder. However, parasomnias such as

nightmare disorder, which may result in significant curtailment of total sleep time, may conceivably manifest with daytime sleepiness.

Hypersomnolence in other mental disorders and medical conditions. Hypersomnolence disorder must be distinguished from hypersomnolence occurring as a symptom of another mental disorder (e.g., major depressive episode, especially episodes with atypical features) or medical condition (e.g., certain cancers, multiple sclerosis). If the predominant complaint of excessive sleepiness is adequately explained by another mental disorder or medical condition, then an additional diagnosis of hypersomnolence disorder is not warranted. However, if the hypersomnolence is not adequately explained by a comorbid mental disorder or medical condition (e.g., the severity and nature of the hypersomnolence far exceed what would be expected with the mental disorder or medical condition), an additional diagnosis of hypersomnolence disorder is warranted.

Comorbidity

Many individuals with hypersomnolence disorder have symptoms of depression that may meet criteria for a depressive disorder. This presentation may be related to the psychosocial consequences of persistent increased sleep need. More than half of individuals with hypersomnolence disorder have attention-deficit/hyperactivity disorder symptoms. Individuals with hypersomnolence disorder are also at risk for substance-related disorders, particularly related to self-medication with stimulants. This general lack of specificity may contribute to very heterogeneous profiles among individuals whose symptoms meet the diagnostic criteria for hypersomnolence disorder. Neurodegenerative conditions, such as Alzheimer's disease, Parkinson's disease, and multiple system atrophy, may also be associated with hypersomnolence.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders,* 3rd Edition (ICSD-3), differentiates nine subtypes of "central disorders of hypersomnolence," including disorders not covered in DSM such as Kleine-Levin syndrome

(recurrent episodes of hypersomnia), hypersomnolence due to a medical/neurological condition or substance use, and insufficient sleep syndrome.

Narcolepsy

Diagnostic Criteria

A. Recurrent periods of an irrepressible need to sleep, lapsing into sleep, or napping occurring within the same day. These must have been occurring at least three times per week over the past 3 months.
B. The presence of at least one of the following:
    1. Episodes of cataplexy, defined as either (a) or (b), occurring at least a few times per month:
        a. In individuals with long-standing disease, brief (seconds to minutes) episodes of sudden bilateral loss of muscle tone with maintained consciousness that are precipitated by laughter or joking.
        b. In children or in individuals within 6 months of onset, spontaneous grimaces or jaw-opening episodes with tongue thrusting or a global hypotonia, without any obvious emotional triggers.
    2. Hypocretin deficiency, as measured using cerebrospinal fluid (CSF) hypocretin-1 immunoreactivity values (less than or equal to one-third of values obtained in healthy subjects tested using the same assay, or less than or equal to 110 pg/mL). Low CSF levels of hypocretin-1 must not be observed in the context of acute brain injury, inflammation, or infection.

3. Nocturnal sleep polysomnography showing rapid eye movement (REM) sleep latency less than or equal to 15 minutes, or a multiple sleep latency test showing a mean sleep latency less than or equal to 8 minutes and two or more sleep-onset REM periods.

Specify whether:

G47.411 Narcolepsy with cataplexy or hypocretin deficiency (type 1): Criterion B1 (episodes of cataplexy) or Criterion B2 (low CSF hypocretin-1 levels) is met.

G47.419 Narcolepsy without cataplexy and either without hypocretin deficiency or hypocretin unmeasured (type 2): Criterion B3 (positive polysomnography/multiple sleep latency test) is met, but Criterion B1 is not met (i.e., no cataplexy is present) and Criterion B2 is not met (i.e., CSF hypocretin-1 levels are not low or have not been measured).

G47.421 Narcolepsy with cataplexy or hypocretin deficiency due to a medical condition

G47.429 Narcolepsy without cataplexy and without hypocretin deficiency due to a medical condition

Coding note: For the subtype narcolepsy with cataplexy or hypocretin deficiency due to a medical condition and the subtype narcolepsy without cataplexy and without hypocretin deficiency due to a medical condition, code first the underlying medical condition (e.g., G71.11 myotonic dystrophy; G47.429 narcolepsy without cataplexy and without hypocretin deficiency due to myotonic dystrophy).

Specify current severity:

Mild: Need for naps only once or twice per day. Sleep disturbance, if present, is mild. Cataplexy, when present, is infrequent (occurring less than once per week).

Moderate: Need for multiple naps daily. Sleep may be moderately disturbed. Cataplexy, when present, occurs daily or every few days.

Severe: Nearly constant sleepiness and, often, highly disturbed nocturnal sleep (which may include excessive body movement and vivid dreams). Cataplexy, when present, is drug-resistant, with multiple attacks daily.

Subtypes

A diagnosis of narcolepsy, type 1 (NT1; i.e., with cataplexy or hypocretin deficiency) is most often based on the presence of recurrent sleepiness and cataplexy (given the limited use of cerebrospinal fluid [CSF] hypocretin determinations). However, cataplexy can emerge years following onset of sleepiness. Therefore, some individuals may be initially assigned a diagnosis of narcolepsy, type 2 (NT2; i.e., without cataplexy and either without hypocretin deficiency or with hypocretin unmeasured), based on sleepiness and positive multiple sleep latency test (MSLT) findings, only to be reassigned to a diagnosis of NT1 following emergence of cataplexy. NT1 established by demonstration of low CSF hypocretin levels may manifest without evidence of clear cataplexy. Other explanations for excessive daytime sleepiness (e.g., sleep deprivation, shift work, other sleep disorders) and episodes of sudden loss of muscle tone (e.g., seizures, falls of other origin, functional neurological symptom disorder [conversion disorder]) should be ruled out. NT2 is established on the basis of chronic sleepiness and characteristic nocturnal sleep polysomnography findings (e.g., short REM sleep latency) or MSLT findings showing short mean sleep latency and two or more sleep-onset REM periods (SOREMPs).

NT1 and NT2 can result from other neurological, infectious, metabolic, and genetic conditions. Inherited disorders, tumors, and head trauma are the most common causes of secondary narcolepsy. In other cases, the destruction of hypocretin neurons may be secondary to trauma or hypothalamic surgery. Head trauma or infections of the central nervous system can, however, produce transitory decreases in CSF hypocretin-1 levels without hypocretin cell loss, complicating the diagnosis.

Other etiologies include inflammatory lesions due to multiple sclerosis and acute disseminated encephalomyelitis, vascular disorders such as

stroke, and encephalitis. Autosomal dominant cerebellar ataxia, deafness, and narcolepsy, or ADCA DN, is a familial degenerative disorder due to missense mutations of the DNA methyltransferase (DNMT1) gene. Cataplexy with some degree of sleepiness can be caused by other neurological conditions, including Prader-Willi syndrome, Niemann-Pick disease type C, Möbius syndrome, and Norrie disease. Hypocretin deficiency has been reported in Parkinson's disease as well. NT2-like physiology has been reported in myotonic dystrophy and Prader-Willi syndrome.

Diagnostic Features

The essential features of narcolepsy are recurrent daytime naps or lapses into sleep that occur typically daily but that must occur at a minimum of three times a week for at least 3 months (Criterion A), and are accompanied by one or more of the following: cataplexy (Criterion B1), hypocretin deficiency (Criterion B2), or characteristic abnormalities on a nocturnal polysomnogram or on the MSLT (Criterion B3). In most individuals with NT1, the first symptom to manifest is sleepiness or increased sleep need, followed by cataplexy. Sleepiness is worse in sedentary circumstances and typically is relieved by brief (10–20 minutes) naps.

NT1 generally manifests with *cataplexy*, typically brief episodes (seconds up to 2 minutes) of sudden, bilateral loss of muscle tone precipitated by emotions. A range of positive emotions can trigger cataplexy, including those associated with laughter, anticipation, or surprise. Less commonly, cataplexy can be triggered by negative emotions such as anger and embarrassment. Muscles affected include those of the neck, jaw, arms, legs, or whole body, resulting in head bobbing, jaw dropping, or complete falls. Individuals are awake and aware during cataplexy. Cataplexy should not be confused with "weakness" occurring in

the context of athletic activities (physiological) or exclusively after unusual emotional triggers such as stress or anxiety (suggesting possible psychopathology).

In children and rarely in adults with acute NT1 symptom onset, cataplexy may manifest as continuous hypotonia rather than episodic bouts of weakness triggered by strong emotions. This continuous hypotonia may result in gait instability, ptosis, and slack jaw. Superimposed on this muscle weakness, some individuals may demonstrate phenomena such as tongue protrusion and grimacing. This static cataplexy is most common within 6 months of a rapid onset.

NT1 is caused by loss of hypothalamic neurons that produce the hypocretin (orexin) neuropeptides, and CSF hypocretin levels are typically less than one-third of control values (< 110 pg/mL in most laboratories). Individuals with cataplexy have been shown to have low CSF hypocretin levels in 85%–90% of cases. In contrast, most individuals with NT2 have normal or intermediate levels of CSF hypocretin. Thus, hypocretin deficiency is a sufficient diagnostic test for NT1 (Criterion B2). If CSF hypocretin is measured and not low, an NT2 diagnosis is based on clinical symptoms (Criterion A) and sleep study data outlined in Criterion B3.

A nocturnal polysomnogram followed by an MSLT is the conventional method for confirming the diagnosis of both NT1 (if hypocretin testing is unavailable or not feasible) and NT2 (Criterion B3). These tests must be performed after the individual has stopped all psychotropic medications (for a duration based on elimination half-life) and obtained adequate sleep time on a normal sleep-wake schedule (as documented with sleep diaries or, preferably, actigraphy), ideally for 2 weeks. Notably, the abrupt discontinuation of antidepressants, α-adrenergic agonist medications, or stimulants or use of these medications during testing can alter REM sleep physiology.

The MSLT result must be positive for a diagnosis of NT2, showing a mean sleep latency of ≤ 8 minutes plus at least two SOREMPs; specifically, REM sleep must occur in at least two of the five nap opportunities. Alternatively, a nocturnal sleep-onset REM period (nSOREMP; REM sleep-onset latency ≤ 15 minutes) during polysomnography is sufficient to confirm the diagnosis and meets Criterion B3. An nSOREMP is highly specific to NT1 (95%–97%) but only moderately sensitive (54%–57%). False positive findings of SOREMPs can occur with shift work, circadian rhythm sleep-wake disorders, severe obstructive sleep apnea, medication effects, and insufficient sleep disorder.

The nocturnal polysomnogram and MSLT are diagnostically limited, especially in NT2. While reliability of diagnostic MSLT testing is relatively high at 85%–95% for NT1, reliability for the NT2 diagnosis is poorer. Test-retest reliability may be < 50%. This poor reliability may be due to day-to-day variability in NT2 physiology and technical aspects of the polysomnogram and MSLT testing, especially inadequate attention to prior sleep time/schedule and medication/drug use.

Normal or intermediate CSF hypocretin levels among individuals with cataplexy symptoms can decline to undetectable levels over time.

Associated Features

When sleepiness is severe, automatic behaviors may occur, with the individual continuing his or her activities in a semiautomatic, hazelike fashion without memory or consciousness. Approximately 20%–60% of individuals experience vivid hypnagogic hallucinations before or upon falling asleep or hypnopompic hallucinations just after awakening. These hallucinations are typically visual or auditory, and sometimes tactile. They are distinct from the less vivid, nonhallucinatory dream-like mentation at sleep onset that occurs in persons with normal sleep.

Approximately 20%–60% of affected individuals experience sleep paralysis upon falling asleep or awakening, leaving them awake but unable to move or speak. However, many normal sleepers also report occasional sleep paralysis, especially with stress or sleep

deprivation. Individuals with narcolepsy can have a range of nocturnal sleep symptoms, including disrupted nighttime sleep (frequent, brief awakenings), vivid and realistic dreams, periodic limb movements of sleep, and REM sleep behavior disorder. Nocturnal eating may occur. Obesity is common. Individuals may appear sleepy or fall asleep in the waiting area or during clinical examination. During cataplexy, individuals may slump in a chair and have slurred speech or drooping eyelids. If the clinician is able to check reflexes during cataplexy (most attacks are < 10 seconds), reflexes are abolished during whole body cataplexy—an important finding

distinguishing genuine cataplexy from functional neurological symptom disorder (conversion disorder).

Although IQ testing is generally normal in individuals with narcolepsy, impairments in working memory and executive functioning have been reported.

Prevalence

Narcolepsy-cataplexy (NT1) affects 0.02%–0.05% of the adult general population worldwide and has an incidence of 0.74 per 100,000 person-years in the United States. Some prevalence variation has been reported, including lower rates in Israel and higher rates in Japan than in Europe and the United States. The true prevalence of NT2 is unknown in part because of diagnostic variability. Narcolepsy affects both genders fairly equally, but this may vary among different populations.

Development and Course

Onset occurs most often in childhood and adolescence or young adulthood but rarely in old age. Peak age at onset is around 15–25 years. Onset can be abrupt or progressive, with cataplexy developing over years. It has been reported that children presenting with abrupt onset of NT1 symptoms have the highest disease severity but that disease severity in these cases tends to partially improve in the first few years after onset. Abrupt onset in young, prepubescent children can be associated with obesity and premature puberty. About 50% of individuals with narcolepsy diagnosed in adulthood recall symptom onset in childhood or adolescence, highlighting problems of diagnostic delays for this condition. Once the disorder has manifested, the course is persistent and lifelong.

In 90% of cases, the first symptom to manifest is sleepiness or increased sleep, followed by cataplexy (within 1 year in 50% of cases, within 3 years in 85%). Sleepiness, hypnagogic hallucinations, vivid dreaming, and REM sleep behavior disorder (vocalizations or complex motor behavior during REM sleep) are early symptoms. Excessive sleep rapidly progresses to an inability to stay awake during the day, and to maintain good sleep at night, without a clear increase in total 24-hour sleep time. In the first months, cataplexy may be atypical, especially in children, manifesting with a

generalized hypotonia rather than with episodic emotionally triggered weakness. In general, narcolepsy symptoms remain fairly stable but may fluctuate with life events such as pregnancy and stressors. Exacerbations of symptoms suggest lack of compliance with medications or development of a concurrent sleep disorder, notably sleep apnea, which has been identified in about a quarter of individuals with narcolepsy.

Young children and adolescents with narcolepsy often develop aggression or behavioral problems secondary to sleepiness and/or nighttime sleep disruption. Workload and social pressure increase through high school and college, reducing available sleep time at night. Pregnancy does not seem to modify symptoms consistently. After retirement, individuals typically have more opportunity for napping, reducing the need for stimulants. Maintaining a regular schedule benefits individuals at all ages.

Risk and Prognostic Factors

Temperamental. Individuals with narcolepsy commonly report that they need more sleep than other family members.

Environmental. Group A streptococcal throat infection, influenza (notably pandemic H1N1 2009), or other winter infections, as well as vaccinations (specifically Pandemrix H1N1 vaccination), may trigger an autoimmune process in some individuals, producing narcolepsy a few months later. Head trauma and abrupt changes in sleep-wake patterns (e.g., job changes, stress) may be additional triggers.

Genetic and physiological. Monozygotic twins are 25%–32% concordant for narcolepsy. The prevalence of narcolepsy is 1%–2% in first-degree relatives (a 10- to 40-fold increase overall). Narcolepsy is strongly associated with HLA DQB1*06:02 (see “Diagnostic Markers”). DQB1*03:01 increases, while DQB1*05:01, DQB1*06:01, and DQB1*06:03 reduce risk in the presence of DQB1*06:02, but the effect is small. Polymorphisms within the T-cell receptor alpha gene and other immune-modulating genes also modulate risk slightly.

Culture-Related Diagnostic Issues

Narcolepsy has been described in many ethnoracial groups and cultural contexts. One study of 1,097 treatment-seeking individuals suggested that among African Americans, more cases may manifest without cataplexy or with atypical cataplexy (although CSF hypocretin is low), and with earlier onset compared with non-Latinx Whites. Diagnosis may be further complicated by the higher presence of obesity and obstructive sleep apnea in this population, which can be related to differential exposure to social determinants of health, including food insecurity, food deserts, and limited access to safe and affordable places for physical activity. Individuals with narcolepsy often experience sleep paralysis, which may be attributed to supernatural forces (e.g., frightening spirit is sitting on the sleeper's chest) in some cultural contexts, contributing to the perceived dangerousness of the condition and to help-seeking decisions.

Diagnostic Markers

Nocturnal polysomnography followed by an MSLT is used to confirm the diagnosis of narcolepsy, especially when the disorder is first being diagnosed and before treatment has begun. In the presence of clear-cut cataplexy, the polysomnography and MSLT are confirmatory for NT1. In the absence of cataplexy and hypocretin deficiency (if measured), the MSLT is diagnostic of NT2. Drug or medication effects (e.g., REM-inhibiting antidepressants or sedating medications), stimulant withdrawal, prior sleep deprivation, shift work, or severe depression may result in an inaccurate MSLT result and must be ruled out prior to performance of the MSLT. In particular, chronically insufficient sleep is common and must be considered.

An nSOREMP is highly specific (approximately 1% positive in control subjects) but moderately sensitive (approximately 50%) for NT1. In contrast, an nSOREMP was only found in 10%–23% of NT2 persons with normal hypocretin levels, suggesting even lower sensitivity in this subtype. The MSLT result is considered positive for narcolepsy if it displays an average sleep latency of ≤ 8 minutes and SOREMPs in two or more naps on a four- or five-nap test. The MSLT result is positive in 90%–95% of individuals with NT1 versus 2%–4% of control subjects or individuals with

other sleep disorders. As noted, poor test-retest reliability for NT2 precludes determination of comparable data for NT2. Additional polysomnographic findings among individuals with narcolepsy often include frequent arousals, decreased sleep efficiency, and increased stage 1 sleep. Periodic limb movements (found in about 40% of individuals with NT1) and sleep apnea are often noted.

Hypocretin deficiency is demonstrated by measuring CSF hypocretin-1 levels. The test is particularly useful in individuals with suspected pseudocataplexy and those without typical cataplexy, or in treatment-refractory cases. The diagnostic value of the test is not affected by medications, sleep deprivation, or the time of day or night when it is collected, but the findings are uninterpretable when the individual is severely ill with a concurrent

infection or head trauma or is comatose. CSF cytology, protein, and glucose are within normal range even when sampled within weeks of rapid onset of the disorder. When measured in individuals with typical cataplexy symptoms, CSF hypocretin-1 is often already very diminished or undetectable.

About 85%–95% of individuals with NT1 are positive for the HLA DQB1*06:02 haplotype. This gene influences immune system antigen presentation, supporting an underlying autoimmune pathophysiology of NT1. Outbreaks of NT1 after specific vaccinations and infections further support an autoimmune etiology. In contrast to NT1, there are no biomarkers of NT2. Only about 40%–50% of individuals with NT2 are positive for DQB1*06:02. As 12%–38% of the general population is DQB1*06:02 positive, testing for this allele is not very helpful for diagnosing NT2 but can be helpful for screening of NT1.

Functional Consequences of Narcolepsy

School performance, driving, work, or other activities that require sustained attention are impaired, and individuals with narcolepsy should avoid jobs that place themselves (e.g., working with machinery) or others (e.g., bus

driver, pilot) in danger. Once the narcolepsy is controlled with therapy, individuals can usually drive, although rarely long distances alone. Untreated individuals are also at risk for social isolation and accidental injury to themselves or others. Social relations may suffer as these individuals strive to avert cataplexy by exerting control over emotions or stimuli that cause emotions.

Differential Diagnosis

Other hypersomnias. Hypersomnolence disorder (also known as idiopathic hypersomnia) and narcolepsy are similar with respect to the presence of chronic daytime sleepiness, age at onset (typically adolescence or early adulthood), and stable course over time, but can be distinguished based on distinctive clinical and laboratory features. Individuals with hypersomnolence disorder typically have longer and less disrupted nocturnal sleep, greater difficulty awakening, more persistent daytime sleepiness (as opposed to more discrete “sleep attacks” in narcolepsy), longer and less refreshing daytime sleep episodes, and little or no dreaming during daytime naps. By contrast, individuals with NT1 generally have cataplexy. Those with NT1 or NT2 may demonstrate recurrent intrusions of elements of REM sleep into the transition between sleep and wakefulness (e.g., sleep-related hallucinations and sleep paralysis). The MSLT typically demonstrates shorter sleep latencies (i.e., greater physiological sleepiness) as well as the presence of multiple SOREMPs in individuals with narcolepsy.

Sleep deprivation and insufficient nocturnal sleep. Sleep deprivation and insufficient nocturnal sleep are common in adolescents and shift workers. In adolescents, difficulties falling asleep at night are common, causing sleep deprivation. The MSLT result may be falsely positive if conducted while the individual is sleep deprived or while his or her sleep is phase delayed.

Sleep apnea syndromes. Obstructive sleep apnea is common in the general population and can be present in individuals with narcolepsy due to obesity. Because obstructive sleep apnea is more frequent than narcolepsy, cataplexy may be overlooked (or absent). Narcolepsy should be considered in individuals with persistent sleepiness despite treatment of their sleep apnea.

Insomnia disorder.

Individuals with narcolepsy may focus on the presence of nocturnal sleep disruption and incorrectly attribute daytime sleepiness to insomnia disorder. Although individuals with narcolepsy, like those with insomnia disorder, may experience frequent awakenings during the night, individuals with narcolepsy typically have no difficulty initiating sleep or returning to sleep in contrast to those with insomnia disorder.

Moreover, insomnia disorder is not typically associated with the severity of daytime sleepiness observed in narcolepsy.

Major depressive disorder. Excessive daytime sleepiness is a common complaint of both individuals with major depression and individuals with narcolepsy. The presence of cataplexy (which is not a feature of major depressive disorder) along with the severity of excessive daytime sleepiness indicates a diagnosis of NT1 rather than major depressive disorder. Moreover, in individuals with major depression, MSLT results are most often normal, and there is dissociation between subjective and objective sleepiness, as measured by the mean sleep latency during the MSLT. In a meta-analysis of individuals with psychiatric disorders evaluated for sleepiness, while 25% had a mean sleep latency of < 8 minutes on the MSLT, only rarely were two or more SOREMPs noted on the MSLT, highlighting the more specific REM sleep dysfunction of narcolepsy.

Functional neurological symptom disorder (conversion disorder; pseudocataplexy). Individuals with functional neurological symptom disorder can present with weakness that may raise questions of cataplexy. However, in functional neurological symptom disorder, the weakness is often long-lasting, has unusual triggers, and can result in frequent falls. Individuals may report sleeping and dreaming during MSLT naps, yet the MSLT does not show the characteristic SOREMP. Home video recordings and video during sleep studies can be helpful to distinguish this condition from true cataplexy. The weakness is usually generalized in pseudocataplexy, without partial attacks. Full-blown, long-lasting pseudocataplexy may occur during consultation, allowing the examining physician enough time to verify reflexes, which remain intact.

Attention-deficit/hyperactivity disorder or other behavioral problems. In children and adolescents, sleepiness can cause behavioral problems, including aggressiveness and inattention, leading to a misdiagnosis of attention-deficit/hyperactivity disorder (ADHD).

Atonic seizures. Atonic seizures, a type of seizure that causes sudden loss of muscle strength, must be distinguished from cataplexy. Atonic seizures are not commonly triggered by emotions and tend to manifest as abrupt falls rather than the slower "melting" quality of cataplexy. Atonic seizures usually occur in individuals with additional seizure types and have distinct signatures on the electroencephalogram.

Syncope. Like syncope, cataplexy usually develops over several seconds, but individuals with cataplexy do not have presyncopal symptoms of dizziness, tunnel vision, and auditory changes.

Chorea and motor disorders. In young children, cataplexy can be misdiagnosed as chorea or pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS), especially in the context of a strep throat infection and high antistreptolysin O antibody levels. Some children may have an overlapping motor disorder close to onset of the cataplexy.

Schizophrenia. In the presence of florid and vivid hypnagogic hallucinations, individuals with narcolepsy may think these experiences are real—a feature that suggests the presence of a true hallucination characteristic of schizophrenia. However, clear differences have been described in the pattern of hallucinatory experiences in narcolepsy compared with schizophrenia. Individuals with narcolepsy tend to report sleep-related multisensory "holistic" hallucinations (visual, auditory, tactile) rather than the predominantly verbal-auditory sensory mode of individuals with schizophrenia. Moreover, high-dose stimulant treatment of individuals with narcolepsy may result in the development of persecutory delusions. If cataplexy is present with hallucinations or delusions, the first clinical supposition would be that these symptoms are secondary to narcolepsy before consideration of a co-occurring diagnosis of schizophrenia.

Comorbidity

Medical and psychiatric comorbidities are common among individuals with narcolepsy and include obesity, bruxism, enuresis, precocious puberty (among individuals with pediatric-onset narcolepsy), mood disorders, and ADHD. Rapid weight gain is common in young children with a sudden disease onset. Parasomnias (e.g., sleepwalking, REM sleep behavior disorder), obstructive sleep apnea, restless legs syndrome, and periodic limb movements are common in individuals who develop narcolepsy. Comorbid sleep apnea should be considered if there is a sudden aggravation of preexisting narcolepsy.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 3rd Edition (ICSD-3), differentiates two subtypes of narcolepsy: NT1 (narcolepsy with cataplexy or hypocretin deficiency) and NT2 (narcolepsy without cataplexy or hypocretin deficiency). NT1 secondary to another medical condition (G47.421) and NT2 secondary to another medical condition (G47.429) are reported in ICSD-3 as secondary narcolepsy subtypes.

Breathing-Related Sleep Disorders

The breathing-related sleep disorders category encompasses three relatively distinct disorders: obstructive sleep apnea hypopnea, central sleep apnea, and sleep-related hypoventilation.

Obstructive Sleep Apnea Hypopnea

Diagnostic Criteria	**G47.33**
A. Either (1) or (2):	

1. Evidence by polysomnography of at least five obstructive apneas or hypopneas per hour of sleep and either of the following sleep symptoms:
    a. Nocturnal breathing disturbances: snoring, snorting/gasping, or breathing pauses during sleep.
    b. Daytime sleepiness, fatigue, or unrefreshing sleep despite sufficient opportunities to sleep that is not better explained by another mental disorder (including a sleep disorder) and is not attributable to another medical condition.
2. Evidence by polysomnography of 15 or more obstructive apneas and/or hypopneas per hour of sleep regardless of accompanying symptoms.

Specify current severity:

Mild: Apnea hypopnea index is less than 15.

Moderate: Apnea hypopnea index is 15–30.

Severe: Apnea hypopnea index is greater than 30.

Specifiers

Disease severity is measured by a count of the number of apneas plus hypopneas per hour of sleep (apnea hypopnea index) using polysomnography or other overnight monitoring.

Apnea refers to the total absence of airflow, and *hypopnea* refers to a reduction in airflow. Overall severity is also informed by levels of nocturnal desaturation and sleep fragmentation (measured by brain cortical arousal frequency and sleep stages) and degree of associated symptoms and daytime impairment. However, the exact number and thresholds may vary according to the specific measurement techniques used, and these numbers may change over time. Regardless of the apnea hypopnea index (count) per se, the disorder is considered to be more severe when apneas and hypopneas

are accompanied by significant oxygen hemoglobin desaturation (e.g., when more than 10% of the sleep time is spent at desaturation levels of < 90%) or when sleep is severely fragmented as shown by an elevated arousal index (arousals per hour of sleep > 30) or reduced time in deep sleep (e.g., percentage stage N3 [slow-wave sleep] < 5%).

Diagnostic Features

Obstructive sleep apnea hypopnea is the most common breathing-related sleep disorder. It is characterized by repeated episodes of upper (pharyngeal) airway obstruction (apneas and hypopneas) during sleep. Each apnea or hypopnea represents a reduction in breathing of at least 10 seconds in duration in adults or two missed breaths in children and is typically associated with drops in oxygen saturation of ≥ 3% and/or an electroencephalographic arousal. Both sleep-related (nocturnal) and wake-time symptoms are common. The cardinal symptoms of obstructive sleep apnea hypopnea are snoring and daytime sleepiness.

Obstructive sleep apnea hypopnea in adults is diagnosed on the basis of findings from a polysomnogram (or sleep testing performed outside of the sleep center, referred to as *out of center sleep testing* [OCST]) and symptoms. The diagnosis is based on symptoms of 1) nocturnal breathing disturbances (i.e., snoring, snorting/gasping, breathing pauses during sleep), or 2) daytime sleepiness, fatigue, or unrefreshing sleep despite sufficient opportunities to sleep that are not better explained by another mental disorder and not attributable to another medical condition, along with 3) evidence by polysomnography (or OCST) of five or more obstructive apneas or hypopneas per hour of sleep (Criterion A1). Diagnosis can be made in the absence of these symptoms if there is evidence by polysomnography (or limited OCST) of 15 or more obstructive apneas and/or hypopneas per hour of sleep (Criterion A2).

Criteria for a diagnosis of obstructive sleep apnea hypopnea in children differ from those for a diagnosis in adults. An obstructive apnea hypopnea index of one or more events per hour or evidence of obstructive hypoventilation in association with snoring or polysomnographic evidence of airflow obstruction is used to define thresholds of abnormality in children. Polysomnographic findings in children may differ from those in

adults in that children may demonstrate labored breathing; partial obstructive hypoventilation (sustained reductions of tidal volume due to upper airway flow limitations) with cyclical oxygen desaturations; hypercapnia; and paradoxical breathing.

Most cases of obstructive sleep apnea remain undiagnosed. Therefore, specific attention to symptoms of disturbed sleep occurring in association with snoring or breathing pauses and physical findings that increase risk of obstructive sleep apnea hypopnea (e.g., central obesity, crowded pharyngeal airway, elevated blood pressure) is important to reduce the chance of failure to diagnose this treatable condition.

Associated Features

Because of the frequency of nocturnal awakenings that occur with obstructive sleep apnea hypopnea, individuals may report symptoms of insomnia. Other common, though nonspecific, symptoms of obstructive sleep apnea hypopnea are heartburn, nocturia, morning headaches, dry mouth, erectile dysfunction, and reduced libido. Individuals may complain of difficulty breathing while lying supine or sleeping. Hypertension may occur in more than 60% of individuals with obstructive sleep apnea hypopnea.

Arterial blood gas measurements while the individual is awake are usually normal, but some individuals may demonstrate waking hypoxemia or hypercapnia. This pattern should alert the clinician to the possibility of coexisting lung disease or hypoventilation. Imaging procedures may reveal narrowing of the upper airway. Cardiac testing may show evidence of impaired ventricular function. Arrhythmias such as sinus pauses, frequent atrial and ventricular ectopic beats, or atrial fibrillation may be present during sleep. Individuals with severe nocturnal oxygen desaturation may also have elevated hemoglobin or hematocrit values.

Prevalence

Obstructive sleep apnea hypopnea is a very common disorder. Prevalence may be particularly high among men as compared with women, ranging from 2:1 to 4:1; older adults; and certain racial and ethnic groups. Prevalence varies cross-nationally, partly because of differences in assessment methods. Because the disorder is strongly associated with obesity, the rise in obesity rates has resulted in an increased prevalence of this disorder.

In the United States, 13% of men and 6% of women have polysomnographic evidence of 15 or more obstructive apneas or hypopneas per hour of sleep, and 14% of men and 5% of women have more than 5 obstructive apneas or hypopneas per hour of sleep, plus symptoms of daytime sleepiness. Gender differences decline in older age, possibly because of increased prevalence in females after menopause; postmenopausal females are 2.6–3.5 times more likely to have obstructive sleep apnea compared with premenopausal females.

In the general community, prevalence rates in the United States of undiagnosed obstructive sleep apnea hypopnea may be very high in elderly individuals. Obstructive sleep apnea also occurs in children, with an estimated prevalence of 1%–4%; there is no gender difference among prepubertal children. Children who are obese have higher rates.

Prevalence of obstructive sleep apnea appears to be higher among African Americans than among U.S. non-Latinx Whites. An increased prevalence among African Americans, American Indians, and Hispanics may be related to higher rates of obesity, which can be associated with differential exposure to social determinants of health, including food insecurity, food deserts, and limited access to safe and affordable places for physical activity.

Development and Course

The age distribution of obstructive sleep apnea hypopnea has several peaks. The first occurs in children ages 3–8 years when the nasopharynx may be compromised by a relatively large mass of tonsillar tissue compared with the size of the upper airway. With growth of the airway and regression of lymphoid tissue during later childhood, there is reduction in prevalence. However, with the increase of obesity in adolescents, a second peak in

prevalence occurs in that age group. Finally, as obesity prevalence continues to increase in midlife and women enter menopause, rates of obstructive sleep apnea hypopnea further increase. The course in older age is unclear; prevalence of the disorder may plateau after age 65 years, but in some individuals, severity may worsen with aging. Polysomnographic results must be interpreted in light of other clinical data. Significant clinical symptoms of insomnia or hypersomnia should be investigated regardless of the individual's age.

Obstructive sleep apnea hypopnea usually has an insidious onset, gradual progression, and persistent course. Typically, the loud snoring has been present for many years, often since childhood, but an increase in its severity may lead the individual to seek evaluation. Weight gain may precipitate an increase in symptoms. Although obstructive sleep apnea hypopnea can occur at any age, it most commonly manifests among individuals ages 40–60 years. Over 4–5 years, the average apnea hypopnea index increases in adults and older individuals by approximately two apneas or hypopneas per hour. The apnea hypopnea index is increased and incident obstructive sleep apnea hypopnea is greater among

individuals who are older, who are male, or who have a higher baseline body mass index (BMI) or increase their BMI over time. Spontaneous resolution of obstructive sleep apnea hypopnea has been reported with weight loss, particularly after bariatric surgery. In children, seasonal variation in obstructive sleep apnea hypopnea has been observed, as has improvement with overall growth.

In young children, the signs and symptoms of obstructive sleep apnea hypopnea may be more subtle than in adults, making diagnosis more difficult to establish. Polysomnography is useful in confirming diagnosis. Evidence of fragmentation of sleep on the polysomnogram may not be as apparent as in studies of older individuals, possibly because of the high homeostatic drive in young individuals. Symptoms such as snoring are usually parent-reported and thus have reduced sensitivity. Agitated arousals and unusual sleep postures, such as sleeping on the hands and knees, may occur. Nocturnal enuresis also may occur and should raise the suspicion of

obstructive sleep apnea hypopnea if it recurs in a child who was previously dry at night. Children may also manifest excessive daytime sleepiness, although this is not as common or pronounced as in adults. Daytime mouth breathing, difficulty in swallowing, and poor speech articulation are also common features in children. Children younger than 5 years more often present with nighttime symptoms, such as observed apneas or labored breathing, than with behavioral symptoms (i.e., the nighttime symptoms are more noticeable and more often bring the child to clinical attention). In children older than 5 years, daytime symptoms such as sleepiness and behavioral problems (e.g., impulsivity and hyperactivity), attention-deficit/hyperactivity disorder, learning difficulties, and morning headaches are more often the focus of concern. Children with obstructive sleep apnea hypopnea also may present with delayed growth, failure to thrive, and developmental delays. Although obesity is a less important risk factor in young children, it nevertheless contributes to the occurrence of obstructive sleep apnea.

Risk and Prognostic Factors

Genetic and physiological. The major risk factors for obstructive sleep apnea hypopnea are obesity and male sex. Others include maxillary-mandibular retrognathia or micrognathia, positive family history of sleep apnea, genetic syndromes that reduce upper airway patency (e.g., Down syndrome, Treacher Collins syndrome), adenotonsillar hypertrophy (especially in young children), menopause (in females), and various endocrine syndromes (e.g., acromegaly). Compared with premenopausal females, males are at increased risk for obstructive sleep apnea hypopnea, possibly reflecting the influences of sex hormones on ventilatory control and body fat distribution, as well as gender differences in airway structure. Medications for mental disorders and medical conditions that tend to induce somnolence may worsen the course of apnea symptoms if these medications are not managed carefully.

Obstructive sleep apnea hypopnea has a strong genetic basis, as evidenced by the significant familial aggregation of the apnea hypopnea index. The prevalence of obstructive sleep apnea hypopnea is approximately twice as high among the first-degree relatives of probands

with obstructive sleep apnea hypopnea as compared with members of control families. One-third of the variance in the apnea hypopnea index is explained by shared familial factors. Although genetic markers with diagnostic or prognostic value are not yet available for use, eliciting a family history of obstructive sleep apnea hypopnea should increase the clinical suspicion for the disorder.

Culture-Related Diagnostic Issues

There is a potential for sleepiness and fatigue to be reported differently across cultures. In some groups, snoring may be considered a sign of normal health and thus may not trigger concerns, leading to underdiagnosis.

Sex- and Gender-Related Diagnostic Issues

Menopause, pregnancy, and polycystic ovarian syndrome increase the risk of obstructive sleep apnea in females. The transition from premenopause to postmenopause is associated with increased severity of obstructive sleep apnea. Women may more commonly report fatigue, lack of energy, or insomnia rather than sleepiness and may underreport snoring.

Diagnostic Markers

Polysomnography provides quantitative data on frequency of sleep-related respiratory disturbances and associated changes in oxygen saturation and sleep continuity. Validated sleep measures (e.g., multiple sleep latency test, maintenance of wakefulness test) may identify sleepiness.

Functional Consequences of Obstructive Sleep Apnea Hypopnea

More than 50% of individuals with moderate to severe obstructive sleep apnea hypopnea report symptoms of daytime sleepiness. A twofold increased risk of occupational accidents has been reported in association with symptoms of snoring and sleepiness. Motor vehicle crashes also have been reported to be as much as sevenfold higher among individuals with

elevated apnea hypopnea index values. Clinicians should be cognizant of state government requirements for reporting this disorder, especially in relationship to commercial drivers. Reduced scores on measures of health-related quality of life are common in individuals with obstructive sleep apnea hypopnea. Although the greatest functional impact is observed in the "vitality" domain, severe obstructive sleep apnea negatively affects general health and physical and social functioning as well.

Differential Diagnosis

Primary snoring and other sleep disorders. Individuals with obstructive sleep apnea hypopnea must be differentiated from individuals with primary snoring (i.e., otherwise asymptomatic individuals who snore and do not have abnormalities on overnight polysomnography). Individuals with obstructive sleep apnea hypopnea may additionally report nocturnal gasping and choking, which can be confused with the presence of asthma or gastroesophageal reflux. The presence of sleepiness or other daytime symptoms not explained by other etiologies suggests the diagnosis of obstructive sleep apnea hypopnea, but this differentiation requires polysomnography. Definitive differential diagnosis between hypersomnolence disorder, central sleep apnea, sleep-related hypoventilation, and obstructive sleep apnea hypopnea also requires polysomnographic studies.

Obstructive sleep apnea hypopnea must be differentiated from other causes of sleepiness, such as narcolepsy, hypersomnolence disorder, insufficient sleep, and circadian rhythm sleep disorders. Obstructive sleep apnea hypopnea can be differentiated from narcolepsy by the absence of cataplexy, sleep-related hallucinations, and sleep paralysis and by the presence of loud snoring, gasping during sleep, or observed apneas in sleep. Daytime sleep episodes in narcolepsy are characteristically shorter, more refreshing, and more often associated with dreaming. Obstructive sleep apnea hypopnea shows characteristic apneas and hypopneas and oxygen desaturation during nocturnal polysomnographic studies. Narcolepsy results in multiple sleep-onset rapid eye movement (REM) periods during the MSLT. Narcolepsy, like obstructive sleep apnea hypopnea, may be associated with obesity, and some individuals have concurrent narcolepsy

and obstructive sleep apnea hypopnea. A diagnosis of narcolepsy does not exclude the diagnosis of obstructive sleep apnea hypopnea, as the two conditions may co-occur.

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Central sleep apnea. Central sleep apnea can be distinguished from obstructive sleep apnea by the presence of repetitive apneas or hypopneas due to reduction or absence of respiratory effort on polysomnogram recording. Snoring may be present, although it may be less prominent than observed in obstructive sleep apnea hypopnea or absent altogether. Individuals with central sleep apnea often exhibit fragmented sleep and may also complain of daytime sleepiness. Central sleep apnea is seen most commonly in individuals with congestive heart failure (Cheyne-Stokes breathing) or neurological disease, or those using opioid medications.

Insomnia disorder. For individuals complaining of difficulty initiating or maintaining sleep or early-morning awakenings, insomnia disorder can be differentiated from obstructive sleep apnea hypopnea by the absence of snoring and the absence of the history, signs, and symptoms characteristic of the latter disorder. However, insomnia and obstructive sleep apnea hypopnea may coexist, and if so, both disorders may need to be addressed concurrently to improve sleep.

Panic attacks. Nocturnal panic attacks may include symptoms of gasping or choking during sleep that may be difficult to distinguish clinically from obstructive sleep apnea hypopnea. However, the lower frequency of episodes, intense autonomic arousal, and lack of excessive sleepiness differentiate nocturnal panic attacks from obstructive sleep apnea hypopnea. Polysomnography (or OCST) in individuals with nocturnal panic attacks does not reveal the typical pattern of apneas or oxygen desaturation characteristic of obstructive sleep apnea hypopnea. Individuals with obstructive sleep apnea hypopnea do not provide a history of daytime panic attacks.

Nocturnal asthma. Nocturnal asthma can often cause sudden awakening from sleep with symptoms of gasping or choking that are indistinguishable from dyspneic episodes resulting from obstructive sleep apnea. However, a

history of asthma is generally present and polysomnography (or OCST) does not find evidence of apneas, hypopneas, or oxygen desaturation indicative of obstructive apnea. Nevertheless, nocturnal asthma and obstructive sleep apnea can coexist, and this can make it difficult to determine the relative contributions of each condition.

Attention-deficit/hyperactivity disorder. Attention-deficit/hyperactivity disorder in children may include symptoms of inattention, academic impairment, hyperactivity, and internalizing behaviors, all of which may also be symptoms of childhood obstructive sleep apnea hypopnea. The presence of other symptoms and signs of childhood obstructive sleep apnea hypopnea (e.g., labored breathing or snoring during sleep and adenotonsillar hypertrophy) would suggest the presence of obstructive sleep apnea hypopnea. Obstructive sleep apnea hypopnea and attention-deficit/hyperactivity disorder may commonly co-occur, and there may be causal links between them; therefore, risk factors such as enlarged tonsils, obesity, or a family history of sleep apnea may help alert the clinician to their co-occurrence.

Substance/medication-induced insomnia or hypersomnia. Substance use and substance withdrawal (including medications) can produce insomnia or hypersomnia. A careful history is usually sufficient to identify the relevant substance/medication, and follow-up shows improvement of the sleep disturbance after discontinuation of the substance/medication. In other cases, the use of a substance/medication (e.g., alcohol, barbiturates, benzodiazepines, opiates) has been shown to exacerbate obstructive sleep apnea hypopnea. An individual with symptoms and signs consistent with obstructive sleep apnea hypopnea should receive that diagnosis, even in the presence of concurrent substance use that is exacerbating the condition.

Comorbidity

Systemic hypertension, coronary artery disease, heart failure, stroke, diabetes, and increased mortality are consistently associated with obstructive sleep apnea hypopnea. Risk

estimates vary from 30% to as much as 300% for moderate to severe obstructive sleep apnea hypopnea. Obstructive sleep apnea and cardiovascular disease are strongly related, and treatment of obstructive sleep apnea reduces morbidity and mortality of cardiovascular disease. Ethnic and racialized groups that have not received adequate health care may be at higher risk for undetected cardiovascular risk factors associated with obstructive sleep apnea. Evidence of pulmonary hypertension and right heart failure (e.g., cor pulmonale, ankle edema, hepatic congestion) is rare in obstructive sleep apnea hypopnea and when present indicates either very severe disease or associated hypoventilation or cardiopulmonary comorbidities. Obstructive sleep apnea hypopnea also may occur with increased frequency in association with a number of medical or neurological conditions (e.g., cerebrovascular disease, Parkinson's disease). Physical findings reflect the co-occurrence of these conditions.

As many as one-third of individuals referred for evaluation of obstructive sleep apnea hypopnea report symptoms of depression, with as many of 10% having depression scores consistent with moderate to severe depression. Severity of obstructive sleep apnea hypopnea, as measured by the apnea hypopnea index, has been found to be correlated with severity of symptoms of depression. This association may be stronger in men than in women.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders,* 3rd Edition (ICSD-3), differentiates 11 subtypes of "sleep-related breathing disorders," including central sleep apneas (CSAs) (e.g., primary CSA, CSA due to a medical/neurological condition, CSA due to a substance or medication), obstructive sleep apnea (adult and pediatric), and sleep-related hypoventilation disorders.

Central Sleep Apnea

Diagnostic Criteria

A. Evidence by polysomnography of five or more central apneas per hour of sleep.

B. The disorder is not better explained by another current sleep disorder.

Specify whether:

G47.31 Idiopathic central sleep apnea: Characterized by repeated episodes of apneas and hypopneas during sleep caused by variability in respiratory effort but without evidence of airway obstruction.

R06.3 Cheyne-Stokes breathing: A pattern of periodic crescendo-decrescendo variation in tidal volume that results in central apneas and hypopneas at a frequency of at least five events per hour, accompanied by frequent arousal.

G47.37 Central sleep apnea comorbid with opioid use: The pathogenesis of this subtype is attributed to the effects of opioids on the respiratory rhythm generators in the medulla as well as the differential effects on hypoxic versus hypercapnic respiratory drive.

Coding note (for G47.37 code only): When an opioid use disorder is present, first code the opioid use disorder: F11.10 mild opioid use disorder or F11.20 moderate or severe opioid use disorder; then code G47.37 central sleep apnea comorbid with opioid use. When an opioid use disorder is not present (e.g., after a one-time heavy use of the substance), code only G47.37 central sleep apnea comorbid with opioid use.

Specify current severity:

Severity of central sleep apnea is graded according to the frequency of the breathing disturbances as well as the extent of associated oxygen desaturation and sleep fragmentation

that occur as a consequence of repetitive respiratory disturbances.

Subtypes

There are several subtypes of central sleep apnea. Idiopathic central sleep apnea (alternatively termed *primary central sleep apnea*) and central sleep apnea with Cheyne-Stokes breathing are characterized by increased gain of the ventilatory control system, also referred to as *high loop gain*, which leads to instability in ventilation and $PaCO_2$ levels. This instability is termed *periodic breathing* and can be recognized by hyperventilation alternating with hypoventilation. Individuals with these disorders typically have pCO_2 levels while awake that are slightly hypocapnic or normocapnic. Central sleep apnea may also manifest during initiation of treatment of obstructive sleep apnea hypopnea (termed *treatment-emergent central sleep apnea*) or may occur in association with obstructive sleep apnea hypopnea syndrome. The occurrence of central sleep apnea in association with obstructive sleep apnea is also considered to be due to high loop gain. In contrast, the pathogenesis of central sleep apnea comorbid with opioid use has been attributed to the effects of opioids on the respiratory rhythm generators in the medulla as well as to its differential effects on hypoxic versus hypercapnic respiratory drive. These individuals may have elevated pCO_2 levels while awake. Individuals receiving chronic methadone maintenance therapy have been noted to have increased somnolence and depression, although the role of opioid-induced breathing disorders in causing these problems has not been studied. Similarly, central apnea due to a medical disorder without Cheyne-Stokes breathing is a result of a pathological process that affects brain-stem ventilatory control centers.

Specifiers

An increase in the central apnea index (i.e., number of central apneas per hour of sleep) reflects an increase in severity of central sleep apnea. Sleep continuity and quality may be markedly impaired with reductions in restorative stages of non–rapid eye movement (NREM) sleep (i.e., decreased slow-wave sleep [stage N3]). In individuals with severe Cheyne-

Stokes breathing, the pattern can also be observed during resting wakefulness, a finding that is thought to be a prognostic marker for increased mortality.

Diagnostic Features

Central sleep apnea disorders are characterized by repeated episodes of apneas and hypopneas during sleep caused by variability in respiratory effort. These are disorders of ventilatory control in which respiratory events occur in a periodic or intermittent pattern. *Idiopathic central sleep apnea* is characterized by sleepiness, insomnia, and awakenings due to dyspnea in association with five or more central apneas per hour of sleep. Individuals with heart failure, stroke, or renal failure who have central sleep apnea typically have a breathing pattern called *Cheyne-Stokes breathing*, which is characterized by a pattern of periodic crescendo-decrescendo variation in tidal volume that results in central apneas and hypopneas occurring at a frequency of at least five events per hour. Events are often associated with arousal, but arousals are not required for the diagnosis. Central sleep apnea observed at high altitude occurs after ascent to high altitude, generally at least 2,500 meters above sea level. Central and obstructive sleep apneas may coexist; a diagnosis of central sleep apnea hypopnea requires that central events be > 50% of the total number of respiratory events.

Alterations in neuromuscular control of breathing can occur in association with medications or substances, which can cause or exacerbate impairments of respiratory rhythm and ventilation. Individuals taking medications with these effects may have a sleep-related breathing disorder that could contribute to sleep disturbances and symptoms such

as sleepiness, confusion, and depression. Specifically, *chronic use of long-acting opioid medications* is often associated with impairment of respiratory control leading to central sleep apnea.

Associated Features

Individuals with central sleep apnea hypopneas may present with sleepiness or insomnia. They may have complaints of sleep fragmentation, including awakening with dyspnea. Some individuals are asymptomatic. Obstructive sleep apnea hypopnea can coexist with Cheyne-Stokes breathing, and thus snoring and abruptly terminated obstructive events may be observed during sleep.

Physical findings seen in individuals with a Cheyne-Stokes breathing pattern relate to its risk factors. Findings consistent with heart failure, such as jugular venous distension, S3 heart sound, lung crackles, and lower-extremity edema, may be present.

Prevalence

The prevalence of idiopathic central sleep apnea is unknown but thought to be rare. The prevalence of Cheyne-Stokes breathing is high in individuals with depressed cardiac ventricular ejection fraction. In individuals with an ejection fraction of < 45%, the prevalence has been reported to range from 15% to 44%. The gender ratio for prevalence in North America, Europe, and Australia is even more highly skewed toward men than for obstructive sleep apnea hypopnea. Prevalence increases with age, and most individuals with the disorder are older than 60 years. Cheyne-Stokes breathing occurs in approximately 20% of individuals with acute stroke as assessed in Barcelona and Toronto. Central sleep apnea comorbid with opioid use occurs in approximately 24% of individuals taking opioids chronically for nonmalignant pain and similarly in individuals receiving methadone maintenance therapy as seen in several high-income countries. Higher opioid doses are associated with greater severity, especially at morphine-equivalent daily dosages > 200 mg. In children assessed in France and Canada, the prevalence ranges from 4% to 6%.

Development and Course

Polysomnography parameters for diagnosing central sleep apnea are different for children than for adults and comprise any of the following: 1) cessation of airflow and respiratory effort for more than 20 seconds, two breath cycles that are associated with an arousal from sleep, or > 3%

oxygen desaturation; or 2) two breath cycles that are associated with bradycardia.

The onset of Cheyne-Stokes breathing appears tied to the development of heart failure. The Cheyne-Stokes breathing pattern is associated with oscillations in heart rate, blood pressure, and oxygen desaturation, and elevated sympathetic nervous system activity that can promote progression of heart failure. The clinical significance of Cheyne-Stokes breathing in the setting of stroke is not known, but Cheyne-Stokes breathing may be a transient finding that resolves with time after acute stroke. Central sleep apnea comorbid with opioid use has been documented with chronic use (i.e., several months).

Risk and Prognostic Factors

Genetic and physiological. Cheyne-Stokes breathing is frequently present in individuals with heart failure. The coexistence of atrial fibrillation further increases risk, as do older age and male sex. Cheyne-Stokes breathing is also seen in association with acute stroke and possibly renal failure. The underlying ventilatory instability in the setting of heart failure has been attributed to increased ventilatory chemosensitivity and hyperventilation due to pulmonary vascular congestion and circulatory delay. Central sleep apnea is seen in individuals taking long-acting opioids. In children, central sleep apnea can be found in

individuals with congenital abnormalities, particularly Arnold-Chiari malformation, or comorbid medical conditions such as gastroesophageal reflux. Rarely, central sleep apnea resulting from a congenital condition may not manifest until adulthood (e.g., Arnold-Chiari malformation and congenital central hypoventilation).

Diagnostic Markers

Polysomnography is used to characterize the breathing characteristics of each breathing-related sleep disorder subtype. Central sleep apneas are recorded when periods of breathing cessation for longer than 10 seconds

occur. Cheyne-Stokes breathing is characterized by a pattern of periodic crescendo-decrescendo variation in tidal volume that results in central apneas and hypopneas occurring at a frequency of at least five events per hour with the number of central apneas and hypopneas > 50% of the total number of apneas and hypopneas. The cycle length of Cheyne-Stokes breathing (or time from end of one central apnea to the end of the next apnea) is about 60 seconds.

Functional Consequences of Central Sleep Apnea

Idiopathic central sleep apnea has been reported to cause symptoms of disrupted sleep, including insomnia and sleepiness. Cheyne-Stokes breathing with comorbid heart failure has been associated with excessive sleepiness, fatigue, and insomnia, although many individuals may be asymptomatic. Coexistence of heart failure and Cheyne-Stokes breathing may be associated with increased cardiac arrhythmias and increased mortality or cardiac transplantation. Individuals with central sleep apnea comorbid with opioid use may present with symptoms of sleepiness or insomnia.

Differential Diagnosis

Idiopathic central sleep apnea must be distinguished from other breathing-related sleep disorders, other sleep disorders, and medical conditions and mental disorders that cause sleep fragmentation, sleepiness, and fatigue. This is achieved using polysomnography.

Other breathing-related sleep disorders and sleep disorders. Central sleep apnea can be distinguished from obstructive sleep apnea hypopnea by the presence of at least five central apneas per hour of sleep. These conditions may co-occur, but central sleep apnea is considered to predominate when central respiratory events are > 50% of the total number of respiratory events.

Cheyne-Stokes breathing can be distinguished from other mental disorders, including other sleep disorders, and other medical conditions that cause sleep fragmentation, sleepiness, and fatigue based on the presence of a predisposing condition (e.g., heart failure or stroke) and signs and polysomnographic evidence of the characteristic breathing pattern.

Polysomnographic respiratory findings can help distinguish Cheyne-Stokes breathing from insomnia due to other medical conditions. For example, central sleep apnea due to high-altitude periodic breathing has a pattern that resembles Cheyne-Stokes breathing but has a shorter cycle time, occurs only at high altitude, and is not associated with heart failure.

Central sleep apnea comorbid with opioid use can be differentiated from other types of breathing-related sleep disorders based on the use of long-acting opioid medications in conjunction with polysomnographic evidence of central apneas and periodic or ataxic breathing. It can be distinguished from insomnia due to drug or substance use based on polysomnographic evidence of central sleep apnea.

Comorbidity

Central sleep apnea disorders are frequently present in users of long-acting opioids, such as methadone. Individuals taking these medications have a breathing-related sleep disorder that could contribute to sleep disturbances and symptoms such as sleepiness,

confusion, and depression. While the individual is asleep, breathing patterns such as central apneas, periodic apneas, and ataxic breathing may be observed. Obstructive sleep apnea hypopnea may coexist with central sleep apnea, and features consistent with this condition can also be present (see “Obstructive Sleep Apnea Hypopnea” earlier in this chapter). Cheyne-Stokes breathing is more commonly observed in association with conditions that include heart failure, stroke, and renal failure and is seen more frequently in individuals with atrial fibrillation. Individuals with Cheyne-Stokes breathing are more likely to be older, to be male, and to have lower weight than individuals with obstructive sleep apnea hypopnea.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 3rd Edition (ICSD-3), includes eight subtypes of central sleep apnea (central sleep apnea with

Cheyne-Stokes breathing, central apnea due to a medical disorder without Cheyne-Stokes breathing, central sleep apnea due to high-altitude periodic breathing, central sleep apnea due to a medication or substance, primary central sleep apnea, primary central sleep apnea of infancy, primary central sleep apnea of prematurity, and treatment-emergent central sleep apnea). As in DSM-5, most of these diagnoses require a frequency of 5 or more central events per hour of sleep. In addition, ICSD-3 criteria also require the presence of signs or symptoms (e.g., complaints of insomnia or daytime sleepiness). Central events must constitute at least 50% of the total number of apneas and hypopneas. Primary central sleep apnea of infancy and primary central sleep apnea of prematurity have their own distinct criteria sets that differ from adult forms of central sleep apnea.

Sleep-Related Hypoventilation

Diagnostic Criteria

A. Polysomnograpy demonstrates episodes of decreased respiration associated with elevated CO_2 levels. (**Note:** In the absence of objective measurement of CO_2, persistent low levels of hemoglobin oxygen saturation unassociated with apneic/hypopneic events may indicate hypoventilation.)

B. The disturbance is not better explained by another current sleep disorder.

Specify whether:

G47.34 Idiopathic hypoventilation: This subtype is not attributable to any readily identified condition.

G47.35 Congenital central alveolar hypoventilation: This subtype is a rare congenital disorder in which the individual typically presents in the perinatal period with shallow breathing, or cyanosis and apnea during sleep.

G47.36 Comorbid sleep-related hypoventilation: This subtype occurs as a consequence of a medical condition, such as a pulmonary disorder (e.g., interstitial lung disease, chronic obstructive pulmonary disease) or a neuromuscular or chest wall disorder (e.g., muscular dystrophies, postpolio syndrome, cervical spinal cord injury, kyphoscoliosis), or medications (e.g., benzodiazepines, opiates). It also occurs with obesity (obesity hypoventilation disorder), where it reflects a combination of increased work of breathing due to reduced chest wall compliance and ventilation-perfusion mismatch and variably reduced ventilatory drive. Such individuals usually are characterized by body mass index of greater than 30 and hypercapnia during wakefulness (with a pCO_2 of greater than 45), without other evidence of hypoventilation.

Specify current severity:

Severity is graded according to the degree of hypoxemia and hypercarbia present during sleep and evidence of end organ impairment due to these abnormalities (e.g., right-sided heart failure). The presence of blood gas abnormalities during wakefulness is an indicator of greater severity.

Subtypes

Subtypes of sleep-related hypoventilation include the following:

- *Idiopathic hypoventilation*, also referred to as *idiopathic central alveolar hypoventilation*, is characterized by reduction of tidal volume and elevated CO_2 during sleep, in the absence of any identifiable comorbidity that would account for the hypoventilation.
- *Congenital central alveolar hypoventilation* is a rare disorder associated with mutation of the gene *PHOX2B*. It typically manifests at birth.

- *Comorbid sleep-related hypoventilation* is due to one of numerous potential comorbidities, including pulmonary disease (e.g., chronic obstructive pulmonary disease [COPD]), chest wall abnormalities (e.g., kyphoscoliosis), neuromuscular disease (e.g., amyotrophic lateral sclerosis), and obesity (referred to as obesity hypoventilation), as well as use of medications or substances, especially opioids.

Diagnostic Features

Sleep-related hypoventilation can occur independently or, more frequently, comorbid with medical or neurological disorders, medication use, or substance use disorder. Although symptoms are not mandatory to make this diagnosis, individuals often report excessive daytime sleepiness, frequent arousals and awakenings during sleep, morning headaches, and insomnia complaints.

Associated Features

Individuals with sleep-related hypoventilation can present with sleep-related complaints of insomnia or sleepiness. Episodes of orthopnea can occur in individuals with diaphragm weakness. Headaches upon awakening may be present. During sleep, episodes of shallow breathing may be observed, and obstructive sleep apnea hypopnea or central sleep apnea may coexist. Consequences of ventilatory insufficiency, including pulmonary hypertension, cor pulmonale (right heart failure), polycythemia, and neurocognitive dysfunction, can be present. With progression of ventilatory insufficiency, blood gas abnormalities extend into wakefulness. Features of a medical condition causing sleep-related hypoventilation can also be present. Episodes of hypoventilation may be associated with frequent arousals or bradytachycardia. Individuals may complain of excessive sleepiness and insomnia or morning headaches or may present with findings of neurocognitive dysfunction or depression. Hypoventilation may not be present during wakefulness.

Prevalence

Idiopathic sleep-related hypoventilation in adults is very uncommon. The prevalence of congenital central alveolar hypoventilation is unknown, but

the disorder is rare. Comorbid sleep-related hypoventilation (i.e., hypoventilation comorbid with other conditions, such as COPD, neuromuscular disorders, or obesity) is more common.

The prevalence of comorbid sleep-related hypoventilation due to obesity in the general population is estimated to be approximately 0.14%–0.6% based on national obesity rates and prevalence of obstructive sleep apnea across several countries. Increasing rates of obesity are associated with increasing prevalence of comorbid sleep-related hypoventilation

due to obesity. In individuals referred to a sleep clinic who have a body mass index > 35 kg/m^2, prevalence may be as high as 42%.

Development and Course

Idiopathic sleep-related hypoventilation is thought to be a slowly progressive disorder of respiratory impairment. When sleep-related hypoventilation disorder occurs comorbidly with other disorders (e.g., COPD, neuromuscular disorders, obesity), disease severity reflects the severity of the underlying condition, and the disorder progresses as the condition worsens. Complications such as pulmonary hypertension, cor pulmonale, cardiac dysrhythmias, polycythemia, neurocognitive dysfunction, and worsening respiratory failure can develop with increasing severity of blood gas abnormalities.

Congenital central alveolar hypoventilation usually manifests at birth with shallow, erratic, or absent breathing. This disorder can also manifest during infancy, childhood, and adulthood because of variable penetrance of the *PHOX2B* mutation.

Risk and Prognostic Factors

Environmental. Ventilatory drive can be reduced in individuals who are using central nervous system depressants, including benzodiazepines, opiates, and alcohol.

Genetic and physiological. Idiopathic sleep-related hypoventilation is associated with reduced ventilatory drive due to a blunted

chemoresponsiveness to CO_2 (reduced respiratory drive; i.e., “won’t breathe”), reflecting underlying neurological deficits in centers governing the control of ventilation. More commonly, sleep-related hypoventilation is comorbid with another medical condition, such as a pulmonary disorder, a neuromuscular or chest wall disorder, or hypothyroidism, or with use of medications (e.g., benzodiazepines, opiates). In these conditions, the hypoventilation may be a consequence of increased work of breathing and/or impairment of respiratory muscle function (i.e., “can’t breathe”) or reduced respiratory drive.

Neuromuscular disorders influence breathing through impairment of respiratory motor innervation or respiratory muscle function. They include conditions such as amyotrophic lateral sclerosis, spinal cord injury, diaphragmatic paralysis, myasthenia gravis, Lambert-Eaton syndrome, toxic or metabolic myopathies, postpolio syndrome, and Charcot-Marie-Tooth syndrome.

Congenital central alveolar hypoventilation is a genetic disorder attributable to mutations of *PHOX2B,* a gene that is crucial for the development of the embryonic autonomic nervous system and neural crest derivatives. Children with congenital central alveolar hypoventilation show blunted ventilatory responses to hypercapnia, especially in non–rapid eye movement sleep.

Sex- and Gender-Related Diagnostic Issues

Gender distributions for sleep-related hypoventilation occurring in association with comorbid conditions reflect the gender distributions of the comorbid conditions. For example, COPD is more frequently present in men and with increasing age. Contrary to previous data, obesity hypoventilation is now thought to occur equally between genders, and in some studies there may be even a slightly greater prevalence in women.

Diagnostic Markers

Sleep-related hypoventilation is diagnosed using polysomnography, which demonstrates sleep-related hypoxemia and hypercapnia that is not better explained by another breathing-related sleep disorder. The documentation

of 1) increased arterial pCO_2 levels to > 55 mmHg during sleep or 2) a ≥ 10-mmHg increase in pCO_2 levels (to a level that also exceeds

50 mmHg) during sleep in comparison to awake supine values, in each case exceeding 10 minutes' duration, is the gold standard for diagnosis. However, obtaining arterial blood gas determinations during sleep is impractical, and non-invasive measures of pCO_2 have not been adequately validated during sleep and are not widely used during polysomnography in adults. Prolonged and sustained decreases in oxygen saturation (oxygen saturation of < 90% for more than 5 minutes with a nadir of at least 85%, or oxygen saturation of < 90% for at least 30% of sleep time) in the absence of evidence of upper airway obstruction are often used as an indication of sleep-related hypoventilation; however, this finding is not specific, as there are other potential causes of hypoxemia, such as that due to lung disease.

Children with congenital central alveolar hypoventilation are more likely to have disorders of the autonomic nervous system, Hirschsprung's disease, neural crest tumors, and characteristic box-shaped face (i.e., the face is short relative to its width).

Functional Consequences of Sleep-Related Hypoventilation

The consequences of sleep-related hypoventilation are related to the effects of chronic exposure to hypercapnia and hypoxemia. These blood gas derangements cause vasoconstriction of the pulmonary vasculature leading to pulmonary hypertension, which, if severe, can result in right-sided heart failure (cor pulmonale). Hypoxemia can lead to dysfunction of organs such as the brain, blood, and heart, leading to outcomes such as cognitive dysfunction, polycythemia, and cardiac arrhythmias. Hypercapnia can depress ventilatory drive, leading to progressive respiratory failure.

Differential Diagnosis

Other medical conditions affecting ventilation. In adults, the idiopathic variety of sleep-related hypoventilation is very uncommon and is determined by

excluding the presence of lung diseases, skeletal malformations, neuromuscular disorders, and other medical and neurological disorders or medications that affect ventilation. Sleep-related hypoventilation must be distinguished from other causes of sleep-related hypoxemia, such as that due to lung disease.

Other breathing-related sleep disorders. Sleep-related hypoventilation can be distinguished from obstructive sleep apnea hypopnea and central sleep apnea based on clinical features and findings on polysomnography. Sleep-related hypoventilation typically shows more sustained periods of oxygen desaturation rather than the periodic episodes seen in obstructive sleep apnea hypopnea and central sleep apnea. Obstructive sleep apnea hypopnea and central sleep apnea also show a pattern of discrete episodes of repeated airflow reductions that can be absent in sleep-related hypoventilation. However, both obstructive and central apneas and hypopneas can occur in association with sleep-related hypoventilation. In obesity hypoventilation, most individuals will have comorbid obstructive sleep apnea.

Comorbidity

Sleep-related hypoventilation often occurs in association with a pulmonary disorder (e.g., interstitial lung disease, COPD), with a neuromuscular or chest wall disorder (e.g., muscular dystrophies, post-polio syndrome, cervical spinal cord injury, kyphoscoliosis), with obesity, or, most relevant to the clinician, with medication use (e.g., benzodiazepines, opiates). Congenital central alveolar hypoventilation often occurs in association with autonomic dysfunction and may occur in association with Hirschsprung's disease. COPD, a disorder of lower airway obstruction usually associated with cigarette smoking, can result in sleep-related hypoventilation and hypoxemia. The presence of coexisting obstructive sleep apnea

hypopnea is thought to exacerbate hypoxemia and hypercapnia during sleep and wakefulness. The relationship between congenital central alveolar hypoventilation and idiopathic sleep-related hypoventilation is unclear; in

some individuals, idiopathic sleep-related hypoventilation may represent cases of late-onset congenital central alveolar hypoventilation.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 3rd Edition (ICSD-3), recognizes six subtypes of sleep hypoventilation disorders. Congenital central alveolar hypoventilation syndrome and idiopathic hypoventilation (idiopathic central alveolar hypoventilation in the ICSD-3) are identically classified in DSM-5 and ICSD-3. However, the ICSD-3 subtypes obesity hypoventilation syndrome, sleep-related hypoventilation due to a medication or substance, and sleep-related hypoventilation due to a medical disorder are subsumed under comorbid sleep-related hypoventilation in DSM-5. The subtype late-onset central hypoventilation with hypothalamic dysfunction is not in DSM-5. The DSM-5 approach to classification reflects the frequent co-occurrence of disorders that lead to hypoventilation and hypoxemia. In contrast, the classification used in ICSD-3 reflects evidence that there are distinct sleep-related pathogenetic processes leading to hypoventilation.

Circadian Rhythm Sleep-Wake Disorders

Diagnostic Criteria

A. A persistent or recurrent pattern of sleep disruption that is primarily due to an alteration of the circadian system or to a misalignment between the endogenous circadian rhythm and the sleep-wake schedule required by an individual's physical environment or social or professional schedule.

B. The sleep disruption leads to excessive sleepiness or insomnia, or both.

C. The sleep disturbance causes clinically significant distress or impairment in social, occupational, and other important areas of functioning.

Specify whether:

G47.21 Delayed sleep phase type: A pattern of delayed sleep onset and awakening times, with an inability to fall asleep and awaken at a desired or conventionally acceptable earlier time.

Specify if:

Familial: A family history of delayed sleep phase is present.

Specify if:

Overlapping with non-24-hour sleep-wake type: Delayed sleep phase type may overlap with another circadian rhythm sleep-wake disorder, non-24-hour sleep-wake type.

G47.22 Advanced sleep phase type: A pattern of advanced sleep onset and awakening times, with an inability to remain awake or asleep until the desired or conventionally acceptable later sleep or wake times.

Specify if:

Familial: A family history of advanced sleep phase is present.

G47.23 Irregular sleep-wake type: A temporally disorganized sleep-wake pattern, such that the timing of sleep and wake periods is variable throughout the 24-hour period.

G47.24 Non-24-hour sleep-wake type: A pattern of sleep-wake cycles that is not synchronized to the 24-hour

environment, with a consistent daily drift (usually to later and later times) of sleep onset and wake times.

G47.26 Shift work type: Insomnia during the major sleep period and/or excessive sleepiness (including inadvertent sleep) during the major awake period associated with a shift work schedule (i.e., requiring unconventional work hours).

G47.20 Unspecified type

Specify if:

Episodic: Symptoms last at least 1 month but less than 3 months.

Persistent: Symptoms last 3 months or longer.

Recurrent: Two or more episodes occur within the space of 1 year.

Delayed Sleep Phase Type

Diagnostic Features

The delayed sleep phase type is based primarily on a history of a delay in the timing of the major sleep period (usually more than 2 hours) in relation to the desired sleep and wake-up time, resulting in symptoms of insomnia and excessive sleepiness. When allowed to set their own schedule, individuals with delayed sleep phase type exhibit normal sleep quality and duration for age. Symptoms of sleep-onset insomnia, difficulty waking in the morning, and excessive sleepiness early in the day are prominent.

Associated Features

Common associated features of delayed sleep phase type include a history of mental disorders or a concurrent mental disorder. Extreme and prolonged difficulty awakening with morning confusion is also common. Insomnia disorder may develop as a result of maladaptive behaviors that impair sleep and increase arousal because of repeated attempts to fall asleep at an earlier time.

Prevalence

The prevalence of delayed sleep phase type is highest in adolescents and young adults, with rates estimated between 3.3% and 4.6% in Norway and Sweden. Studies of adult prevalence yield significantly lower rates, estimated to be 0.2%–1.7% in Norway and New Zealand. Although the prevalence of familial delayed sleep phase type has not been established, a family history of delayed sleep phase is often present in individuals with delayed sleep phase type.

Development and Course

Course is persistent, with intermittent exacerbations throughout adulthood in some individuals. Although age at onset is variable, symptoms begin typically in adolescence and early adulthood and persist for several months to years before diagnosis is established. Severity may decrease with age. Relapse of symptoms is common.

Clinical expression may vary across the life span depending on social, school, and work obligations. Exacerbation is usually triggered by a change in work or school schedule that requires an early rise time. Individuals who can alter their work schedules to accommodate the delayed circadian sleep and wake timing can experience remission of symptoms.

Increased prevalence in adolescence may be a consequence of both physiological and behavioral factors. Hormonal changes may be involved specifically, as delayed sleep phase is associated with the onset of puberty. Thus, delayed sleep phase type in adolescents should be differentiated from the common delay in the timing of circadian rhythms in this age group. In the familial form, the course is persistent and may not improve significantly with age.

Risk and Prognostic Factors

Genetic and physiological. Predisposing factors may include a longer than average circadian period, changes in light sensitivity, and impaired homeostatic sleep drive. Some individuals with delayed sleep phase type may be hypersensitive to evening light, which can serve as a delay signal to

the circadian clock, or they may be hyposensitive to morning light such that its phase-advancing effects are reduced. Genetic factors may play a role in the pathogenesis of familial and sporadic forms of delayed sleep phase type. A study of unrelated families showing strong heritability of the disorder described a mutation in the clock gene, *CRY1*, occurring in about 0.6% of the population, which results in increased inhibition of transcription of the activator clock genes, *CLOCK* and *BMAL1*.

Diagnostic Markers

Confirmation of the diagnosis includes a complete history and use of a sleep diary or actigraph (i.e., a wrist-worn motion detector that monitors motor activity for prolonged periods; if measured for at least 7 days, motor activity can be used as a proxy for sleep-wake patterns). The period covered should include weekends, when social and occupational obligations are less strict, to ensure that the individual exhibits a consistently delayed sleep-wake pattern. The most commonly available laboratory-derived phase marker is salivary dim light melatonin onset (DLMO) time. However, not all individuals with diagnosed delayed sleep phase exhibit delayed DLMO. An investigation of rigorously diagnosed individuals found that only 57% exhibited physiological phase delays (as gauged by a DLMO time occurring subsequent to the desired bedtime), whereas the remaining 43% had DLMO times that occurred before the desired bedtimes. As noted above, behavior, rather than circadian physiological alteration, may play a more predominant role in the latter (earlier DLMO) group. Given this, phase markers may ultimately demonstrate more value for optimization of treatment timing and/or as a measure of treatment response.

Functional Consequences of Delayed Sleep Phase Type

Excessive early day sleepiness is prominent. Extreme and prolonged difficulty awakening with morning confusion (i.e., sleep inertia) is also common. The severity of insomnia and excessive sleepiness symptoms varies substantially among individuals and largely depends on the occupational and social demands on the individual.

Differential Diagnosis

Normative variations in sleep. Delayed sleep phase type must be distinguished from "normal" sleep patterns in which a person has a late schedule that does not cause personal, social, or occupational distress (most commonly seen in adolescents and young adults).

Other sleep disorders. Insomnia disorder and other circadian rhythm sleep-wake disorders should be included in the differential. Excessive sleepiness may also be caused by other sleep disturbances, such as breathing-related sleep disorders, insomnias, restless legs syndrome, and medical, neurological, and mental disorders. Overnight polysomnography may help in evaluating for other comorbid sleep disorders, such as sleep apnea. The circadian nature of delayed sleep phase type, however, should differentiate it from other disorders with similar complaints.

Comorbidity

Delayed sleep phase type is associated with depressive disorders, personality disorders, somatic symptom disorder or illness anxiety disorder, obsessive-compulsive disorder, attention-deficit/hyperactivity disorder, and autism spectrum disorder. In addition, comorbid sleep disorders, such as insomnia disorder, restless legs syndrome, and sleep apnea, as

well as depressive and bipolar disorders and anxiety disorders, can exacerbate symptoms of insomnia and excessive sleepiness. Delayed sleep phase type may overlap with another circadian rhythm sleep-wake disorder, non-24-hour sleep-wake type. Sighted individuals with non-24-hour sleep-wake type disorder commonly also have a history of delayed circadian sleep phase.

Advanced Sleep Phase Type

Specifiers

The presence of a family history of advanced sleep phase type may be indicated with the specifier "familial." In the familial form, specific

mutations demonstrate an autosomal dominant mode of inheritance, the course is persistent, and the severity of symptoms may increase with age. The prevalence of familial advanced sleep phase type has not been established.

Diagnostic Features

Advanced sleep phase type is characterized by sleep-wake times that are several hours earlier than desired or conventional times. Diagnosis is based primarily on a history of an advance in the timing of the major sleep period (usually more than 2 hours) in relation to the desired sleep and wake-up time, with symptoms of early-morning insomnia and excessive daytime sleepiness. When allowed to set their schedule, individuals with advanced sleep phase type will exhibit normal sleep quality and duration for age.

Associated Features

Individuals with advanced sleep phase type are “morning types,” having earlier sleep-wake times, with the timing of circadian biomarkers such as melatonin and core body temperature rhythms occurring 2–4 hours earlier than normal. When required to keep a conventional schedule requiring a delay of bedtime, these individuals will continue to have an early rise time, leading to persistent sleep deprivation and daytime sleepiness. Use of hypnotics or alcohol to combat sleep-maintenance insomnia and stimulants to reduce daytime sleepiness may lead to substance abuse in these individuals.

Prevalence

The estimated prevalence of advanced sleep phase type is approximately 1% in middle-age adults in the United States. Sleep-wake times and circadian phase advance in older individuals probably account for the higher prevalence in this population.

Development and Course

Onset is usually in late adulthood, although in the familial form, onset can be earlier (during childhood or early adulthood). The course is typically

persistent, lasting more than 3 months, but the severity may increase depending on work and social schedules. The advanced sleep phase type is more common in older adults.

Clinical expression may vary across the life span depending on social, school, and work obligations. Individuals who can alter their work schedules to accommodate the advanced circadian sleep and wake timing can experience remission of symptoms. Increasing age tends to advance the sleep phase. However, it is unclear whether the common age-associated advanced sleep phase type is attributable solely to a change in circadian timing (as seen in the familial form) or also to age-related changes in the homeostatic regulation of sleep, resulting in earlier awakening. Severity, remission, and relapse of symptoms are dependent on adherence to behavioral and environmental treatments designed to control sleep and wake structure and light exposure.

Risk and Prognostic Factors

Environmental. Decreased late afternoon/early evening exposure to light and/or increased exposure to early-morning light because of early-morning awakening can increase the risk of advanced sleep phase type by advancing circadian rhythms. By going to bed early, these individuals are not exposed to light in the phase delay region of the curve, resulting in perpetuation of advanced phase. In familial advanced sleep phase type, a shortening of the endogenous circadian period can result in an advanced sleep phase, although circadian period does not appear to systematically decrease with age.

Genetic and physiological. Advanced sleep phase type has demonstrated an autosomal dominant mode of inheritance, including a *PER2* gene mutation causing hypophosphorylation of the PER2 protein and a missense mutation in *CKI*.

Diagnostic Markers

A sleep diary and actigraphy are used as diagnostic markers, as described earlier for delayed sleep phase type.

Functional Consequences of Advanced Sleep Phase Type

Excessive sleepiness associated with advanced sleep phase can have a negative effect on cognitive performance, social interaction, and safety. Use of wake-promoting agents to combat sleepiness later in the day or sedatives to inhibit early-morning awakening may increase potential for substance abuse.

Differential Diagnosis

Normal variations in sleep. Behavioral factors such as irregular sleep schedules, voluntary early awakening, and exposure to light in the early morning should be considered, particularly in older adults.

Other disorders that cause early-morning awakening. Careful attention should be taken to rule out other sleep-wake disorders (e.g., insomnia disorder), other mental disorders (e.g., depressive disorders, bipolar disorders), and medical conditions that can cause early-morning awakening.

Comorbidity

Repetitive attempts to resume sleep and the development of maladaptive cognitions and sleep-related behaviors may result in the development of a comorbid insomnia disorder that requires clinical attention.

Irregular Sleep-Wake Type

Diagnostic Features

Irregular sleep-wake type is characterized by a lack of discernible sleep-wake circadian rhythm. The diagnosis of irregular sleep-wake type is based primarily on a history of symptoms of insomnia at night (during the usual sleep period) and excessive sleepiness (napping) during the day. There is no major sleep period, and sleep and wake periods across 24 hours are fragmented, with sleep fragmented into at least three periods during the 24-hour day. The longest sleep period tends to occur between 2:00 A.M. and 6:00 A.M. and is usually < 4 hours.

Associated Features

A history of isolation or reclusion may occur in association with the disorder and contribute to the symptoms via a lack of external stimuli to help entrain a normal pattern.

Individuals or their caregivers report frequent naps throughout the day. Irregular sleep-wake type is most commonly associated with neurodegenerative disorders, such as major neurocognitive disorder, and many neurodevelopmental disorders in children.

Prevalence

Prevalence of irregular sleep-wake type in the general population is unknown.

Development and Course

The course of irregular sleep-wake type is persistent. Age at onset is variable, but the disorder is more common in older adults.

Risk and Prognostic Factors

Environmental. Decreased exposure to environmental light and structured daytime activity can be associated with a low-amplitude circadian rhythm. Hospitalized individuals are especially prone to such weak external entraining stimuli, and even outside the hospital setting, individuals with major neurocognitive disorder are exposed to significantly less bright light.

Genetic and physiological. Neurodegenerative disorders, such as Alzheimer's disease, Parkinson's disease, and Huntington's disease, and neurodevelopmental disorders in children increase the risk for irregular sleep-wake type.

Diagnostic Markers

A detailed sleep history and a sleep diary (by a caregiver) or actigraphy help confirm the irregular sleep-wake pattern.

Functional Consequences of Irregular Sleep-Wake Type

Lack of a clearly discernible major sleep and wake period in irregular sleep-wake type results in insomnia or excessive sleepiness at irregular times of the day. Disruption of the caregiver's sleep also often occurs and is an important consideration.

Differential Diagnosis

Normative variations in sleep. Irregular sleep-wake type should be distinguished from a voluntary irregular sleep-wake schedule and poor sleep hygiene, which can result in insomnia and excessive sleepiness.

Other medical conditions and mental disorders. Other causes of insomnia and daytime sleepiness, including comorbid medical conditions and mental disorders or medication, should be considered.

Comorbidity

Irregular sleep-wake type is often comorbid with neurodegenerative and neurodevelopmental disorders, such as major neurocognitive disorder, intellectual developmental disorder (intellectual disability), and traumatic brain injury. It is also comorbid with other medical conditions and mental disorders in which there is social isolation and/or lack of light and structured activities.

Non-24-Hour Sleep-Wake Type

Diagnostic Features

The diagnosis of non-24-hour sleep-wake type is based primarily on a history of symptoms of insomnia or excessive sleepiness related to abnormal synchronization between the 24-hour light-dark cycle and the endogenous circadian rhythm. Individuals typically present with periods of insomnia, excessive sleepiness, or both, which alternate with short

asymptomatic periods. Starting with the asymptomatic period, when the individual's sleep phase is aligned to the external environment, sleep latency will gradually increase and the individual will complain of sleep-onset insomnia. As the sleep phase continues to drift so that sleep time is now in the daytime, the individual will have trouble staying awake during the day and will complain of sleepiness. Because the circadian period is not aligned to the external 24-hour environment, symptoms will depend on when an individual tries to sleep in relation to the circadian rhythm of sleep propensity.

Associated Features

Non-24-hour sleep-wake type is most common among blind or visually impaired individuals who have decreased light perception. In sighted individuals, there is often a history of delayed sleep phase and of decreased exposure to light and structured social and physical activity. Sighted individuals with non-24-hour sleep-wake type also demonstrate increased sleep duration.

Prevalence

Prevalence of non-24-hour sleep-wake type in the general population is unclear, but the disorder appears rare in sighted individuals. The prevalence in blind individuals in the United States is estimated to be 50%.

Development and Course

Course of non-24-hour sleep-wake type is persistent, with intermittent remission and exacerbations as a result of changes in work and social schedules throughout the life span. Age at onset is variable, depending on the onset of visual impairment. In sighted individuals, because of the overlap with delayed sleep phase type, non-24-hour sleep-wake type may develop in adolescence or early adulthood. Remission and relapse of symptoms in blind and sighted individuals largely depend on adherence to treatments designed to control sleep and wake structure and light exposure.

Clinical expression may vary across the life span depending on social, school, and work obligations. In adolescents and adults, irregular sleep-

wake schedules and exposure to light or lack of light at critical times of the day can exacerbate the effects of sleep loss and disrupt circadian entrainment. Consequently, symptoms of insomnia, daytime sleepiness, and school, occupational, and interpersonal functioning may worsen.

Risk and Prognostic Factors

Environmental. In sighted individuals, decreased exposure or sensitivity to light and social and physical activity cues may contribute to a free-running circadian rhythm. With the high frequency of mental disorders involving social isolation and cases of non-24-hour sleep-wake type developing after a change in sleep habits (e.g., night shift work, job loss), behavioral factors in combination with physiological tendency may precipitate and perpetuate this disorder in sighted individuals. Hospitalized individuals with neurological and psychiatric disorders can become insensitive to social cues, predisposing them to the development of non-24-hour sleep-wake type.

Genetic and physiological. Blindness is a risk factor for non-24-hour sleep-wake type. Non-24-hour sleep-wake type has been associated with traumatic brain injury.

Diagnostic Markers

Diagnosis is confirmed by history and sleep diary or actigraphy for an extended period. Sequential measurement of phase markers (e.g., melatonin) can help determine circadian phase in both sighted and blind individuals.

Functional Consequences of Non-24-Hour Sleep-Wake Type

Complaints of insomnia (sleep onset and sleep maintenance), excessive sleepiness, or both are prominent. The unpredictability of sleep and wake times (typically a daily delay drift) results in difficulty attending school or maintaining a steady job and may increase potential for social isolation.

Differential Diagnosis

Other circadian rhythm sleep-wake disorders. In sighted individuals, non-24-hour sleep-wake type should be differentiated from delayed sleep phase type, as individuals with delayed sleep phase type may display a similar progressive delay in sleep period for several days.

Depressive disorders. Depressive disorders may result in similar circadian dysregulation and symptoms.

Comorbidity

Blindness is often comorbid with non-24-hour sleep-wake type, as are depressive and bipolar disorders with social isolation.

Shift Work Type

Diagnostic Features

Diagnosis is primarily based on a history of the individual working outside of the normal 8:00 A.M. to 6:00 P.M. daytime window (particularly at night) on a regularly scheduled (i.e., non-overtime) basis. Symptoms of excessive sleepiness at work, and impaired sleep at home, on a persistent basis are prominent. Presence of both sets of symptoms are usually required for a diagnosis of shift work type. Typically, when the individual reverts to a day-work routine, symptoms resolve.

Prevalence

The prevalence of shift work type is unclear, but the disorder is estimated to affect 5%–10% of the night worker population in the United States (16%–20% of the workforce). Prevalence rises with advancement into middle age and beyond.

Development and Course

Shift work type can appear in individuals of any age but is more prevalent in individuals older than 50 years and typically worsens with the passage of time if the disruptive work schedule persists. Although older adults may

show similar rates of circadian phase adjustment to a change in routine as do younger adults, they appear to experience significantly more sleep disruption as a consequence of the circadian phase shift.

Risk and Prognostic Factors

Temperamental. Predisposing factors include a morning-type disposition and a need for long (i.e., more than 8 hours) sleep durations in order to feel well rested.

Environmental. Trying to balance strong competing social and domestic needs (e.g., in parents of young children) can lead to the development of the shift work type. Persons who are able to commit to a nocturnal lifestyle, with few competing day-oriented demands, appear at lower risk for shift work type.

Genetic and physiological. Because shift workers are more likely than day workers to be obese, obstructive sleep apnea may be present and may exacerbate the symptoms.

Diagnostic Markers

A history and sleep diary or actigraphy may be useful in diagnosis, as discussed earlier for delayed sleep phase type.

Functional Consequences of Shift Work Type

Individuals with shift work type not only may perform poorly at work but also appear to be at risk for accidents both at work and on the drive home. Individuals with a history of bipolar disorder are particularly vulnerable to shift work type–related episodes of mania resulting from missed nights of sleep. Shift work type often results in interpersonal problems.

Differential Diagnosis

Normative variations in sleep with shift work. The diagnosis of shift work type, as opposed to the “normal” difficulties of shift work, depends to some

extent on the severity of symptoms and/or level of distress experienced by the individual.

Other sleep disorders. The presence of shift work type symptoms even when the individual is able to live on a day-oriented routine for several weeks at a time may suggest the presence of other sleep disorders, such as sleep apnea, insomnia, and narcolepsy, which should be ruled out.

Jet lag. Individuals who travel across many time zones on a very frequent basis may experience effects similar to those experienced by individuals with shift work type who work rotating shifts. The distinction should be clear, based on the travel history.

Comorbidity

Shift work type has been associated with increased alcohol use disorder, other substance use disorders, and depression. A variety of physical health disorders (e.g., gastrointestinal disorders, cardiovascular disease, diabetes, cancer) have been found to be associated with prolonged exposure to shift work.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 3rd Edition, circadian rhythm sleep-wake disorders closely parallel DSM-5 but also include jet lag type.

Parasomnias

Parasomnias are disorders characterized by abnormal behavioral, experiential, or physiological events occurring in association with sleep, specific sleep stages, or sleep-wake transitions. The most common parasomnias are non–rapid eye movement (NREM) sleep arousal disorders and rapid eye movement (REM) sleep behavior disorder. These conditions each have distinct pathophysiology, clinical characteristics, and prognostic

and therapeutic considerations discussed in the following sections specific to each disorder.

Non–Rapid Eye Movement Sleep Arousal Disorders

Diagnostic Criteria

A. Recurrent episodes of incomplete awakening from sleep, usually occurring during the first third of the major sleep episode, accompanied by either one of the following:
   1. **Sleepwalking:** Repeated episodes of rising from bed during sleep and walking about. While sleepwalking, the individual has a blank, staring face; is relatively unresponsive to the efforts of others to communicate with him or her; and can be awakened only with great difficulty.
   2. **Sleep terrors:** Recurrent episodes of abrupt terror arousals from sleep, usually beginning with a panicky scream. There is intense fear and signs of autonomic arousal, such as mydriasis, tachycardia, rapid breathing, and sweating, during each episode. There is relative unresponsiveness to efforts of others to comfort the individual during the episodes.

B. No or little (e.g., only a single visual scene) dream imagery is recalled.

C. Amnesia for the episodes is present.

D. The episodes cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).

F. Coexisting mental disorders and medical conditions do not explain the episodes of sleepwalking or sleep terrors.

Specify whether:

F51.3 Sleepwalking type
Specify if:

With sleep-related eating

With sleep-related sexual behavior (sexsomnia)

F51.4 Sleep terror type

Diagnostic Features

The essential feature of non–rapid eye movement (NREM) sleep arousal disorders is the repeated occurrence of incomplete arousals, usually beginning during the first third of the major sleep episode (Criterion A), that typically are brief, lasting 1–10 minutes, but may be protracted, lasting up to 1 hour. The maximum duration of an event is unknown. The eyes are typically open during these events. Many individuals exhibit both subtypes of arousal (i.e., sleepwalking type and sleep terror type) on different occasions, which underscores the unitary underlying pathophysiology. The subtypes reflect varying degrees of simultaneous occurrence of wakefulness and NREM sleep, resulting in complex behaviors arising from sleep with varying degrees of conscious awareness, motor activity, and autonomic activation.

The essential feature of *sleepwalking* is repeated episodes of complex motor behavior initiated during sleep, including rising from bed and walking about (Criterion A1). Sleepwalking episodes begin during any stage of NREM sleep, most commonly during slow-wave sleep and therefore most often occurring during the first third of the night. During episodes, the individual has reduced alertness and responsiveness, a blank stare, and relative unresponsiveness to communication with others or efforts by others to awaken the individual. If awakened during the episode (or on awakening the following morning), the

individual has limited recall for the episode. After the episode, there may initially be a brief period of confusion or difficulty orienting, followed by full recovery of cognitive function and appropriate behavior.

The essential feature of *sleep terrors* is the repeated occurrence of precipitous awakenings from sleep, usually beginning with a panicky scream or cry (Criterion A2). Sleep terrors usually begin during the first third of the major sleep episode and last 1–10 minutes, but they may last considerably longer, particularly in children. The episodes are accompanied by impressive autonomic arousal and behavioral manifestations of intense fear. During an episode, the individual is difficult to awaken or comfort. If the individual awakens after the sleep terror, little or none of the dream, or only fragmentary, single images, are recalled. During a typical episode of sleep terrors, the individual abruptly sits up in bed screaming or crying, with a frightened expression and autonomic signs of intense anxiety (e.g., tachycardia, rapid breathing, sweating, dilation of the pupils). The individual may be inconsolable and is usually unresponsive to the efforts of others to awaken or comfort him or her. Sleep terrors are also called “night terrors” or “pavor nocturnus.”

For both subtypes of NREM sleep arousal disorders, the determination of “disorder” depends on a number of factors, which may vary on an individual basis and will depend on the frequency of events, potential for violence or injurious behaviors, embarrassment, or disruption/distress of other household members. Severity determination is best made based on the nature or consequence of the behaviors rather than simply on frequency.

Associated Features

Sleepwalking episodes can include a wide variety of behaviors. Episodes may begin with confusion: the individual may simply sit up in bed, look about, or pick at the blanket or sheet. This behavior then becomes progressively complex. The individual may actually leave the bed and walk into closets, out of the room, and even out of buildings. Individuals may use the bathroom, eat, talk, or engage in more complex behaviors. Running and frantic attempts to escape some apparent threat can also occur. Most

behaviors during sleepwalking episodes are routine and of low complexity. However, cases of unlocking doors and even operating machinery (driving an automobile) have been reported. Sleepwalking can also include inappropriate behavior (e.g., commonly, urinating in a closet or wastebasket). Most episodes last for several minutes to a half hour but may be more protracted. Inasmuch as sleep is a state of relative analgesia, painful injuries sustained during sleepwalking may not be appreciated until awakening after the fact.

There are two “specialized” forms of sleepwalking: sleep-related eating behavior and sleep-related sexual behavior (sexsomnia or sleep sex). Individuals with *sleep-related eating* experience unwanted recurrent episodes of eating with varying degrees of amnesia, ranging from no awareness to full awareness without the ability to avoid or stop eating. During these episodes, inappropriate foods or even nonfood items (i.e., candy wrappers, small food boxes, or even small toys) may be ingested. Individuals with sleep-related eating disorder may find evidence of their eating only the next morning. In *sexsomnia,* varying degrees of sexual activity (e.g., masturbation, fondling, groping, sexual intercourse) occur as complex behaviors arising from sleep without conscious awareness. This condition is more common in males and may result in serious interpersonal relationship problems or medicolegal consequences.

During a typical episode of sleep terrors, there is often a sense of overwhelming dread, with a compulsion to escape. Although fragmentary vivid dream images may occur, a storylike dream sequence (as in nightmares) is not reported. Most commonly, the individual does not awaken fully, but returns to sleep and has amnesia for the episode on awakening the next morning. Usually only one episode will occur on any one night. Occasionally, several episodes may occur at intervals throughout the night. These events rarely arise during daytime naps.

Prevalence

Isolated or infrequent NREM sleep arousal behaviors are very common in the general population worldwide. From 10% to 30% of children have had

at least one episode of sleepwalking, and the cross-national 12-month prevalence rate for sleepwalking in children is approximately 5%. The prevalence of sleepwalking episodes (not sleepwalking disorder) is estimated to be 12%–14.5% of children in Canada and 1.0%–7.0% among adults in the United Kingdom, with weekly to monthly episodes occurring in just 0.5%–0.7% of adults. Estimates for the lifetime prevalence of sleepwalking overall range from approximately 6.9% to 29.2% around the world, with a past-year prevalence of sleepwalking of 1.5%–3.6% in adults.

The prevalence of sleep terror disorder in the general population is unknown. The prevalence of sleep terror episodes (as opposed to sleep terror disorder, in which there is recurrence and distress or impairment) is approximately 34.4%–36.9% at 18 months of age and 19.7% at 30 months of age in Canadian toddlers, and 2.2% in Canadian and British adults.

Development and Course

NREM sleep arousal disorders occur most commonly in childhood and diminish in frequency with increasing age. Sleepwalking and sleep terrors are frequently outgrown following infancy and childhood and become less frequent by adolescence, with remission rates between 50% and 65%; for individuals ages 10–18 years, frequency is reported at 1.1% for sleepwalking and 0.6% for sleep terrors.

Violent or sexual activity during sleepwalking episodes is more likely to occur in adults. The onset of sleepwalking in adults with no history of sleepwalking as children should prompt a search for specific etiologies, such as obstructive sleep apnea, nocturnal seizures, or effect of medication.

Older children and adults may provide a more detailed recollection of fearful images associated with sleep terrors than do younger children, who are more likely to have complete amnesia or report only a vague sense of fear.

Risk and Prognostic Factors

Environmental. Sedative use, sleep deprivation, sleep-wake schedule disruptions, fatigue, and physical or emotional stress increase the likelihood of episodes. Fever and sleep deprivation can produce an increased frequency of NREM sleep arousal disorders.

Genetic and physiological. A family history of sleepwalking or sleep terrors may occur in up to 80% of individuals who sleepwalk. The risk for sleepwalking is further increased (to as much as 60% of offspring) when both parents have a history of the disorder. Familial aggregation of sleep terrors and sleepwalking has been described, as parental sleepwalking history predicts incident and persistent sleep terrors in their offspring. Individuals with sleep terrors frequently have a positive family history of either sleep terrors or sleepwalking, with as high as a 10-fold increase in the prevalence of the disorder among first-degree biological relatives. Sleep terrors are much more common in monozygotic twins as compared with dizygotic twins. The exact mode of inheritance is unknown.

Sex- and Gender-Related Diagnostic Issues

Eating during sleepwalking episodes is more commonly seen in women. Sleepwalking occurs more often in girls during childhood but more often in men during adulthood.

Among children, sleep terrors are more common in boys than in girls. Among adults, they are equally common in men and women.

Diagnostic Markers

NREM sleep arousal disorders arise from any stage of NREM sleep but most commonly from deep NREM sleep (slow-wave sleep). They are most likely to appear in the first third of the night and do not commonly occur during daytime naps. During the episode, the polysomnogram may be obscured with movement artifact. In the absence of such artifact, the electroencephalogram (EEG) may show a variety of patterns, including continuation of rhythmic delta activity into awakening, indicating partial or incomplete arousal; or alternatively, theta, alpha, or mixed frequency EEG activity may be seen during the episode, with frequent mixed slow/mixed frequency EEG arousals during slow-wave sleep being more common in individuals with NREM sleep arousal disorders than in control subjects. In distinction to an epileptic seizure, NREM sleep parasomnia disorders of

arousal do not show features of spatiotemporal evolution of EEG rhythms during the episode.

Polysomnography in conjunction with audiovisual monitoring can be used to document episodes of sleepwalking. In the absence of actually capturing an event during a polysomnographic recording, there are no reliable polysomnographic features that can serve as a marker for sleepwalking. Sleep deprivation may increase the likelihood of capturing an event. As a group, individuals who sleepwalk show instability of deep NREM sleep, but the overlap in findings with persons who do not sleepwalk is great enough to preclude use of this indicator in establishing a diagnosis. Unlike arousals from REM sleep associated with nightmares, in which there is an increase in heart rate and respiration prior to the arousal, the NREM sleep arousals of sleep terrors begin precipitously from sleep, without anticipatory autonomic changes. The arousals are associated with impressive autonomic activity, with doubling or tripling of the heart rate. The pathophysiology is poorly understood, but there appears to be instability in the deeper stages of NREM sleep. Aside from capturing an event during a formal sleep study, there are no reliable polysomnographic indicators of the tendency to experience sleep terrors.

Functional Consequences of Non–Rapid Eye Movement Sleep Arousal Disorders

For the diagnosis of an NREM sleep arousal disorder to be made, the individual or household members must experience clinically significant distress or impairment, although such symptoms may occur occasionally in nonclinical populations and would be subthreshold for the diagnosis. Embarrassment concerning the episodes can impair social relationships. Social isolation or occupational difficulties can result. Uncommonly, NREM sleep arousal disorders may result in serious injury to the individual or to someone trying to console the individual. Injuries to others are confined to those in close proximity; individuals are not “sought out.” For individuals with sleep-related eating behaviors, unknowingly preparing or eating food during the sleep period may create problems such as poor diabetes control, weight gain, injury (cuts and burns), or consequences of

eating dangerous or toxic inedibles. NREM sleep arousal disorders may rarely result in violent or injurious behaviors with forensic implications.

Differential Diagnosis

Nightmare disorder. In contrast to individuals with NREM sleep arousal disorders, individuals with nightmare disorder typically awaken easily and completely, report vivid storylike dreams accompanying the episodes, and tend to have episodes later in the night. NREM sleep arousal disorders occur during NREM sleep, whereas nightmares usually occur during REM sleep. Parents of children with NREM sleep arousal disorders may misinterpret reports of fragmentary imagery as nightmares.

Breathing-related sleep disorders. Breathing disorders during sleep can also produce confusional arousals with subsequent amnesia. However, breathing-related sleep disorders are also characterized by symptoms of snoring, breathing pauses, and daytime sleepiness. In some individuals, a breathing-related sleep disorder may precipitate episodes of sleepwalking.

REM sleep behavior disorder. REM sleep behavior disorder may be difficult to distinguish from NREM sleep arousal disorders. REM sleep behavior disorder is characterized by episodes of prominent, complex movements, often involving personal injury arising from sleep. In contrast to NREM sleep arousal disorders, REM sleep behavior disorder occurs during REM sleep. Individuals with REM sleep behavior disorder may be awakened easily during an episode and report more detailed and vivid dream content than do individuals with NREM sleep arousal disorders. These individuals and/or their bed partners often report that they “act out dreams.”

Parasomnia overlap syndrome. Parasomnia overlap syndrome consists of clinical and polysomnographic features of both sleepwalking and REM sleep behavior disorder.

Sleep-related seizures. Some types of seizures can produce episodes of very unusual behaviors that occur predominantly or exclusively during sleep. Nocturnal seizures may closely mimic NREM sleep arousal disorders but tend to be more stereotypic in nature, occur multiple times nightly, and be

more likely to occur from daytime naps. In addition, seizures may arise from wakefulness, which does not occur with NREM sleep arousal disorders. The presence of sleep-related seizures does not preclude the presence of NREM sleep arousal disorders. When recurrent, sleep-related seizures are considered to be a form of epilepsy.

Alcohol-induced blackouts. Alcohol-induced blackouts may be associated with extremely complex behaviors in the absence of other suggestions of intoxication. They do not involve the loss of consciousness but rather reflect an isolated disruption of memory for events during a drinking episode. By history, these behaviors may be indistinguishable from those seen in NREM sleep arousal disorders.

Dissociative amnesia, with dissociative fugue. Dissociative fugue may be extremely difficult to distinguish from sleepwalking. Unlike all other parasomnias, nocturnal dissociative fugue arises from a period of wakefulness during sleep, rather than precipitously from sleep without intervening wakefulness. A history of recurrent childhood physical or sexual abuse is usually present (but may be difficult to obtain).

Malingering or other voluntary behavior. As with dissociative fugue, malingering or other voluntary behavior occurs during wakefulness.

Panic disorder. Panic attacks may also cause abrupt awakenings from deep NREM sleep accompanied by fearfulness, but these episodes produce rapid and complete awakening without the confusion, amnesia, or motor activity typical of NREM sleep arousal disorders.

Medication-induced complex behaviors. Behaviors similar to those in NREM sleep arousal disorders can be induced by use of, or withdrawal from, substances or medications (e.g., benzodiazepines, nonbenzodiazepine sedative-hypnotics, opiates, cocaine, nicotine, antipsychotics or other dopamine receptor–blocking agents, tricyclic antidepressants, chloral hydrate). Such behaviors may arise from the sleep period and may be extremely complex. The underlying pathophysiology appears to be a relatively isolated amnesia. In such cases, substance/medication-induced sleep disorder, parasomnia type, should be diagnosed (see “Substance/Medication-Induced Sleep Disorder” later in this chapter).

Night eating syndrome.

In contrast to the sleep-related eating form of sleepwalking, which is characterized by recurrent episodes of eating during incomplete arousals from sleep, night eating syndrome is considered to be an abnormality in the circadian rhythm of meal timing, with a normal circadian timing of sleep onset in which the individual wakes up in the middle of the night and overeats.

Comorbidity

Typically, sleepwalking in both children and adults is not associated with significant mental disorders. However, in adults, there is an association between sleepwalking and major depressive episodes and obsessive-compulsive disorder. Children or adults with sleep terrors may have elevated scores for depression and anxiety on personality inventories.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 3rd Edition, includes "confusional arousal, sleep terrors, and sleepwalking" as NREM sleep arousal disorders.

Nightmare Disorder

Diagnostic Criteria F51.5

A. Repeated occurrences of extended, extremely dysphoric, and well-remembered dreams that usually involve efforts to avoid threats to survival, security, or physical integrity and that generally occur during the second half of the major sleep episode.
B. On awakening from the dysphoric dreams, the individual rapidly becomes oriented and alert.

C. The sleep disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The nightmare symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication).

E. Coexisting mental disorders and medical conditions do not adequately explain the predominant complaint of dysphoric dreams.

Specify if:

During sleep onset

Specify if:

With mental disorder, including substance use disorders

With medical condition

With another sleep disorder

Coding note: The code F51.5 applies to all three specifiers. Code also the relevant associated mental disorder, medical condition, or other sleep disorder immediately after the code for nightmare disorder in order to indicate the association.

Specify if:

Acute: Duration of period of nightmares is 1 month or less.

Subacute: Duration of period of nightmares is greater than 1 month but less than 6 months.

Persistent: Duration of period of nightmares is 6 months or greater.

Specify current severity:

Severity can be rated by the frequency with which the nightmares occur:

Mild: Less than one episode per week on average.

Moderate: One or more episodes per week but less than nightly.

Severe: Episodes nightly.

Recording Procedures

The specifiers “with mental disorder, including substance use disorders”; “with medical condition”; and “with another sleep disorder” are available to allow the clinician to note clinically relevant comorbidities. In such cases, record F51.5 nightmare disorder with [name of comorbid condition(s) or disorder(s)] followed by the diagnostic code(s) for the comorbid conditions or disorders (e.g., F51.5 nightmare disorder with moderate alcohol use disorder and rapid eye movement sleep behavior disorder; F10.20 moderate alcohol use disorder; G47.52 REM sleep behavior disorder).

Diagnostic Features

Nightmares are typically lengthy, elaborate, story-like sequences of dream imagery that seem real and that incite anxiety, fear, or other dysphoric emotions. Nightmare content typically focuses on attempts to avoid or cope with imminent danger but may involve themes that evoke other negative emotions. Nightmares occurring after traumatic experiences may replicate the threatening situation (“replicative nightmares”), but most do not. On awakening, nightmares are well remembered and can be described in detail. They arise almost exclusively during REM sleep and can thus occur throughout sleep but are more likely in the second half of the major sleep episode when dreaming is longer and more intense. Factors that increase early-night REM intensity, such as sleep fragmentation or deprivation, jet lag, and medications that affect REM sleep, might facilitate nightmares earlier in the night, including at sleep onset.

Nightmares usually terminate with awakening and rapid return of full alertness. However, the dysphoric emotions may persist into wakefulness and contribute to difficulty returning to sleep and lasting daytime distress. Some nightmares, known as “bad dreams,” may not induce awakening and are recalled only later. If nightmares occur during sleep-onset REM periods (*hypnagogic*), the dysphoric emotion is frequently accompanied by an

awakening and being unable to move voluntarily (*sleep paralysis*), which may also occur in isolation without a preceding dream or nightmare.

Associated Features

Mild autonomic arousal, including sweating, tachycardia, and tachypnea, may characterize nightmares. Body movements and vocalizations are not characteristic because of REM sleep–related loss of skeletal muscle tone. When talking or emoting occurs in nightmare disorder, the vocal or motor behaviors are typically brief events terminating the nightmare. Distinct from such motor or vocal activity, true dream enactment behavior may occur when there is a loss of normal REM atonia (REM sleep behavior disorder).

Prevalence

Prevalence of nightmares during childhood is approximately 1%–5%. From 1.3% to 3.9% of parents report that their preschool children have nightmares “often” or “always.” Prevalence increases to 5.2% in children ages 5–15 years. Family history of nightmares, parasomnia symptoms, and daytime consequences of temper outbursts/mood disturbance and poor academic performance are associated with frequent nightmares during childhood and adolescence, with comorbid insomnia seen in approximately 20% of children with frequent nightmares. Among adults, prevalence of nightmares at least monthly is 6%. Among adults in several countries, prevalence of weekly nightmares is 2%–6%, whereas prevalence of frequent nightmares is 1%–5%. Estimates often combine idiopathic and posttraumatic nightmares indiscriminately.

Development and Course

Nightmares often begin between ages 3 and 6 years but reach a peak prevalence and severity in late adolescence or early adulthood. Nightmares most likely appear in children

exposed to acute or chronic psychosocial stressors and thus may not resolve spontaneously. In a minority, frequent nightmares persist into adulthood, becoming virtually a lifelong disturbance. Although specific nightmare content may reflect the individual's age, the essential features of the disorder are the same across age groups.

Risk and Prognostic Factors

Frequent nightmares in middle-age adults in the general community population have been shown in two studies in Hong Kong and Finland to be associated with low income, mood disturbance, insomnia or sleep-disordered breathing, and use of antidepressants or frequent heavy alcohol use.

Environmental. Sleep deprivation or fragmentation, and irregular sleep-wake schedules that alter the timing, intensity, or quantity of REM sleep can put individuals at risk for nightmares. Individuals who experience nightmares report more frequent past adverse events, but not necessarily trauma.

Genetic and physiological. Twin studies have identified genetic effects on the disposition to nightmares and their co-occurrence with other nocturnal behaviors (e.g., sleeptalking).

Course modifiers. Adaptive parental bedside behaviors, such as soothing the child following nightmares, may protect against developing chronic nightmares.

Culture-Related Diagnostic Issues

The significance attributed to nightmares may vary by culture, and sensitivity to such beliefs may facilitate disclosure. In several cultural contexts, nightmares may be viewed as important indicators of the individual's spiritual status or the condition of those who have died (e.g., among Indonesian civil war survivors, American Indian veterans, and Cambodian refugees). Frequent nightmares among Cambodian refugees are strongly associated with the presence of posttraumatic stress disorder (PTSD); assessment of the temporal sequence and severity of nightmares relative to other symptoms is needed to determine whether a separate diagnosis of nightmare disorder is warranted. Among Hmong immigrants to

the United States, frequent nightmares are more common than among non-Latinx Whites in the same region and are associated with traumatic experiences and other sleep disorders, such as sleep paralysis and restless sleep.

Sex- and Gender-Related Diagnostic Issues

Adult women report having nightmares more frequently than adult men, but this gender difference was not found in children and the elderly. Nightmare content differs by gender, with women tending to report themes of sexual harassment or of loved ones disappearing/dying, and men tending to report themes of physical aggression or war/terror.

Diagnostic Markers

Polysomnographic studies demonstrate abrupt awakenings from REM sleep, usually during the second half of the night, prior to report of a nightmare. Heart, respiratory, and eye movement rates may quicken or increase in variability before awakening. Nightmares following traumatic events may also arise during light non-REM (NREM) sleep, particularly stage 2 sleep (now called N2 sleep). The typical sleep of individuals with nightmares is mildly impaired (e.g., reduced efficiency, less slow-wave sleep, more awakenings), with more frequent periodic leg movements in sleep and relative sympathetic nervous system activation after REM sleep deprivation.

Association With Suicidal Thoughts or Behavior

Individuals with frequent nightmares are at substantially greater risk for suicidal thoughts or behavior, even when gender and mental illness are taken into account.

Functional Consequences of Nightmare Disorder

Nightmares cause more significant subjective distress than demonstrable social or occupational impairment. However, if awakenings are frequent or

result in sleep avoidance, individuals may experience excessive daytime sleepiness, poor concentration, depression, anxiety, or irritability. Frequent childhood nightmares (e.g., several per week) may cause significant distress to parents and children.

Differential Diagnosis

Sleep terror disorder. Both nightmare disorder and sleep terror disorder include awakenings or partial awakenings with fearfulness and autonomic activation, but the two disorders can be readily differentiated. Nightmares typically occur later in the night, during REM sleep, and produce vivid, story-like, and clearly recalled dreams; mild autonomic arousal; and complete awakenings. Sleep terrors typically arise in the first third of the night during deep NREM sleep (especially during stage 3 sleep, now called N3 sleep) and produce either no dream recall or images without an elaborate story-like quality. Sleep terrors are thought to be caused by partial awakenings intermixed with persisting sleep, with clinical manifestations of confusion, disorientation, and only partial responsiveness, and often with substantial autonomic arousal. There is usually amnesia for the event in the morning.

REM sleep behavior disorder. The presence of complex vocal and motor activity during frightening dreams should prompt further evaluation for REM sleep behavior disorder, which occurs more typically among late middle- and older-age men but may also affect women. Although nightmares are typically characteristic of REM sleep behavior disorder, unlike nightmare disorder, REM sleep behavior disorder is associated with dream enactment that may cause nocturnal injuries. If the nightmares precede REM sleep behavior disorder and warrant independent clinical attention, an additional diagnosis of nightmare disorder may be given.

Bereavement. Dysphoric dreams may occur during bereavement but typically involve loss and sadness and are followed by self-reflection and insight, rather than distress, on awakening.

PTSD or acute stress disorder. Nightmares in which the content or affect of the dream is related to a traumatic event may be a component of PTSD or acute stress disorder. An additional diagnosis of nightmare disorder may be

warranted if the severity or frequency of the nightmares necessitates independent clinical attention.

Narcolepsy. Nightmares are a frequent complaint in narcolepsy, but the presence of excessive sleepiness, with or without cataplexy, differentiates this condition from nightmare disorder.

Sleep-related seizures. Nocturnal seizures usually involve stereotyped motor activity. Associated nightmares, if recalled, are often also repetitively stereotyped in nature or reflect epileptogenic features such as the content of diurnal auras, phosphenes (visual sensations in the absence of light input), or ictal imagery.

Breathing-related sleep disorders. Breathing-related sleep disorders can lead to awakenings with autonomic arousal, but these are not usually accompanied by recall of nightmares.

Panic disorder. Panic attacks arising during sleep can produce abrupt awakenings with autonomic arousal and fearfulness, but nightmares are typically not reported and symptoms are similar to panic attacks arising during wakefulness.

Sleep-related dissociative disorders. Individuals may recall actual physical or emotional trauma as a “dream” during electroencephalography-documented awakenings.

Substance or medication use. Numerous substances/medications can precipitate nightmares, including dopaminergic drugs; β-adrenergic antagonists and other antihypertensives; amphetamine-type substances, cocaine, and other stimulants; antidepressants; smoking cessation aids; and melatonin. Withdrawal of REM sleep–suppressant medications

(e.g., antidepressants) and alcohol can produce REM sleep rebound accompanied by nightmares. If nightmares are sufficiently severe to warrant independent clinical attention, a diagnosis of substance/medication-induced sleep disorder should be considered.

Comorbidity

Nightmares may be comorbid with several medical conditions, including coronary heart disease, cancer, parkinsonism, and pain, and can accompany medical treatments, such as hemodialysis, or withdrawal from medications or substances of abuse. Nightmares frequently co-occur with other mental disorders, including PTSD, acute stress disorder, insomnia disorder, REM sleep behavior disorder, and psychotic, mood, anxiety, adjustment, and personality disorders, as well as with grief during bereavement. A concurrent nightmare disorder diagnosis should only be considered when independent clinical attention is warranted. These conditions should be listed with the appropriate comorbid category specifier (e.g., "with REM sleep behavior disorder"); see also "Recording Procedures."

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 3rd Edition, presents similar diagnostic criteria for nightmare disorder.

Rapid Eye Movement Sleep Behavior Disorder

Diagnostic Criteria — G47.52

A. Repeated episodes of arousal during sleep associated with vocalization and/or complex motor behaviors.

B. These behaviors arise during rapid eye movement (REM) sleep and therefore usually occur more than 90 minutes after sleep onset, are more frequent during the later portions of the sleep period, and uncommonly occur during daytime naps.

C. Upon awakening from these episodes, the individual is completely awake, alert, and not confused or disoriented.

D. Either of the following:

   1. REM sleep without atonia on polysomnographic recording.

2. A history suggestive of REM sleep behavior disorder and an established synucleinopathy diagnosis (e.g., Parkinson's disease, multiple system atrophy).

E. The behaviors cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (which may include injury to self or the bed partner).

F. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.

G. Coexisting mental disorders and medical conditions do not explain the episodes.

Diagnostic Features

The essential feature of rapid eye movement (REM) sleep behavior disorder is repeated episodes of vocalizations and/or complex motor behaviors arising from REM sleep (Criterion A). These behaviors often reflect motor responses to the content of action-filled or violent dreams of being attacked or trying to escape from a threatening situation, which may be termed *dream enacting behaviors*. The vocalizations are often loud, emotion-filled, and profane. These behaviors may be very bothersome to the individual and the bed partner and

may result in significant injury (e.g., falling, jumping, or flying out of bed; running, punching, thrusting, hitting, or kicking). However, individuals with REM sleep behavior disorder may also present with relatively subtle vocal or motor behaviors during REM sleep, which are typically not the primary presenting sleep complaint but manifest during history taking or polysomnography in sleep, neurological, and psychiatric clinical visits. Upon awakening, the individual is usually immediately awake, alert, and oriented (Criterion C) and is often able to recall dream mentation, which closely correlates with the observed behavior. The eyes typically remain

closed during these events. The presence of REM sleep without atonia during a polysomnogram is typically required for the diagnosis of REM sleep behavior disorder. Alternatively, if polysomnography has not been performed, a provisional diagnosis of probable REM sleep behavior disorder may be given if there is an established synucleinopathy diagnosis (e.g., Parkinson's disease, multiple system atrophy) and the history is suggestive of REM sleep behavior disorder (Criterion D). The diagnosis of REM sleep behavior disorder requires clinically significant distress or impairment (Criterion E); this determination will depend on a number of factors, including the frequency of events, the potential for violence or injurious behaviors, embarrassment, and distress in other household members.

Severity determination is best made based on the nature or consequence of the behavior rather than simply on frequency. Although the behaviors are typically prominent and violent, lesser behaviors may also occur.

Prevalence

The prevalence of REM sleep behavior disorder was approximately 1% in a middle- to older-age general population sample in Switzerland and approximately 2% in an elderly general population sample in South Korea. One prevalence study found an equal prevalence between men and women in individuals younger than 50 years, while another study reported a prevalence of just over 1% with no difference between men and women in a population with a mean age of 59 years. Prevalence in individuals with psychiatric disorders may be greater, possibly related to medications prescribed for the psychiatric disorder.

Development and Course

The onset of REM sleep behavior disorder may be gradual or rapid. Because of the very high association with the later appearance of an underlying neurodegenerative disorder, the neurological status of individuals with REM sleep behavior disorder should be closely monitored. In individuals with idiopathic REM sleep behavior disorder, the risk of developing a defined neurodegenerative disease, most often a synucleinopathy (i.e., Parkinson’s disease, major or mild neurocognitive

disorder with Lewy bodies, or multiple system atrophy), is approximately 75% within 10–15 years following diagnosis, with an annualized risk of approximately 6%–7% per year.

Symptoms in young individuals, particularly young women, should raise the possibility of narcolepsy; substance/medication-induced sleep disorder, parasomnia type; a brainstem lesion; or an autoimmune encephalopathy.

Culture-Related Diagnostic Issues

Chinese individuals diagnosed with REM sleep behavior disorder by a neurology service in Taiwan had similar clinical and laboratory characteristics as non-Latinx White individuals in the United States; however, they differed in having a higher rate of nocturnal wandering out of the bedroom and a lower rate of sleep-related injuries, possibly as a result of earlier detection by the family.

Sex- and Gender-Related Diagnostic Issues

REM sleep behavior disorder is more common in men older than 50 years, but increasingly this disorder is being identified in women and in younger individuals. Women are younger than men in age at onset and age at diagnosis.

Diagnostic Markers

Associated laboratory findings from polysomnography indicate increased tonic and/or phasic electromyographic activity during REM sleep, which is normally associated with muscle atonia. The increased muscle activity variably affects different muscle groups; more extensive electromyographic monitoring with arm electromyography (e.g., biceps brachii) should be considered because this measure is more specific for a REM sleep behavior disorder diagnosis. It is suggested that electromyographic monitoring also include the submentalis, bilateral flexor digitorum superficialis, and bilateral anterior tibialis muscle groups. Continuous video monitoring should accompany the polysomnography. Other polysomnographic findings

may include very frequent periodic and aperiodic extremity electromyography activity during non-REM (NREM) sleep. *REM sleep without atonia* is present in virtually all cases of REM sleep behavior disorder but may also be an asymptomatic polysomnographic finding. It is not known whether isolated REM sleep without atonia is a precursor to REM sleep behavior disorder, although a pilot study suggested that isolated REM sleep without atonia may also be associated with neurodegenerative markers (i.e., hyposmia, orthostatic hypotension, color vision loss) and that 7%–14% of individuals with isolated REM sleep without atonia later develop clinical REM sleep behavior disorder. Thresholds for normative REM sleep without atonia levels have also been published that may serve to distinguish borderline cases and those whose neurological status should be further monitored.

Functional Consequences of Rapid Eye Movement Sleep Behavior Disorder

The most serious consequences of REM sleep behavior disorder are the short-term risks for injury to the individual or bed partner related to attacks of dream enactment, and the long-term risk of developing a defined neurodegenerative disease. According to surveys of individuals and their bed partners, approximately 55% of individuals with REM sleep behavior disorder may experience injury as a consequence of their attacks, with 12% of injuries being serious (including long bone or rib fractures or subdural hematomas) and requiring medical attention.

Differential Diagnosis

Other parasomnias. Confusional arousals, sleepwalking, and sleep terrors can easily be confused with REM sleep behavior disorder. In general, these disorders occur in individuals younger than 50 years. Unlike REM sleep behavior disorder, they arise from NREM sleep and therefore tend to occur in the early portion of the sleep period. Awakening from a confusional arousal is associated with confusion, disorientation, and incomplete recall of dream mentation accompanying the behavior. Polysomnographic monitoring in the disorders of arousal generally reveals normal REM sleep atonia unless there is a comorbid parasomnia.

Medication-induced sleep disorder, parasomnia type. Many widely prescribed medications, including tricyclic antidepressants, selective serotonin reuptake inhibitors, and serotonin-norepinephrine reuptake inhibitors, may result in polysomnographic evidence of REM sleep without atonia and in frank REM sleep behavior disorder, which is diagnosed as a medication-induced sleep disorder, parasomnia type. It is not known whether the medications per se result in REM sleep without atonia and/or REM sleep behavior disorder, or whether they unmask an underlying predisposition.

Asymptomatic REM sleep without atonia. Clinical dream-enacting behaviors coupled with the polysomnographic finding of REM sleep without atonia is necessary for the diagnosis of REM sleep behavior disorder. REM sleep without atonia without a clinical history of dream-enacting behaviors is simply an asymptomatic polysomnographic observation with an as yet unknown clinical significance.

Nocturnal seizures. Nocturnal seizures may mimic REM sleep behavior disorder, but the behaviors characteristic of nocturnal seizures are generally stereotyped. Polysomnographic monitoring employing a full electroencephalographic seizure montage may differentiate the two. REM sleep without atonia is generally not present on polysomnographic monitoring in individuals with epilepsy.

Obstructive sleep apnea. Obstructive sleep apnea may result in vocalizations and motor behaviors that very closely resemble REM sleep behavior disorder, such as talking, shouting, gesturing, and punching, along with unpleasant dreams. Polysomnographic monitoring is necessary to differentiate between these two disorders. In REM sleep behavior disorder, the parasomnia symptoms occur during periods of REM sleep without atonia. In obstructive sleep apnea, the parasomnia symptoms only occur during arousals at the end of the obstructive sleep apneic events and resolve following effective treatment of the obstructive sleep apnea (continuous positive airway pressure). REM sleep without atonia is not typically observed in obstructive sleep apnea.

Other specified dissociative disorder (sleep-related psychogenic dissociative disorder).

Unlike virtually all other parasomnias, which arise precipitously from NREM or REM sleep, psychogenic dissociative behaviors arise from a period of well-defined wakefulness during the sleep period. Unlike REM sleep behavior disorder, this condition is more prevalent in young women.

Malingering. Many cases of malingering in which the individual reports problematic sleep movements mimic the clinical features of REM sleep behavior disorder, and polysomnographic documentation is mandatory.

Comorbidity

REM sleep behavior disorder is present concurrently in approximately 30% of patients with narcolepsy. When it occurs in narcolepsy, the demographics reflect the younger age range of narcolepsy, with equal frequency in men and women. Based on findings from individuals presenting to sleep clinics, most individuals (>70%) with initially "idiopathic" REM sleep behavior disorder will eventually develop a neurodegenerative disease—most notably, one of the synucleinopathies (Parkinson's disease, multiple system atrophy, or major or mild neurocognitive disorder with Lewy bodies). REM sleep behavior disorder often predates any other sign of these disorders by many years (often more than a decade).

Relationship to International Classification of Sleep Disorders

REM sleep behavior disorder is virtually identical to REM sleep behavior disorder in the *International Classification of Sleep Disorders,* 3rd Edition.

Restless Legs Syndrome

Diagnostic Criteria	**G25.81**

A. An urge to move the legs, usually accompanied by or in response to uncomfortable and unpleasant sensations in the legs, characterized by all of the following:

1. The urge to move the legs begins or worsens during periods of rest or inactivity.
2. The urge to move the legs is partially or totally relieved by movement.

3. The urge to move the legs is worse in the evening or at night than during the day, or occurs only in the evening or at night.

B. The symptoms in Criterion A occur at least three times per week and have persisted for at least 3 months.
C. The symptoms in Criterion A are accompanied by significant distress or impairment in social, occupational, educational, academic, behavioral, or other important areas of functioning.
D. The symptoms in Criterion A are not attributable to another mental disorder or medical condition (e.g., arthritis, leg edema, peripheral ischemia, leg cramps) and are not better explained by a behavioral condition (e.g., positional discomfort, habitual foot tapping).
E. The symptoms are not attributable to the physiological effects of a drug of abuse or medication (e.g., akathisia).

Diagnostic Features

Restless legs syndrome (RLS) is a sensorimotor, neurological sleep disorder characterized by a desire to move the legs or arms, usually associated with uncomfortable sensations typically described as creeping, crawling, tingling, burning, or itching (Criterion A). Frequent movements of the legs occur in an effort to relieve the uncomfortable sensations. Although symptoms can occur during the daytime, they commonly occur in the late afternoon or evening hours, and in some individuals, symptoms occur only in the evening or night. Symptoms are often most severe at night when the individual is at rest, such as sitting or lying in bed. Evening worsening occurs independently of any differences in activity. The diagnosis of RLS is

based primarily on individual self-report and history. It is important to differentiate RLS from other conditions that cause leg discomfort, such as positional discomfort and leg cramps (Criterion D).

The symptoms of RLS can delay sleep onset and awaken the individual from sleep and are associated with significant sleep fragmentation. The relief obtained from moving the legs may no longer be apparent in severe cases. RLS is associated with daytime sleepiness and is frequently accompanied by significant clinical distress or functional impairment.

Associated Features

Periodic leg movements in sleep (PLMS) can serve as corroborating evidence for RLS, with up to 90% of individuals diagnosed with RLS demonstrating PLMS when recordings are taken over multiple nights. Periodic leg movements during wakefulness are also supportive of an RLS diagnosis. Reports of difficulty initiating and maintaining sleep and of excessive daytime sleepiness substantiate a diagnosis of RLS. Additional supportive features include a family history of RLS among first-degree relatives and a reduction in symptoms, at least initially, with dopaminergic treatment.

Prevalence

Prevalence rates of RLS vary widely when broad criteria are utilized. When frequency of symptoms is at least three times per week with moderate or severe distress, the prevalence rate in the United States and Europe has been estimated as 1.6%. RLS that is severe enough to significantly impair functioning or is associated with mental disorders, including depression and anxiety, occurs in approximately 2%–3% of the population, as assessed in Western Europe, the United States, and South Korea. RLS is about twice as common in women as men and increases in prevalence with age. Reports of RLS vary across geographic regions, with lower prevalence in several Asian populations (e.g., Japan, South Korea).

Development and Course

The onset of RLS typically occurs in the second or third decade. Approximately 40% of individuals diagnosed with RLS during adulthood report having experienced symptoms before age 20 years, and 20% report having experienced symptoms before age 10 years. Prevalence rates of RLS increase steadily with age until about age 60 years, with symptoms remaining stable or decreasing slightly in older age groups. Compared with nonfamilial cases, familial RLS usually has a younger age at onset and a slower progressive course. The clinical course of RLS differs by age at onset. When onset occurs before age 45, there is often a slow progression of symptoms. In late-onset RLS, rapid progression is typical, and aggravating factors are common. The RLS phenotype appears similar across the life span.

Diagnosis of RLS in children can be difficult because of the centrality of self-report in establishing the diagnosis. While Criterion A for adults assumes that the description of "urge to move" is by the individual, pediatric diagnosis also requires a description in the child's own words rather than by a parent or caretaker. Typically children age 6 years or older are able to provide detailed, adequate descriptors of RLS. However, children rarely use or understand the word "urge," reporting instead that their legs "have to" or "got to" move. Also, potentially related to prolonged periods of sitting during class, two-thirds of children and adolescents with RLS report daytime leg sensations. Thus, for diagnostic Criterion A3, it is important to compare equal duration of sitting or lying down in the day to sitting or lying down in the evening or night. Nocturnal worsening tends to persist even in pediatric RLS. As with RLS in adults, there is a significant negative impact on sleep, mood, cognition, and function. Impairment in children and adolescents is manifested more often in behavioral and educational domains.

Risk and Prognostic Factors

Genetic and physiological. Predisposing factors include female sex, advancing age, genetic risk variants, and family history of RLS. Precipitating factors such as iron deficiency are often time-limited, with most individuals resuming normal sleep patterns after the triggering event has disappeared. Genetic risk variants also play a role in RLS secondary to

disorders such as uremia, suggesting that individuals with a genetic susceptibility develop RLS in the presence of additional risk factors.

Genome-wide association studies have found that RLS is significantly associated with multiple genetic variants in intronic or intergenic regions. The variant in *MEIS1* has the strongest association with RLS of these genes, with nearly double the risk of RLS in the 7% of the population with this polymorphism among European-ancestry samples studied.

Pathophysiological mechanisms in RLS also include disturbances in the central dopaminergic and opioidergic systems and disturbances in iron metabolism. Treatment efficacy of dopaminergic drugs, opioids, and iron provides further support that these systems play a role in the pathophysiology of RLS. RLS may predispose to depression, and the effective treatment of RLS may significantly reduce depressive symptoms. However, serotonergic antidepressants can induce or aggravate RLS in some individuals.

Culture-Related Diagnostic Issues

Among indigenous-descent Latin American adult populations in the United States, including Mexican Americans with low acculturation to U.S. society, the reported prevalence of RLS appears to be lower when compared with Mexican Americans with higher acculturation. Among participants who reported RLS in a large population-based survey, risk factors associated with RLS were different in Mexican Americans (higher among women and persons who smoke) compared with non-Latinx Whites (older age, defined as ≥ 48 years).

Sex- and Gender-Related Diagnostic Issues

Although RLS is more prevalent in women than in men, there are no diagnostic differences according to gender. The prevalence of RLS during pregnancy is two to three times greater

than in the general population. RLS associated with pregnancy peaks during the third trimester and improves or resolves in most cases soon after

delivery. The sex difference in prevalence of RLS is explained at least in part by parity, with nulliparous females being at the same risk for RLS as age-matched males.

Diagnostic Markers

Polysomnography demonstrates significant abnormalities in RLS, including increased latency to sleep and higher arousal index. Periodic limb movements are the motor sign of RLS and are usually present on overnight polysomnography, as well as during waking immobilization tests and during quiet resting, both of which can provoke RLS symptoms.

Functional Consequences of Restless Legs Syndrome

Although the impact of milder symptoms is less well characterized, individuals with RLS complain of disruption in at least one activity of daily living, with up to 50% reporting a negative impact on mood and a lack of energy. A common consequence of RLS is sleep disturbance, including difficulty falling asleep and sleep fragmentation, with associated reduction in total sleep time. RLS is also associated with quality-of-life impairments. RLS can result in daytime sleepiness or fatigue and is frequently accompanied by significant distress or impairment in affective, social, occupational, educational, academic, behavioral, or cognitive functioning.

Differential Diagnosis

The most important conditions in the differential diagnosis of RLS are leg cramps, positional discomfort, arthralgias/arthritis, myalgias, positional ischemia (numbness), leg edema, peripheral neuropathy, radiculopathy, and habitual foot tapping. Muscle cramps, relief with a single postural shift, limitation to joints, soreness to palpation (myalgias), and other abnormalities on physical examination are not characteristic of RLS. Unlike RLS, nocturnal leg cramps do not typically manifest with the desire to move the limbs nor are there frequent limb movements. Less common conditions to be differentiated from RLS include neuroleptic-induced akathisia, myelopathy, symptomatic venous insufficiency, peripheral artery disease, eczema, other orthopedic problems, and anxiety-induced

restlessness. Worsening at night and periodic limb movements are more common in RLS than in medication-induced akathisia or peripheral neuropathy.

While it is important that RLS symptoms not be solely accounted for by another medical or behavioral condition, it should also be appreciated that any of these similar conditions can occur in an individual with RLS. This necessitates a separate focus on each possible condition in the diagnostic process and when assessing impact. For cases in which the diagnosis of RLS is not certain, evaluation for the supportive features of RLS, particularly PLMS or a family history of RLS, may be helpful. Clinical features, such as response to a dopaminergic agent and positive family history for RLS, can help with the differential diagnosis.

Comorbidity

RLS is associated with higher rates of depression, generalized anxiety disorder, panic disorder, and posttraumatic stress disorder. The main medical condition comorbid with RLS is cardiovascular disease. There may be an association with numerous other medical conditions, including hypertension, migraine, Parkinson's disease, multiple sclerosis, peripheral neuropathy, diabetes mellitus, fibromyalgia, osteoporosis, obesity, thyroid disease, and cancer, as well as other sleep disorders including narcolepsy and obstructive sleep apnea. RLS is common in those with iron deficiency, pregnancy, and chronic renal failure and can dramatically improve once these conditions resolve.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 3rd Edition, presents similar diagnostic criteria for RLS but does not contain a criterion specifying frequency or duration of symptoms.

Substance/Medication-Induced Sleep Disorder

Diagnostic Criteria

A. A prominent and severe disturbance in sleep.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

   1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication.
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a sleep disorder that is not substance/medication-induced. Such evidence of an independent sleep disorder could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced sleep disorder (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the

symptoms in Criterion A predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance/medication]-induced sleep disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given. If a mild substance use disorder is comorbid with the substance-induced sleep disorder, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced sleep disorder (e.g., "mild cocaine use disorder with cocaine-induced sleep disorder"). If a moderate or severe substance use disorder is comorbid with the substance-induced sleep disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance-induced sleep disorder.

There are two exceptions to this coding convention as it applies to caffeine- and tobacco-induced sleep disorders. Because caffeine use disorder is not an official DSM-5 category,

there is only a single ICD-10-CM code for caffeine-induced sleep disorder: F15.982. Moreover, because ICD-10-CM assumes that tobacco-induced sleep disorder can only occur in the context of moderate or severe tobacco use disorder, the ICD-10-CM code for tobacco-induced sleep disorder is F17.208.

	ICD-10-CM

	ICD-10-CM		
	With mild use disorder	**With moderate or severe use disorder**	**Without use disorder**
Alcohol	F10.182	F10.282	F10.982
Caffeine	NA	NA	F15.982
Cannabis	F12.188	F12.288	F12.988
Opioid	F11.182	F11.282	F11.982
Sedative, hypnotic, or anxiolytic	F13.182	F13.282	F13.982
Amphetamine-type substance (or other stimulant)	F15.182	F15.282	F15.982
Cocaine	F14.182	F14.282	F14.982
Tobacco	NA	F17.208	NA
Other (or unknown) substance	F19.182	F19.282	F19.982

Specify whether:

Insomnia type: Characterized by difficulty falling asleep or maintaining sleep, frequent nocturnal awakenings, or nonrestorative sleep.

Daytime sleepiness type: Characterized by predominant complaint of excessive sleepiness/fatigue during waking hours or, less commonly, a long sleep period.

Parasomnia type: Characterized by abnormal behavioral events during sleep.

Mixed type: Characterized by a substance/medication-induced sleep problem characterized by multiple types of sleep symptoms, but no symptom clearly predominates.

Specify (see Table 1 in the chapter “Substance-Related and Addictive Disorders,” which indicates whether “with onset during intoxication” and/or “with onset during withdrawal” applies to a given substance class; or *specify* “with onset after medication use”):

With onset during intoxication: If criteria are met for intoxication with the substance and the symptoms develop

during the intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: If symptoms developed at initiation of medication, with a change in use of medication, or during withdrawal of medication.

Recording Procedures

The name of the substance/medication-induced sleep disorder begins with the specific substance (e.g., alcohol) that is presumed to be causing the sleep disturbance. The ICD-10-CM code that corresponds to the applicable drug class is selected from the table included in the criteria set. For substances that do not fit into any of the classes (e.g., fluoxetine), the ICD-10-CM code for the other (or unknown) substance class should be used and the name

of the specific substance recorded (e.g., F19.982 fluoxetine-induced sleep disorder, insomnia type). In cases in which a substance is judged to be an etiological factor but the specific substance is unknown, the ICD-10-CM code for the other (or unknown) substance class is used and the fact that the substance is unknown is recorded (e.g., F19.982 unknown substance-induced sleep disorder, hypersomnia type).

To record the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by "with substance/medication-induced sleep disorder" (incorporating the name of the specific etiological substance/medication), followed by the specification of onset (i.e., with onset during intoxication, with onset during withdrawal, with onset after medication use), followed by the subtype designation (i.e., insomnia type, daytime sleepiness type, parasomnia type, mixed type). For example, in the case of insomnia occurring during withdrawal in a man with a severe lorazepam use disorder, the diagnosis is F13.282 severe lorazepam use

disorder with lorazepam-induced sleep disorder, with onset during withdrawal, insomnia type. A separate diagnosis of the comorbid severe lorazepam use disorder is not given. If the substance-induced sleep disorder occurs without a comorbid substance use disorder (e.g., with medication use as prescribed), no accompanying substance use disorder is noted (e.g., F19.982 bupropion-induced sleep disorder, with onset during medication use, insomnia type). When more than one substance is judged to play a significant role in the development of the sleep disturbance, each should be listed separately (e.g., F10.282 severe alcohol use disorder with alcohol-induced sleep disorder, with onset during intoxication, insomnia type; F14.282 severe cocaine use disorder with cocaine-induced sleep disorder, with onset during intoxication, insomnia type).

Specifiers

Depending on the substance involved, one of four types of sleep disturbances is indicated. Insomnia type and daytime sleepiness type are most common, whereas parasomnia type is seen less often. The mixed type is noted when more than one type of sleep disturbance–related symptom is present and none predominates.

Diagnostic Features

The essential feature of substance/medication-induced sleep disorder is a prominent sleep disturbance that is sufficiently severe to warrant independent clinical attention (Criterion A). The sleep disturbance may be characterized by insomnia, daytime sleepiness, a parasomnia, or some combination of these. The sleep disturbance is judged to be primarily associated with the pharmacological effects of a substance (i.e., a drug of abuse, a medication, toxin exposure) (Criterion B). The disturbance must not be better explained by another sleep disorder that is not substance/medication-induced (Criterion C). A substance/medication-induced sleep disorder is distinguished from insomnia disorder or a disorder associated with excessive daytime sleepiness by considering onset and course. For drugs of abuse, there must be evidence of intoxication or withdrawal from the history, physical examination, or laboratory findings. Substance/medication-induced sleep disorder arises only in association with

intoxication or discontinuation/withdrawal states, whereas other sleep disorders may precede the onset of substance use or occur during times of sustained abstinence. As discontinuation/withdrawal states for some substances can be protracted, onset of the sleep disturbance can occur 4 weeks after cessation of substance use, and the disturbance may have features atypical of other sleep disorders (e.g., atypical age at onset or course). The diagnosis is not made if the sleep disturbance occurs only during a delirium (Criterion D). The symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E). This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and when the symptoms warrant independent clinical attention.

Associated Features

During periods of substance/medication use, intoxication, or withdrawal, individuals frequently complain of dysphoric mood, including depression and anxiety, irritability, cognitive impairment, inability to concentrate, and fatigue.

Prominent and severe sleep disturbances can occur in association with intoxication with the following classes of substances: alcohol; caffeine; cannabis; opioids; sedatives, hypnotics, or anxiolytics; stimulants (including cocaine); and other (or unknown) substances. Prominent and severe sleep disturbances can occur in association with withdrawal from the following classes of substances: alcohol; caffeine; cannabis; opioids; sedatives, hypnotics, or anxiolytics; stimulants (including cocaine); tobacco; and other (or unknown) substances. Medications that invoke sleep disturbances include adrenergic agonists and antagonists, dopamine agonists and antagonists, cholinergic agonists and antagonists, serotonergic agonists and antagonists, antihistamines, and corticosteroids.

Alcohol. Alcohol-induced sleep disorder typically occurs as insomnia type. During acute intoxication with doses >1 g/kg, alcohol produces an immediate sedative effect depending on dose, accompanied by a reduction

in sleep latency, increased non–rapid eye movement (NREM) sleep stages 2 and 3 (N2 and N3), and reduced rapid eye movement (REM) sleep. Following these initial effects, there may be increased wakefulness, restless sleep, and vivid and anxiety-laden dreams for the remaining sleep period. In parallel, N2 and N3 are reduced, and wakefulness and REM sleep are increased during the latter portion of the night. With habitual use, alcohol continues to show a short-lived sedative effect in the first half of the night, followed by sleep continuity disruption in the second half. During acute alcohol withdrawal, there is extremely disrupted sleep continuity, and an increased amount and intensity of REM sleep, associated frequently with vivid dreaming, which in extreme form constitutes part of alcohol withdrawal delirium. After acute withdrawal, chronic alcohol users may continue to complain of light, fragmented sleep for months to years associated with a persistent prolongation of sleep latency and deficit in slow-wave sleep. Alcohol also aggravates breathing-related sleep disorder, including obstructive sleep apnea and sleep-related hypoventilation.

Caffeine. Caffeine consumed in low to moderate doses during the morning hours typically produces no significant effect on nighttime sleep in normal sleepers or those with insomnia. Caffeine may produce insomnia in a dose- and timing–dependent manner, particularly when larger doses are consumed later in the day or during evening hours. Prolongation of sleep latency, reduction of slow-wave sleep, increased nocturnal awakening, and reduced sleep duration are reported. Some individuals, particularly high consumers, may present with daytime sleepiness and performance impairments related to withdrawal.

Cannabis. Acute administration of cannabis may shorten sleep latency, though arousing effects with increments in sleep latency also occur. Cannabis enhances slow-wave sleep and suppresses REM sleep after acute administration. In chronic users, tolerance to the sleep-inducing and slow-wave sleep–enhancing effects develops. Upon withdrawal, sleep difficulties and unpleasant dreams have been reported lasting for several weeks. Polysomnographic studies demonstrate reduced slow-wave sleep and increased REM sleep during this phase.

Opioids. Opioids may produce an increase in sleepiness and in subjective depth of sleep, and reduced REM and slow-wave sleep, during acute short-

term use. With continued administration, tolerance to the sedative effects of opioids develops and there are complaints of insomnia. Polysomnographic studies demonstrate reduced sleep efficiency and total sleep time, with reduction of slow-wave sleep and possibly REM sleep. Consistent with their respiratory depressant effects, opioids exacerbate obstructive sleep apnea. Emergence of central sleep apnea is also observed, especially with chronic use of longer-acting opioids.

Sedative, hypnotic, or anxiolytic substances. Sedatives, hypnotics, and anxiolytics (e.g., barbiturates, benzodiazepine receptor agonists, meprobamate, glutethimide, methyprylon) have similar effects as opioids on sleep. During acute intoxication, sedative-hypnotic drugs produce the expected increase in sleepiness and decrease in wakefulness. Daytime sleepiness may occur, primarily with longer-acting agents. Chronic benzodiazepine use may be associated with development of tolerance, rebound insomnia, and potentially serious withdrawal effects. Newer benzodiazepine receptor agonists such as zolpidem and eszopiclone have been shown to maintain efficacy over periods of 6 months to 2 years, without evidence of dosage escalation or major withdrawal effects. Newer hypnotic agents such as ramelteon, low-dose doxepin, and suvorexant do not appear to have significant abuse potential, respiratory depression, or major withdrawal syndromes. Sedative, hypnotic, or anxiolytic drugs with short durations of action are most likely to produce complaints of rebound insomnia. Some sedative-hypnotic drugs may increase the frequency and severity of obstructive sleep apnea events, although neither benzodiazepines nor benzodiazepine receptor agonists have been found to definitively worsen obstructive sleep apnea. Hypoventilation may worsen in susceptible individuals. Parasomnias (sleepwalking and sleep-related eating) have been associated with use of benzodiazepine receptor agonists, especially when these medications are taken at higher doses and when they are combined with other sedative drugs.

Amphetamine-type substances, other stimulants, and MDMA. Sleep disorders induced by amphetamine-type substances and other stimulants are characterized by insomnia during intoxication and excessive sleepiness

during withdrawal. During acute intoxication, stimulants reduce the total amount of sleep, increase sleep latency and sleep continuity disturbances, and decrease REM sleep. Slow-wave sleep tends to be reduced. During withdrawal from chronic stimulant use, there is both prolonged nocturnal sleep duration and excessive daytime sleepiness. Multiple sleep latency tests may show increased daytime sleepiness during the withdrawal phase. Drugs like 3,4-methylenedioxymethamphetamine (MDMA; "ecstasy") and related substances lead to restless and disturbed sleep within 48 hours of intake; frequent use of these compounds is associated with persisting symptoms of anxiety, depression, and sleep disturbances, even during longer-term abstinence. There is also evidence that suggests an increased frequency of obstructive sleep apnea in young MDMA users, even after a period of abstention from the drug.

Tobacco. Chronic tobacco use is associated primarily with symptoms of insomnia, decreased slow-wave sleep with a reduction of sleep efficiency, and increased daytime sleepiness. Withdrawal from tobacco can lead to impaired sleep. Individuals who smoke heavily may experience regular nocturnal awakenings caused by tobacco craving.

Other or unknown substances/medications. Other substances/medications may produce sleep disturbances, particularly medications that affect the central or autonomic nervous systems (e.g., adrenergic agonists and antagonists, dopamine agonists and antagonists, cholinergic agonists and antagonists, serotonergic agonists and antagonists, antihistamines, corticosteroids).

Development and Course

Insomnia in children can be identified by either a parent or the child. Often the child has a clear sleep disturbance associated with initiation of a medication but may not report symptoms, although parents observe the sleep disturbances. The use of some recreational substances (e.g., cannabis, ecstasy) is prevalent in adolescence and early adulthood. Insomnia or any other sleep disturbance encountered in this age group should prompt careful consideration of whether the sleep disturbance is attributable to consumption of these substances. Help-seeking behavior for the sleep disturbance in these age groups is limited, and

thus corroborative report may be elicited from a parent, caregiver, or teacher. Older individuals take more medications and are at increased risk for developing a substance/medication-induced sleep disorder. They may interpret sleep disturbance as part of normal aging and fail to report symptoms. Individuals with major neurocognitive disorder (e.g., dementia) are at risk for substance/medication-induced sleep disorders but may not report symptoms, making corroborative report from caregiver(s) particularly important.

Risk and Prognostic Factors

Risk and prognostic factors involved in substance or medication use are normative for certain age groups. They are relevant for, and likely applicable to, the type of sleep disturbance encountered (see the chapter "Substance-Related and Addictive Disorders" for descriptions of respective substance use disorders).

Temperamental. Substance use generally precipitates or accompanies insomnia in vulnerable individuals. Thus, presence of insomnia in response to stress or change in sleep environment or timing can represent a risk for developing substance/medication-induced sleep disorder. A similar risk may be present for individuals with other sleep disorders (e.g., individuals with hypersomnia who use stimulants).

Sex- and Gender-Related Diagnostic Issues

The same amount and duration of consumption of a given substance may lead to highly different sleep-related outcomes in males and females based on, for example, sex-specific differences in hepatic functioning.

Diagnostic Markers

Each of the substance/medication-induced sleep disorders produces electroencephalographic sleep patterns that are associated with, but cannot be considered diagnostic of, other disorders. The electroencephalographic sleep profile for each substance is related to the stage of use and whether it

is in the context of intake/intoxication, chronic use, or withdrawal following discontinuation of the substance. All-night polysomnography can help define the severity of insomnia complaints, while the multiple sleep latency test provides information about the severity of daytime sleepiness. Monitoring of nocturnal respiration and periodic limb movements with polysomnography may verify a substance's impact on nocturnal breathing and motor behavior. Sleep diaries for 2 weeks and actigraphy are considered helpful in confirming the presence of substance/medication-induced sleep disorder, especially in the case of suspected insomnia type. Drug screening can be of use when the individual is not aware or unwilling to relate information about substance intake.

Functional Consequences of Substance/Medication-Induced Sleep Disorder

While there are many functional consequences associated with sleep disorders, the only unique consequence for substance/medication-induced sleep disorder is increased risk for relapse. For example, the degree of sleep disturbance during alcohol withdrawal (e.g., REM sleep rebound) predicts risk of relapse of drinking. Monitoring of sleep quality and daytime sleepiness during and after withdrawal may provide clinically meaningful information on whether an individual is at increased risk for relapse.

Differential Diagnosis

Substance intoxication and substance withdrawal. Sleep disturbances are commonly encountered in the context of substance intoxication and substance withdrawal. A diagnosis of substance/medication-induced sleep disorder should be made instead of a

diagnosis of substance intoxication or substance withdrawal only when the sleep disturbance is predominant in the clinical picture and is sufficiently severe to warrant independent clinical attention.

Delirium. If the substance/medication-induced sleep disturbance occurs exclusively during the course of a delirium, it is not diagnosed separately.

Other sleep disorders. A substance/medication-induced sleep disorder is distinguished from another sleep disorder if a substance/medication is judged to be etiologically related to the symptoms. A substance/medication-induced sleep disorder attributed to a prescribed medication for a mental disorder or medical condition must have its onset while the individual is receiving the medication or during discontinuation, if there is a discontinuation/withdrawal syndrome associated with the medication. Once treatment is discontinued, the sleep disturbance will usually remit within days to several weeks. If symptoms persist beyond 4 weeks, other causes for the sleep disturbance–related symptoms should be considered. Not infrequently, individuals with another sleep disorder use medications or drugs of abuse to self-medicate their symptoms (e.g., alcohol for management of insomnia). If the substance/medication is judged to play a significant role in the exacerbation of the sleep disturbance, an additional diagnosis of a substance/medication-induced sleep disorder may be warranted.

Sleep disorder associated with medical condition. Substance/medication-induced sleep disorder and sleep disorders with a medical condition (i.e., insomnia disorder, hypersomnolence disorder, and nightmare disorder) may produce similar symptoms of insomnia, daytime sleepiness, or nightmares, respectively. Many medical conditions that cause sleep disturbance are treated with medications that may also cause sleep disturbances. The chronology of symptoms is the most important factor in distinguishing between these two sources of sleep symptoms. Difficulties with sleep in an individual with a comorbid medical condition that clearly preceded the use of any medication for treatment of that medical condition would suggest a diagnosis of insomnia disorder, hypersomnolence disorder, or nightmare disorder with the specifier “with [specific medical condition]” applicable to the diagnosis. Conversely, sleep symptoms that appear only after the initiation of a particular substance/medication suggest a substance/medication-induced sleep disorder. If the sleep disturbance is comorbid with another medical condition and is also exacerbated by substance use, both diagnoses are given (i.e., insomnia disorder, hypersomnolence disorder, or nightmare disorder, “with [specific medical condition]” respectively; and [specific substance/medication]-induced sleep

disorder). When there is insufficient evidence to determine whether the sleep disturbance is attributable to a substance/medication or a medical condition, or independent (i.e., not attributable to either a substance/medication or a medical condition), a diagnosis of unspecified sleep-wake disorder is indicated.

Comorbidity

See the "Comorbidity" sections for other sleep disorders in this chapter, including insomnia disorder, hypersomnolence disorder, central sleep apnea, sleep-related hypoventilation, and circadian rhythm sleep-wake disorders, shift work type.

Relationship to International Classification of Sleep Disorders

The *International Classification of Sleep Disorders*, 3rd Edition (ICSD-3), lists sleep disorders "due to a medication or substance" under their respective phenotypes (e.g., hypersomnia, movement disorder, parasomnia). ICSD-3 does not identify a separate diagnosis for "insomnia due to a medication or substance" based on evidence that the reliability of distinguishing specific, single etiological factors for chronic insomnia is poor.

Other Specified Insomnia Disorder

G47.09

This category applies to presentations in which symptoms characteristic of insomnia disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for insomnia disorder or any of the disorders in the sleep-wake disorders diagnostic class. The other specified insomnia disorder

category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for insomnia disorder or any specific sleep-wake disorder. This is done by recording “other specified insomnia disorder” followed by the specific reason (e.g., “short-term insomnia disorder”).

Examples of presentations that can be specified using the “other specified” designation include the following:

1. **Short-term insomnia disorder:** Duration is less than 3 months.
2. **Restricted to nonrestorative sleep:** Predominant complaint is nonrestorative sleep unaccompanied by other sleep symptoms such as difficulty falling asleep or remaining asleep.

Unspecified Insomnia Disorder

G47.00

This category applies to presentations in which symptoms characteristic of insomnia disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for insomnia disorder or any of the disorders in the sleep-wake disorders diagnostic class. The unspecified insomnia disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for insomnia disorder or a specific sleep-wake disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Other Specified Hypersomnolence Disorder

G47.19

This category applies to presentations in which symptoms characteristic of hypersomnolence disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for hypersomnolence disorder or any of the disorders in the sleep-wake disorders diagnostic class. The other specified hypersomnolence disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for hypersomnolence disorder or any specific sleep-wake disorder. This is done by recording “other specified hypersomnolence disorder” followed by the specific reason (e.g., “brief-duration hypersomnolence,” as in Kleine-Levin syndrome).

Unspecified Hypersomnolence Disorder

G47.10

This category applies to presentations in which symptoms characteristic of hypersomnolence disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for hypersomnolence disorder or any of the disorders in the sleep-wake disorders diagnostic class. The unspecified hypersomnolence disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for hypersomnolence disorder or a specific sleep-wake disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Other Specified Sleep-Wake Disorder

G47.8

This category applies to presentations in which symptoms characteristic of a sleep-wake disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the sleep-wake disorders diagnostic class and do not qualify for a diagnosis of other specified insomnia disorder or other specified hypersomnolence disorder. The other specified sleep-wake disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific sleep-wake disorder. This is done by recording “other specified sleep-wake disorder” followed by the specific reason (e.g., “repeated arousals during rapid eye movement sleep without polysomnography or history of Parkinson’s disease or other synucleinopathy”).

Unspecified Sleep-Wake Disorder

G47.9

This category applies to presentations in which symptoms characteristic of a sleep-wake disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the sleep-wake disorders diagnostic class and do not qualify for a diagnosis of unspecified insomnia disorder or unspecified hypersomnolence disorder. The unspecified sleep-wake disorder category is used in situations in which the clinician chooses *not* to specify the reason that the

criteria are not met for a specific sleep-wake disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Sexual Dysfunctions

Sexual dysfunctions include delayed ejaculation, erectile disorder, female orgasmic disorder, female sexual interest/arousal disorder, genito-pelvic pain/penetration disorder, male hypoactive sexual desire disorder, premature (early) ejaculation, substance/medication-induced sexual dysfunction, other specified sexual dysfunction, and unspecified sexual dysfunction. Sexual dysfunctions are a heterogeneous group of disorders that are typically characterized by a clinically significant disturbance in a person's ability to respond sexually or to experience sexual pleasure. An individual may have several sexual dysfunctions at the same time. In such cases, all of the dysfunctions should be diagnosed.

Clinical judgment should be used to determine if the sexual difficulties are the result of inadequate sexual stimulation; in these cases, there may still be a need for care, but a diagnosis of a sexual dysfunction would not be made. These cases may include, but are not limited to, conditions in which lack of knowledge about effective stimulation prevents the experience of arousal or orgasm.

Subtypes are used to designate the onset of the difficulty. In many individuals with sexual dysfunctions, the time of onset may indicate different etiologies and interventions. *Lifelong* refers to a sexual problem that has been present from first sexual experiences, and *acquired* applies to sexual dysfunctions that develop after a period of relatively normal sexual function. *Generalized* refers to sexual difficulties that are not limited to certain types of stimulation, situations, or partners, and *situational* refers to sexual difficulties that only occur with certain types of stimulation, situations, or partners.

In addition to the lifelong/acquired and generalized/situational subtypes, a number of factors must be considered during the assessment of sexual dysfunction, given that they may be relevant to etiology or treatment and

may contribute, to varying degrees, across individuals: 1) partner factors (e.g., partner's sexual problems; partner's health status); 2) relationship factors (e.g., poor communication; discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image; history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural or religious factors (e.g., inhibitions related to prohibitions against sexual activity or pleasure; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment.

Clinical judgment about the diagnosis of sexual dysfunction should take into consideration cultural factors that may influence expectations or engender prohibitions about the experience of sexual pleasure. Aging and relationship duration may be associated with a normative decrease in sexual response.

Sexual response has a requisite biological underpinning, yet is usually experienced in an intrapersonal, interpersonal, and cultural context. Thus, sexual function involves a complex interaction among biological, sociocultural, and psychological factors. In many clinical contexts, a precise understanding of the etiology of a sexual problem is unknown. Nonetheless, a sexual dysfunction diagnosis requires ruling out problems that are better explained by a nonsexual mental disorder, by the effects of a substance (e.g., drug or

medication), by a medical condition (e.g., due to pelvic nerve damage), or by severe relationship distress, partner violence, or other stressors.

The population of gender-diverse persons, including transgender, nonbinary, and agender, may not identify with or appear to fit into the existing sex- and gender-based diagnostic categories described in this chapter. Despite the names given to male hypoactive sexual desire disorder and female sexual interest arousal disorder, the diagnostic criteria describe symptoms and experiences that are not dependent on the individual's specific sex or gender. As such, either diagnosis can be applied to gender-diverse individuals based on clinical judgment. For diagnoses linked to reproductive anatomy (e.g., erectile dysfunction, premature [early]

ejaculation, delayed ejaculation, and genito-pelvic pain/penetration disorder), diagnoses should be based on the individual's current anatomy and not on the individual's sex assigned at birth. Much more research is needed to understand experiences of sexual dysfunction among gender-diverse persons. In the meantime, as with all categories in DSM, clinicians should use their best judgment.

If the sexual dysfunction is mostly explainable by another nonsexual mental disorder (e.g., depressive or bipolar disorder, anxiety disorder, posttraumatic stress disorder, psychotic disorder), then only the other mental disorder diagnosis should be made. If the problem is thought to be better explained by the use/misuse or discontinuation of a drug or substance, it should be diagnosed accordingly as a substance/medication-induced sexual dysfunction. If the sexual dysfunction is attributable to another medical condition (e.g., peripheral neuropathy), the person would not receive a psychiatric diagnosis. If severe relationship distress, partner violence, or significant stressors better explain the sexual difficulties, then a sexual dysfunction diagnosis is not made, but an appropriate Z code for the relationship problem or stressor may be listed (e.g., Z63.0 Relationship distress with spouse or intimate partner); see the chapter "Other Conditions That May Be a Focus of Clinical Attention." In many cases, a precise etiological relationship between another condition (e.g., a medical condition) and a sexual dysfunction cannot be established. It is possible to have a diagnosis of a sexual dysfunction and a coexisting medical condition, nonsexual mental disorder, or use/misuse or discontinuation of a drug or substance; and it is possible to have one or more diagnoses of sexual dysfunction.

Delayed Ejaculation

Diagnostic Criteria F52.32

A. Either of the following symptoms must be experienced on almost all or all occasions (approximately 75%–100%) of partnered sexual activity (in identified situational contexts or,

if generalized, in all contexts), and without the individual desiring delay:

1. Marked delay in ejaculation.
2. Marked infrequency or absence of ejaculation.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

The essential feature of delayed ejaculation is a marked delay in or inability to achieve ejaculation or marked infrequency of ejaculation on all or almost all occasions of partnered sexual activity, despite the presence of adequate sexual stimulation and the desire to ejaculate (Criterion A). In order to qualify for a DSM-5 diagnosis of delayed ejaculation, the symptoms must have persisted for a minimum duration of approximately 6 months (Criterion B) and must cause clinically significant distress in the individual (Criterion C). The partnered sexual activity may include manual, oral, coital, or anal stimulation. In most cases, the diagnosis will be made by self-report, although for men in heterosexual partnered relationships, it is frequently the female partner's distress that motivates treatment seeking. It is common for men who present with delayed ejaculation to be able to ejaculate with self-stimulation, but not during partnered sexual activity.

The definition of "delay" does not have precise boundaries, as there is no consensus as to what constitutes a reasonable time to reach orgasm or what is unacceptably long for most men and their sexual partners. Although the definitions of delayed ejaculation apply equally well to both heterosexual and homosexual orientation, the vast majority of the research focus has been based on the concept of intravaginal latency, and therefore male-female intercourse. The findings from those studies document that the majority of men's intravaginal ejaculatory latency time (IELT) range is approximately 4–10 minutes. There is also no clear diagnostic delineation between delayed ejaculation as a sexual dysfunction and delay that is a consequence of normal aging. Therefore, the diagnosis of delayed ejaculation is based on clinical judgment, taking into consideration the individual's psychosexual and medical history, age, relationship context, and sexual stimulation patterns and behaviors. The diagnosis of delayed ejaculation should not be given if the clinician judges that the individual's dissatisfaction is entirely attributable to unrealistic expectations.

Associated Features

The man and his partner may report prolonged thrusting to achieve orgasm to the point of exhaustion or genital discomfort and sometimes even injury to himself and/or his partner before finally ceasing. Some males may report avoiding sexual activity because of a repetitive pattern of difficulty ejaculating.

Delayed ejaculation is associated with highly frequent masturbation, use of masturbation techniques not easily duplicated by a partner, and marked disparities between sexual fantasies during masturbation and the reality of sex with a partner.

Males with delayed ejaculation typically report less coital activity, higher levels of relationship distress, sexual dissatisfaction, lower subjective arousal, anxiety about their sexual performance, and general health issues than sexually functional men.

In addition to considerations of applicable subtypes (i.e., whether the ejaculatory delay has been present since the individual became sexually active or began after a period of relatively normal sexual function, and whether the ejaculatory delay is generalized or occurs only with certain types of stimulation, situations, or partners), the following factors are important to consider in the assessment of delayed ejaculation: 1) partner factors (e.g., partner’s sexual problems or health); 2) relationship factors (e.g., poor communication, discrepancies

in desire for sexual activity); 3) individual vulnerability factors (e.g., hypoactive sexual desire), psychiatric comorbidity (e.g., depression, anxiety), or stressors such as job loss or stress; 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); 5) medical factors, particularly hypogonadism or neurological disorders (e.g., multiple sclerosis, diabetic neuropathy); and 6) use of substances or medications that might inhibit ejaculation (e.g., use of serotonergic drugs).

Prevalence

The prevalence of delayed ejaculation in the United States is estimated at 1%–5% but has ranged as high as 11% in international studies. However, variation in syndrome definitions across studies may have contributed to differences in the prevalence of the DSM-5 disorder.

Development and Course

Lifelong delayed ejaculation begins with early sexual experiences and continues throughout the course of an individual's life. Acquired delayed ejaculation begins after a period of normal sexual function. A number of biomedical, psychosocial, and cultural factors can contribute to the predisposition to or maintenance of lifelong or acquired delayed ejaculation (see the section "Risk and Prognostic Factors"), and either subtype can be generalized or situational in nature.

The prevalence of delayed ejaculation increases with age. As males age, they are more likely to have progressively more of the following changes in ejaculatory function, including, but not limited to, reduced ejaculatory volume, force, and sensation, and increased "refractory time." Refractory latency increases for males secondary to surgical, medical, and pharmaceutical complications, as well as aging.

Risk and Prognostic Factors

Ejaculatory latency is an end point consequence that is determined by a range of factors. A large number of psychosocial factors increase the probability of an individual experiencing delayed ejaculation, with depression and relationship dissatisfaction being predominant contributors.

Genetic and physiological. Numerous medical conditions may lead to delayed ejaculation, including procedures that disrupt sympathetic or somatic innervation to the genital region such as radical prostatectomy for cancer treatment. Neurological and endocrine disorders, including spinal cord injury, stroke, multiple sclerosis, pelvic-region surgery, severe diabetes, epilepsy, hormonal abnormalities, and sleep apnea, as well as alcohol abuse, bowel dysfunction, cannabis use, and environmental factors, may be associated with delayed ejaculation.

Additionally, medications that inhibit α-adrenergic innervation of the ejaculatory system (e.g., tamsulosin) are associated with delayed

ejaculation, as well as antihypertensive agents, antidepressants (e.g., selective serotonin reuptake inhibitors), and antipsychotic drugs.

Age-related loss of the fast-conducting peripheral sensory nerves and age-related decreased sex steroid secretion may be associated with an increase in delayed ejaculation in males as they age. Reduced androgen levels with age may also be associated with delayed ejaculation.

Sex- and Gender-Related Diagnostic Issues

By definition, the diagnosis of delayed ejaculation is only given to males. Distressing difficulties with orgasm in women would be considered under female orgasmic disorder.

Functional Consequences of Delayed Ejaculation

Delayed ejaculation is often associated with considerable psychological distress in one or both partners.

Difficulty with ejaculation may contribute to difficulties in conception and lead to significant fertility assessment, as the lack of ejaculation is not often spontaneously discussed by individuals unless there is direct inquiry from their physician.

Differential Diagnosis

Another medical condition or injury and/or its treatment. A major diagnostic challenge is differentiating between a delayed ejaculation that is fully explained by another medical condition or injury (or its treatment) and a delayed ejaculation attributable to a variety of proportionally different biomedical-psychosocial and cultural factors that determine the symptom(s). A number of medical conditions or injuries, along with their treatments, may produce delays in ejaculation independent of psychosocial and cultural issues.

Delayed ejaculation must be differentiated from a number of urological conditions (especially other ejaculatory disorders), including retrograde ejaculation or *anejaculation*, which is typically the result of etiologies

ranging from hormonal to neurological and/or anatomical abnormalities, including ejaculatory duct obstruction and other urological disorders.

Substance/medication use. A number of pharmacological agents, such as antidepressants, antipsychotics, α sympathetic drugs, alcohol, and opioid drugs, can cause ejaculatory problems. In such cases, the diagnosis is substance/medication-induced sexual dysfunction instead of delayed ejaculation.

Dysfunction with orgasm. It is important in the history to ascertain whether the complaint concerns delayed ejaculation or the sensation of orgasm, or both. Ejaculation occurs in the genitals, whereas the experience of orgasm is believed to be primarily subjective. Ejaculation and orgasm usually occur together but not always. For example, a male with a normal ejaculatory pattern may complain of decreased pleasure (i.e., anhedonic ejaculation). Such a complaint would not be coded as delayed ejaculation but could be coded as other specified sexual dysfunction or unspecified sexual dysfunction.

Comorbidity

There is some evidence to suggest that delayed ejaculation may be more common in severe forms of major depressive disorder.

Erectile Disorder

Diagnostic Criteria	**F52.21**

A. At least one of the three following symptoms must be experienced on almost all or all (approximately 75%–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts):

   1. Marked difficulty in obtaining an erection during sexual activity.

2. Marked difficulty in maintaining an erection until the completion of sexual activity.
3. Marked decrease in erectile rigidity.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

The essential feature of erectile disorder is a marked difficulty in obtaining or maintaining an erection or a marked decrease in erectile rigidity in all or almost all occasions of sexual activity (Criterion A) that has persisted for at least 6 months (Criterion B) and that causes clinically significant distress in the individual (Criterion C). A careful sexual history is necessary to ascertain that the problem has been present for a significant duration of time (i.e., at least approximately 6 months) and occurs on the majority of sexual occasions (i.e., at least 75% of the time). Symptoms may occur only in specific situations involving certain types of stimulation or partners, or they may occur in a generalized manner in all types of situations, stimulation, or partners.

This chapter uses the terms *erectile disorder* and *erectile dysfunction*, which are not synonymous. *Erectile dysfunction* is a widely used descriptive term (including in ICD-10) that refers to difficulty getting and maintaining an erection. *Erectile disorder* is the more specific DSM-5 diagnostic category in which erectile dysfunction persists for at least 6 months and causes distress in the individual.

Associated Features

Many males with erectile disorder may have low self-esteem, low self-confidence, and a decreased sense of masculinity, and may experience depressed mood. Erectile dysfunction is also strongly associated with feelings of guilt, self-blame, sense of failure, anger, and concern about disappointing one's partner. Fear and/or avoidance of future sexual encounters may occur. Decreased sexual satisfaction and reduced sexual desire in the individual's partner are common.

In addition to considerations of applicable subtypes (i.e., whether the erectile dysfunction has been present since the individual became sexually active or began after a period of relatively normal sexual function, and whether the erectile dysfunction is generalized or occurs only with certain types of stimulation, situations, or partners), the following factors are important to consider in the assessment of erectile disorder: 1) partner factors (e.g., partner's sexual problems or health); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3)

individual vulnerability factors (e.g., hypoactive sexual desire), psychiatric comorbidity (e.g., depression, anxiety), or stressors such as job loss or stress; 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); 5) medical factors, particularly surgery (e.g., transurethral resection of the prostate), hypogonadism, or neurological conditions

(e.g., multiple sclerosis, diabetic neuropathy); and 6) use of substances or medications that might inhibit ejaculation (e.g., use of serotonergic drugs).

Prevalence

The prevalence of lifelong versus acquired erectile disorder is unknown. There is a strong age-related increase in both prevalence and incidence of problems with erection, particularly after age 50 years. Internationally, the prevalence of erectile disorder in the general population is approximately 13%–21% of males ages 40–80 years. Rates appear to be lower than 10% in males younger than 40 years, about 20%–40% in males in their 60s, and 50%–75% in males older than 70 years. In a longitudinal study in Australia, 80% of males age 70 and older experienced erectile disorder. In a review of studies largely from Western countries, about 20% of males feared erectile problems on their first sexual experience, whereas approximately 8% experienced erectile problems that hindered penetration during their first sexual experience. Among U.S.-based respondents to an online survey, there was no statistically significant difference in the prevalence of erectile disorder by ethnoracial background. Nationally representative U.S. data show that the prevalence of erectile difficulties is similar in older males who have sex with males or with both males and females.

Development and Course

Erectile failure on first sexual attempt has been found to be related to having sex with a previously unknown partner, concomitant use of drugs or alcohol, not wanting to have sex, and peer pressure. There is minimal evidence regarding the persistence of such problems after the first attempt.

It is assumed that most of these problems spontaneously remit without professional intervention, but some males may continue to have episodic problems. In contrast, acquired erectile disorder is often associated with biological factors such as diabetes and cardiovascular disease. Acquired erectile disorder is likely to be persistent in most men.

The natural history of lifelong erectile disorder is unknown. Clinical observation supports the association of lifelong erectile disorder with psychological factors that are self-limiting or responsive to psychological interventions, whereas, as noted above, acquired erectile disorder is more likely to be related to biological factors and to be persistent. The incidence of erectile disorder increases with age. A minority of males diagnosed as having moderate erectile failure may experience spontaneous remission of symptoms without medical intervention. Distress associated with erectile disorder is lower in older males as compared with younger males.

Risk and Prognostic Factors

Course modifiers. Risk factors for acquired erectile dysfunction and, as a consequence, erectile disorder include age, smoking tobacco, lack of physical exercise, diabetes, and decreased desire.

Culture-Related Diagnostic Issues

Prevalence of erectile disorder varies across countries. It is unclear to what extent these variations represent differences in cultural expectations as opposed to genuine differences in the frequency of erectile failure. Differential endorsement may be related to cultural concerns about appearing weak or less masculine or to diverse cultural norms about changes in erectile function during healthy aging. Cultural expectations concerning marital relationships, sexual performance, fertility, and gender roles can influence anxieties that may contribute to erectile disorder. Based on responses to an online survey, erectile disorder may be associated with concern about genital size in the United States and the Middle East and with fears of male infertility more frequently in the Middle East.

Sex- and Gender-Related Diagnostic Issues

By definition, the diagnosis of erectile dysfunction is only given to males. Distressing difficulties with sexual arousal in women would be considered under female sexual interest/arousal disorder.

Diagnostic Markers

Nocturnal penile tumescence testing and measured erectile turgidity during sleep can be employed to help differentiate organic from psychogenic erectile problems on the assumption that adequate erections during rapid eye movement sleep indicate a psychological etiology to the problem. A number of other diagnostic procedures may be employed depending on the clinician's assessment of their relevance given the individual's age, comorbid medical problems, and clinical presentation. Doppler ultrasonography and intravascular injection of vasoactive drugs, as well as invasive diagnostic procedures such as dynamic infusion cavernosography, can be used to assess vascular integrity. Pudendal nerve conduction studies, including somatosensory evoked potentials, can be employed when a peripheral neuropathy is suspected. Testing for low levels of serum bioavailable or free testosterone is appropriate especially when diabetes is present, for men who also experience hypoactive desire, and for those who do not respond to phosphodiesterase type 5 inhibitors. Thyroid function may also be assessed. Determination of fasting serum glucose is useful to screen for the presence of diabetes mellitus. The assessment of serum lipids is important, as erectile disorder in males 40 years and older is predictive of the future risk for coronary artery disease.

Association With Suicidal Thoughts or Behavior

Among males receiving treatment for erectile disorder with comorbid depression, elevated rates of suicidal thoughts or behavior have been observed; while the affected males attributed the suicidal symptoms to their erectile disorder, the presence of depression was also a likely contributing factor. Elevated suicide rates among males with prostate cancer may in part be related to treatment-associated erectile dysfunction and consequent depressive symptoms.

Functional Consequences of Erectile Disorder

Erectile disorder can interfere with fertility and produce both individual and interpersonal distress. Fear and/or avoidance of sexual encounters may interfere with the ability to develop intimate relationships. Significant psychological distress may occur among males presenting with erectile disorder.

Differential Diagnosis

Nonsexual mental disorders. Major depressive disorder and erectile disorder are closely associated, and erectile disorder accompanying severe depressive disorder may occur. If the erectile difficulties are better explained by another mental disorder, such as major depression, then a diagnosis of erectile disorder would not be made.

Normal erectile function. The differential should include consideration of normal erectile function in males with excessive expectations.

Substance/medication use. An onset of erectile dysfunction that coincides with the beginning of substance/medication use and that dissipates with discontinuation of the substance/medication or dose reduction is suggestive of a substance/medication-induced sexual dysfunction, which should be diagnosed instead of erectile disorder.

Another medical condition. The most difficult aspect of the differential diagnosis of erectile disorder is ruling out erectile problems that are fully explained by medical factors. Such cases would not receive a diagnosis of a mental disorder. The distinction between erectile disorder as a mental disorder and erectile dysfunction as the result of another medical condition is usually unclear, and many cases will have complex, interactive biological and psychiatric etiologies. If the individual is older than 40–50 years and/or has concomitant medical problems, the differential diagnosis should include medical etiologies, especially vascular disease. The presence of an organic disease known to cause erectile problems does not confirm a causal relationship. For example, a male with diabetes mellitus can develop erectile disorder in response to psychological stress. In general, erectile

dysfunction due to organic factors is generalized and gradual in onset. An exception would be erectile problems after traumatic injury to the nervous innervation of the genital organs (e.g., spinal cord injury). Erectile problems that are situational and inconsistent and that have an acute onset after a stressful life event are most often attributable to psychological events. An age younger than 40 years is also suggestive of a psychological etiology to the difficulty.

Comorbidity

Erectile disorder can be comorbid with other sexual diagnoses, such as premature (early) ejaculation and male hypoactive sexual desire disorder, as well as with anxiety and depressive disorders. The risk of depression is significantly higher in males with erectile disorder, with a markedly higher risk of depression in the first year after onset. In males diagnosed with posttraumatic stress disorder, erectile problems are common. Erectile disorder is common in males with lower urinary tract symptoms related to prostatic hypertrophy. Erectile disorder may be comorbid with dyslipidemia, cardiovascular disease, hypogonadism, multiple sclerosis, diabetes mellitus, and other diseases that interfere with the vascular, neurological, or endocrine function necessary for normal erectile function.

Female Orgasmic Disorder

Diagnostic Criteria **F52.31**

A. Presence of either of the following symptoms and experienced on almost all or all (approximately 75%–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts):

   1. Marked delay in, marked infrequency of, or absence of orgasm.
   2. Markedly reduced intensity of orgasmic sensations.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress (e.g., partner violence) or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify if:

Never experienced an orgasm under any situation.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

Female orgasmic disorder is characterized by difficulty experiencing orgasm and/or markedly reduced intensity of orgasmic sensations (Criterion A). Women show wide variability in the type or intensity of stimulation that elicits orgasm. Similarly, subjective descriptions of orgasm are extremely varied, suggesting that it is experienced in very different ways, both across women and on different occasions by the same woman. For a diagnosis of female orgasmic disorder, symptoms must be experienced on almost all or all (approximately 75%–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts) and have a minimum duration of approximately 6 months. The use of the minimum severity and duration criteria is intended to distinguish transient orgasm difficulties from more persistent orgasmic dysfunction. The inclusion of "approximately" in Criterion B allows for clinician judgment in cases in which symptom duration does not meet the recommended 6-month threshold.

For a woman to have a diagnosis of female orgasmic disorder, clinically significant distress must accompany the symptoms (Criterion C). In many cases of orgasm problems, the causes are multifactorial or cannot be determined. If female orgasmic disorder is deemed to be better explained by another mental disorder, the effects of a substance/medication, or a medical condition, then a diagnosis of female orgasmic disorder would not be made. Finally, if interpersonal or significant contextual factors, such as severe relationship distress, intimate partner violence, or other significant stressors, are present, then a diagnosis of female orgasmic disorder would not be made.

Many women require clitoral stimulation to reach orgasm, and a relatively small proportion of women report that they always experience orgasm during penile-vaginal intercourse. Thus, a woman's experiencing orgasm through clitoral stimulation but not during intercourse does not meet criteria for a clinical diagnosis of female orgasmic disorder. It is also important to consider whether orgasm difficulties are the result of inadequate sexual stimulation; in these cases, there may still be a need for care, but a diagnosis of female orgasmic disorder would not be made.

Associated Features

Associations between specific patterns of personality traits or psychopathology and orgasmic dysfunction have generally not been supported. Compared with women without the disorder, some women with female orgasmic disorder may have greater difficulty communicating about sexual issues. Overall sexual satisfaction, however, is not strongly correlated with orgasmic experience. Many women report high levels of sexual satisfaction despite rarely or never experiencing orgasm. Orgasm difficulties in women often co-occur with problems related to sexual interest and arousal.

In addition to the subtypes "lifelong/acquired" and "generalized/situational," the following five factors must be considered during assessment and diagnosis of female orgasmic disorder given that they may be relevant to etiology or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor

communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Each of these factors may contribute differently to the presenting symptoms of different women with this disorder.

Prevalence

Reported prevalence rates for orgasm problems in premenopausal women vary widely, from 8% to 72%, depending on multiple factors (e.g., age, cultural background and context, duration, severity of symptoms); however, these estimates do not take into account the presence of distress. Only a proportion of women experiencing orgasm difficulties also report associated distress. Variation in how symptoms are assessed (e.g., the duration of symptoms and the recall period) also influence prevalence rates.

Internationally, approximately 10% of women do not experience orgasm throughout their lifetime.

Development and Course

By definition, lifelong female orgasmic disorder indicates that the orgasm difficulties have always been present, whereas the acquired subtype would be assigned if the woman's orgasm difficulties developed after a period of normal orgasmic functioning.

A woman's first experience of orgasm can occur any time from the prepubertal period to well into adulthood. Women show a more variable pattern in age at first orgasm than do men, and women's reports of having experienced orgasm increase with age. Many women learn to experience orgasm as they experience a wide variety of stimulation and acquire more knowledge about their bodies. Women's rates of orgasmic consistency (defined as "usually or always" experiencing orgasm) are higher during masturbation than during sexual activity with a partner.

Risk and Prognostic Factors

Temperamental. A wide range of psychological factors, such as anxiety and concerns about pregnancy, can potentially interfere with a woman's ability to experience orgasm.

Environmental. There is a strong association between relationship problems, physical health, and mental health and orgasm difficulties in women. Sociocultural factors (e.g., gender role expectations and religious norms) are also important influences on the experience of orgasm difficulties.

Genetic and physiological. Many physiological factors may influence a woman's experience of orgasm, including medical conditions and medications. Conditions such as multiple sclerosis, pelvic nerve damage from radical hysterectomy, and spinal cord injury can all influence orgasmic functioning in women. Selective serotonin reuptake inhibitors are known to delay or inhibit orgasm in women. Women with vulvovaginal atrophy (characterized by symptoms such as vaginal dryness, itching, and pain) are significantly more likely to report orgasm difficulties than are women without this condition. Menopausal status is not consistently associated

with the likelihood of orgasm difficulties. There may be a significant genetic contribution to variation in female orgasmic function. However, psychological, sociocultural, and physiological factors likely interact in complex ways to influence women's experience of orgasm and of orgasm difficulties.

Culture-Related Diagnostic Issues

The degree to which lack of orgasm in women is regarded as a problem that requires treatment may vary depending on cultural context. Cultural views that undervalue women's

sexual satisfaction or that perceive marital sex as a duty for women rather than a pleasurable activity are associated with lower help-seeking. In addition, women differ in how important orgasm is to their sexual satisfaction. There may be marked sociocultural and generational differences in women's orgasmic ability. For example, reports of the prevalence of inability to reach orgasm vary over a twofold range across world regions.

Sex- and Gender-Related Diagnostic Issues

By definition, the diagnosis of female orgasmic disorder is given only to women. Distressing difficulties with orgasm in men would be considered under delayed ejaculation.

Diagnostic Markers

Although measurable physiological changes occur during female orgasm, including changes in hormones, pelvic floor musculature, and brain activation, there is significant variability in these indicators of orgasm across women. In clinical situations, the diagnosis of female orgasmic disorder is based on a woman's self-report.

Association With Suicidal Thoughts or Behavior

Dysfunctions of sexual arousal and satisfaction have been associated with suicidal thoughts among female veterans and military service members even after adjustment for probable posttraumatic stress disorder, probable depression, history of deployment, married status, age, service in the army, and race.

Functional Consequences of Female Orgasmic Disorder

The functional consequences of female orgasmic disorder are unclear. Although there is a strong association between relationship problems and orgasm difficulties in women, it is unclear whether relationship factors are risk factors for orgasm difficulties or are consequences of those difficulties.

Differential Diagnosis

Nonsexual mental disorders. If the orgasm difficulties are better explained by another mental disorder, such as major depression, then a diagnosis of female orgasmic disorder would not be made.

Substance/medication-induced sexual dysfunction. An onset of orgasmic dysfunction that coincides with the beginning of substance/medication use and that dissipates with discontinuation of the substance/medication or dose reduction is suggestive of a substance/medication-induced sexual dysfunction, which should be diagnosed instead of female orgasmic disorder.

Another medical condition. If the disorder is attributable to another medical condition (e.g., multiple sclerosis, spinal cord injury), then a diagnosis of female orgasmic disorder would not be made.

Interpersonal factors. If interpersonal or significant contextual factors, such as severe relationship distress, intimate partner violence, or other significant stressors, are associated with the orgasm difficulties, then a diagnosis of female orgasmic disorder would not be made.

Other sexual dysfunctions. Female orgasmic disorder may occur in association with other sexual dysfunctions (e.g., female sexual interest/arousal disorder). The presence of another sexual dysfunction does not rule out a diagnosis of female orgasmic disorder. Occasional orgasm difficulties that are short-term or infrequent and are not accompanied by

clinically significant distress or impairment are not diagnosed as female orgasmic disorder. A diagnosis is also not appropriate if the problems are the result of inadequate sexual stimulation.

Comorbidity

Women with female orgasmic disorder may have co-occurring sexual interest/arousal difficulties. Women with diagnoses of other nonsexual mental disorders, such as major depressive disorder, may experience lower sexual interest/arousal, and this may indirectly increase the likelihood of orgasm difficulties.

Female Sexual Interest/Arousal Disorder

Diagnostic Criteria F52.22

A. Lack of, or significantly reduced, sexual interest/arousal, as manifested by at least three of the following:
   1. Absent/reduced interest in sexual activity.
   2. Absent/reduced sexual/erotic thoughts or fantasies.
   3. No/reduced initiation of sexual activity, and typically unreceptive to a partner's attempts to initiate.
   4. Absent/reduced sexual excitement/pleasure during sexual activity in almost all or all (approximately 75%–100%) sexual encounters (in identified situational contexts or, if generalized, in all contexts).
   5. Absent/reduced sexual interest/arousal in response to any internal or external sexual/erotic cues (e.g., written, verbal, visual).
   6. Absent/reduced genital or nongenital sensations during sexual activity in almost all or all (approximately 75%–

100%) sexual encounters (in identified situational contexts or, if generalized, in all contexts).

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress (e.g., partner violence) or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

In assessing female sexual interest/arousal disorder, interpersonal context must be taken into account. A “desire discrepancy,” in which a woman has

lower desire for sexual activity than her partner, is not sufficient to diagnose female sexual interest/arousal disorder. For the criteria for the disorder to be met, there must be absence or reduced frequency or

intensity of at least three of six indicators (Criterion A) for a minimum duration of approximately 6 months (Criterion B). There may be different symptom profiles across women, as well as variability in how sexual interest and arousal are expressed. For example, in one woman, sexual interest/arousal disorder may be expressed as a lack of interest in sexual activity, an absence of erotic or sexual thoughts, and reluctance to initiate sexual activity and respond to a partner's sexual invitations. In another woman, an inability to become sexually excited, an inability to respond to sexual stimuli with sexual desire, and a corresponding lack of signs of physical sexual arousal may be the primary features. Difficulties in desire and sexual arousal may also occur simultaneously, as women with loss of sexual desire may be nine times more likely to also have lost sexual excitement or arousal. Short-term changes in sexual interest or arousal are common and may be adaptive responses to events in a woman's life and do not represent a sexual dysfunction. Diagnosis of female sexual interest/arousal disorder requires a minimum duration of symptoms of approximately 6 months as a reflection that the symptoms must be a persistent problem. The estimation of persistence may be determined by clinical judgment when a duration of 6 months cannot be ascertained precisely.

There may be absent or reduced frequency or intensity of interest in sexual activity (Criterion A1), which was previously the single criterion for *hypoactive sexual desire disorder;* this condition is now represented by female sexual interest/arousal disorder. The frequency or intensity of sexual and erotic thoughts or fantasies may be absent or reduced (Criterion A2). The expression of fantasies varies widely across women and may include memories of past sexual experiences. The normative decline in sexual thoughts with age should be taken into account when this criterion is being assessed. Absence or reduced frequency of initiating sexual activity and of receptivity to a partner's sexual invitations (Criterion A3) is a behaviorally

focused criterion. A couple's beliefs and preferences for sexual initiation patterns are highly relevant to the assessment of this criterion. There may be absent or reduced sexual excitement or pleasure during sexual activity in almost all or all (approximately 75%–100%) sexual encounters (Criterion A4). Lack of pleasure is a common presenting clinical complaint in women with low desire. Among women who report low sexual desire, there are fewer sexual or erotic cues that elicit sexual interest or arousal (i.e., there is a lack of "responsive desire"). Evidence suggests that there may be at least two types of female sexual interest/arousal disorder: one based on low sensitivity to sexual cues and a second based on overactivation of sexual inhibition. Assessment of the adequacy of sexual stimuli will assist in determining if there is a difficulty with responsive sexual desire (Criterion A5). Frequency or intensity of genital or nongenital sensations during sexual activity may be reduced or absent (Criterion A6). This may include reduced vaginal lubrication/vasocongestion, but because physiological measures of genital sexual response do not differentiate women who report sexual arousal concerns from those who do not, the self-report of reduced or absent genital or nongenital sensations is sufficient.

For a diagnosis of female sexual interest/arousal disorder to be made, clinically significant distress must accompany the symptoms in Criterion A. Distress may be experienced as a result of the lack of sexual interest/arousal or as a result of significant interference in a woman's life and well-being. If a lifelong lack of sexual desire is better explained by one's self-identification as "asexual," then a diagnosis of female sexual interest/arousal disorder would not be made.

Associated Features

Female sexual interest/arousal disorder is frequently associated with problems in experiencing orgasm, pain experienced during sexual activity, infrequent sexual activity, and couple-level discrepancies in desire. Relationship difficulties, chronic stress, and mood disorders are also frequently associated features of female sexual interest/arousal disorder. Unrealistic expectations and norms regarding the "appropriate" level of sexual

interest or arousal, along with poor sexual techniques and lack of information about sexuality, may also be evident in women diagnosed with female sexual interest/arousal disorder. The latter, as well as normative beliefs about gender roles, are important factors to consider.

In addition to the subtypes “lifelong/acquired” and “generalized/situational,” the following five factors must be considered during assessment and diagnosis of female sexual interest/arousal disorder given that they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner’s sexual problems, partner’s health status, partner-related distress); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity; relationship duration); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Note that each of these factors may contribute differently to the presenting symptoms of different women with this disorder.

Prevalence

Approximately 30% of women experience chronic low desire, with approximately half of these experiencing significant partner-related distress and a quarter experiencing personal distress. The prevalence of low sexual desire and of problems with sexual arousal (with and without associated distress) may vary markedly in relation to age, cultural context, duration of symptoms, and presence of distress. Regarding duration of symptoms, there are striking differences in prevalence estimates between short-term and persistent problems related to lack of sexual interest. When distress about sexual functioning is required, prevalence estimates are markedly lower. Though there is a strong relationship between low desire and age, the prevalence of sex-related distress associated with low desire decreases as women age.

Development and Course

By definition, lifelong female sexual interest/arousal disorder suggests that the lack of sexual interest or arousal has been present for the woman's entire sexual life. For Criteria A3, A4, and A6, which assess functioning during sexual activity, a subtype of lifelong would mean presence of symptoms since the individual's first sexual experiences. The acquired subtype would be assigned if the difficulties with sexual interest or arousal developed after a period of nonproblematic sexual functioning. Adaptive and normative changes in sexual functioning may result from partner-related, interpersonal, or personal events and may be transient in nature. However, persistence of symptoms for approximately 6 months or more would constitute a sexual dysfunction.

There are normative changes in sexual interest and arousal across the life span. Furthermore, women in relationships of longer duration are more likely to report engaging in sex despite no obvious feelings of sexual desire at the outset of a sexual encounter compared with women in shorter-duration relationships. Vaginal dryness in older women is related to age and menopausal status.

Risk and Prognostic Factors

Temperamental. Temperamental factors include negative cognitions and attitudes about sexuality and past history of mental disorders. Differences in propensity for sexual excitation and sexual inhibition may also predict the likelihood of developing sexual problems.

Environmental. Environmental factors include relationship difficulties, partner sexual functioning, and developmental history, such as early relationships with caregivers and childhood stressors.

Genetic and physiological. Some medical conditions (e.g., diabetes mellitus, thyroid dysfunction) can be risk factors for female sexual interest/arousal disorder. There appears to be a strong influence of genetic factors on vulnerability to sexual problems in women. Psychophysiological research

using vaginal photoplethysmography has not found differences between women with and without perceived lack of genital arousal.

Culture-Related Diagnostic Issues

There is marked variability in reported prevalence rates of low desire across world regions, ranging from 26% to 43%. Low levels of sexual desire have been reported by some ethnoracial and migrant groups. Although lower levels of reported sexual desire and arousal may reflect less interest in sex, such group differences may be an artifact of the measures used to quantify desire and of cultural factors affecting response, such as the desirability of reporting sexual activity by nonmarried, menopausal, or widowed women. A judgment about whether low sexual desire reported by a woman from a certain ethnocultural background meets criteria for female sexual interest/arousal disorder must take into account the fact that different cultural groups may vary in norms and expectations for sexual behavior.

Sex- and Gender-Related Diagnostic Issues

By definition, the diagnosis of female sexual interest/arousal disorder is only given to women. Distressing difficulties with sexual desire in men would be considered under male hypoactive sexual desire disorder. There are no data showing that rates or expressions of female sexual interest/arousal disorder differ between heterosexual and lesbian women.

Association With Suicidal Thoughts or Behavior

Dysfunctions of sexual arousal and satisfaction have been associated with suicidal thoughts among female veterans and military service members even after adjustment for probable PTSD, probable depression, history of deployment, married status, age, service in the army, and race.

Functional Consequences of Female Sexual Interest/Arousal Disorder

Difficulties in sexual interest/arousal are often associated with decreased relationship satisfaction.

Differential Diagnosis

Nonsexual mental disorders. Nonsexual mental disorders, such as major depressive disorder, in which there is "markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day," may explain the lack of sexual interest/arousal. If the lack of interest or arousal is completely attributable to another mental disorder, then a diagnosis of female sexual interest/arousal disorder would not be made.

Substance/medication use. An onset of difficulties in desire or arousal that coincides with the beginning of substance/medication use and that dissipates with discontinuation of the substance/medication or dose reduction is suggestive of a substance/medication-induced sexual dysfunction, which should be diagnosed instead of female sexual interest/arousal disorder.

Another medical condition. If the sexual symptoms are considered to be almost exclusively associated with the effects of another medical condition (e.g., diabetes mellitus,

endothelial disease, thyroid dysfunction, central nervous system disease), then a diagnosis of female sexual interest/arousal disorder would not be made.

Interpersonal factors. If interpersonal or significant contextual factors, such as severe relationship distress, intimate partner violence, or other significant stressors, explain the sexual interest/arousal symptoms, then a diagnosis of female sexual interest/arousal disorder would not be made.

Other sexual dysfunctions. The presence of another sexual dysfunction does not rule out a diagnosis of female sexual interest/arousal disorder. It is common for women to experience more than one sexual dysfunction. For example, the presence of chronic genital pain may lead to a lack of desire for the (painful) sexual activity. Lack of interest and arousal during sexual activity may impair orgasmic ability. For some women, all aspects of the sexual response may be unsatisfying and distressing.

Inadequate or absent sexual stimuli.

When differential diagnoses are being considered, it is important to assess the adequacy of sexual stimuli within the woman's sexual experience. In cases where inadequate or absent sexual stimuli are contributing to the clinical picture, there may be evidence for clinical care, but a sexual dysfunction diagnosis would not be made. Similarly, transient and adaptive alterations in sexual functioning that are secondary to a significant life or personal event must be considered in the differential diagnosis.

Comorbidity

Comorbidity between sexual interest/arousal problems and other sexual difficulties is extremely common. Sexual distress and dissatisfaction with sex life are also highly correlated in women with low sexual desire. Distressing low desire is associated with depression, thyroid problems, anxiety, urinary incontinence, and other medical factors. Arthritis and inflammatory or irritable bowel disease are also associated with sexual arousal problems. Low desire appears to be comorbid with depression, sexual and physical abuse in adulthood, and use of alcohol.

Genito-Pelvic Pain/Penetration Disorder

Diagnostic Criteria F52.6

A. Persistent or recurrent difficulties with one (or more) of the following:
   1. Vaginal penetration during intercourse.
   2. Marked vulvovaginal or pelvic pain during vaginal intercourse or penetration attempts.
   3. Marked fear or anxiety about vulvovaginal or pelvic pain in anticipation of, during, or as a result of vaginal penetration.
   4. Marked tensing or tightening of the pelvic floor muscles during attempted vaginal penetration.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of a severe relationship distress (e.g., partner violence) or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

Genito-pelvic pain/penetration disorder refers to four commonly comorbid symptom dimensions: 1) difficulty having intercourse, 2) genito-pelvic pain, 3) fear of pain or vaginal penetration, and 4) tension of the pelvic floor muscles (Criterion A). Because major difficulty in any one of these symptom dimensions is often sufficient to cause clinically significant distress, a diagnosis can be made on the basis of marked difficulty in only one symptom dimension. However, all four symptom dimensions should be

assessed even if a diagnosis can be made on the basis of only one symptom dimension.

Marked difficulty having vaginal intercourse/penetration (Criterion A1) can vary from a total inability to experience vaginal penetration in any situation (e.g., intercourse, gynecological examinations, tampon insertion) to the ability to easily experience penetration in one situation but not in another. Although the most common clinical situation is when a female is unable to experience intercourse or penetration with a partner, difficulties in undergoing required gynecological examinations may also be present. *Marked vulvovaginal or pelvic pain during vaginal intercourse or penetration attempts* (Criterion A2) refers to pain occurring in different locations in the genito-pelvic area. Location of pain as well as intensity should be assessed. Typically, pain can be characterized as superficial (vulvovaginal or occurring during penetration) or deep (pelvic; i.e., not felt until deeper penetration). The intensity of the pain is often not linearly related to distress or interference with vaginal penetration or other sexual activities. Some genito-pelvic pain only occurs when provoked (i.e., by intercourse or mechanical stimulation); other genito-pelvic pain may be spontaneous as well as provoked. Genito-pelvic pain can also be usefully characterized qualitatively (e.g., “burning,” “cutting,” “shooting,” “throbbing”). The pain may persist for a period after intercourse is completed and may also occur during urination. Typically, the pain experienced during vaginal intercourse can be reproduced during a gynecological examination.

Marked fear or anxiety about vulvovaginal or pelvic pain either in anticipation of, or during, or as a result of vaginal penetration (Criterion A3) is commonly reported by females who have regularly experienced pain during vaginal penetration. This “normal” reaction may lead to avoidance of sexual/intimate situations. In other cases, this marked fear does not appear to be closely related to the experience of pain but nonetheless leads to avoidance of intercourse and vaginal penetration situations. Some have described this as similar to a phobic reaction except that the phobic object may be vaginal penetration or the fear of pain.

Marked tensing or tightening of the pelvic floor muscles during attempted vaginal penetration (Criterion A4) can vary from reflexive-like spasm of the pelvic floor in response to attempted vaginal entry to

“normal/voluntary” muscle guarding in response to the anticipated or the repeated experience of pain or to fear or anxiety. In the case of “normal/guarding” reactions, penetration may be possible under circumstances of relaxation. The characterization and assessment of pelvic floor dysfunction is often best undertaken by a specialist gynecologist or by a pelvic floor physical therapist.

Symptoms of genito-pelvic pain/penetration disorder may be characterized by previous terms, including *dyspareunia* (pain during sexual intercourse) and *vaginismus* (defined by

involuntary contraction of muscles making penetration painful or impossible). Specific medical disorders, such as vulvodynia (chronic idiopathic vulvar pain lasting at least 3 months) and provoked vestibulodynia (contact-induced vulvodynia localized to the vulvar vestibule), may be a primary cause of genito-pelvic pain/penetration disorder and may be a focus in studies of the disorder. Females diagnosed with these other conditions report significant distress, and their symptoms are likely to meet criteria for genito-pelvic pain/penetration disorder.

Associated Features

Genito-pelvic pain/penetration disorder is frequently associated with other sexual dysfunctions, particularly reduced sexual desire and interest (female sexual interest/arousal disorder). Sometimes desire and interest are preserved in sexual situations that are not painful or do not require penetration. Even when individuals with genito-pelvic pain/penetration disorder report sexual interest/motivation, there is often behavioral avoidance of sexual situations and opportunities. Avoidance of gynecological examinations despite medical recommendations is also frequent. The pattern of avoidance is similar to that seen in phobic disorders. It is common for females who have not succeeded in having vaginal penetration to come for treatment only when they wish to conceive. Many females with genito-pelvic pain/penetration disorder will experience

associated relationship/marital problems; they also often report that the symptoms significantly diminish their feelings of femininity.

In addition to the subtype "lifelong/acquired," five factors should be considered during assessment and diagnosis of genito-pelvic pain/penetration disorder because they may be relevant to etiology or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., partner responses to the pain, including solicitous, negative, and facilitative responses; discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Each of these factors may contribute differently to the presenting symptoms of different females with this disorder.

Prevalence

The prevalence of genito-pelvic pain/penetration disorder is unknown. However, approximately 10%–28% of females of reproductive age in the United States report recurrent pain during intercourse. Difficulties having intercourse appear to be a frequent referral to sexual dysfunction clinics and to specialist clinicians. Internationally, prevalence of genito-pelvic pain upon intercourse ranges from 8% to 28% among females of reproductive age and varies by country.

Prevalence of genito-pelvic pain during sexual activities involving vaginal penetration among lesbian women relative to heterosexual women remains uncertain but may be similar or lower. Prevalence rates among other sexual minorities, including transgender women, are unknown.

Development and Course

The development and course of genito-pelvic pain/penetration disorder is unclear. Because women generally do not seek treatment until they experience problems in sexual functioning, it can, in general, be difficult to characterize genito-pelvic pain/penetration disorder as lifelong (primary) or

acquired (secondary). Although women typically come to clinical attention after the initiation of sexual activity, there are often earlier clinical signs. For example, difficulty with or the avoidance of use of tampons is an important predictor of later

problems. Difficulties with vaginal penetration (inability or fear or pain) may not be obvious until intercourse is attempted during sexual activity. Even once intercourse is attempted, the frequency of attempts may not be significant or regular. In cases where it is difficult to establish whether symptomatology is lifelong or acquired, it is useful to determine the presence of any consistent period of successful pain-, fear-, and tension-free intercourse. If the experience of such a period can be established, then genito-pelvic pain/penetration disorder can be characterized as acquired. Once symptomatology is present for a period of approximately 6 months, the probability of spontaneous and significant symptomatic remission appears to diminish.

Complaints related to genito-pelvic pain peak during early adulthood and in the peri- and postmenopausal period. There may also be an increase in genito-pelvic pain–related symptoms in the postpartum period.

Risk and Prognostic Factors

Temperamental. Females with antecedent mood and anxiety disorders are four times more likely to develop symptoms of genito-pelvic pain/penetration disorder compared with those without these antecedent disorders. Psychosocial factors (e.g., pain catastrophizing, pain self-efficacy, avoidance of pain, negative mood) and interpersonal factors (e.g., insecure attachment, negative partner responses to the pain, sexual motives that focus on avoiding negative relationship outcomes) may exacerbate and maintain symptoms.

Environmental. Females with genito-pelvic pain/penetration disorder are more likely to report a history of sexual and/or physical abuse, and fear of abuse than females without this disorder, although not all women with presenting symptoms have this history.

Genetic and physiological. Females experiencing superficial pain during vaginal penetration often report the onset of the pain after a history of vaginal infections. Even after the infections have resolved and there are no known residual physical findings, the pain persists. Pain during tampon insertion or the inability to insert tampons before any sexual contact has been attempted is an important risk factor for genito-pelvic pain/penetration disorder.

Additional biomedical risk factors include early puberty, inflammation, early use of oral contraceptives, vulvar pain receptor proliferation (i.e., increase in the number of receptors) and sensitization (i.e., touch may become perceived as pain), and lower touch and pain thresholds. Abnormalities of the pelvic floor muscles while at rest, including hypertonicity, poor muscle control, hypersensitivity, and altered contractility, may close the vaginal hiatus and interfere with penetration.

Culture-Related Diagnostic Issues

Cultural contexts can affect the experience and reporting of genito-pelvic pain related to intercourse. Affected females experience negative implications related to social narratives of womanhood, sexuality, and femininity, including pressures to prioritize men's sexual desire and penetrative sex, and depictions of sex as easy and natural. Cultural views that devalue female sexual experience may affect the way women interpret the experience of pain during sex, their help-seeking choices, and how they discuss their symptoms with their caregivers. For example, some females may not report genito-pelvic pain specifically but rather refer to being unhappy in their marriages.

In the United States, Hispanic females endorse significantly higher rates of genito-pelvic pain and are more likely to report pain with first intercourse (i.e., primary genito-pelvic pain/penetration disorder) compared with non-Hispanic women. In a Minneapolis, Minnesota survey, only about half of females with genito-pelvic pain sought treatment, and those who did frequently reported feeling stigmatized. Such experiences may be heightened for

sexual minorities and underserved ethnic and racialized groups, especially given evidence of inequities in pain treatment for females and African Americans.

Sex- and Gender-Related Diagnostic Issues

Gendered social constructions relating to womanhood and femininity are implicated in the experience of genito-pelvic pain/penetration disorder, including the prioritization of both penetrative sex and men's sexual desires above women's own needs and desires. The disorder is associated with feelings of shame and inadequacy as a female, contributing further to enhanced psychological distress.

By definition, the diagnosis of genito-pelvic pain/penetration disorder is only given to females. There is relatively new research concerning urological chronic pelvic pain syndrome in males, suggesting that males may experience some similar problems. The prevalence of male genito-pelvic pain is estimated to be 2.2%–9.7% worldwide. The research and clinical experience are not sufficiently developed yet to justify the application of this diagnosis to males. Other specified sexual dysfunction or unspecified sexual dysfunction may be diagnosed in males appearing to fit this pattern.

Diagnostic Markers

Validated physiological measures of Criterion A2 symptoms (*Marked vulvovaginal or pelvic pain during vaginal intercourse or penetration attempts*) can be assessed in real time (e.g., the cotton-swab test, vulvalgesiometer, tampon test). Although these measures are well validated for pain intensity during penetration attempts, none approximate the sexual context in which the pain is experienced, which can only be assessed via self-report. Criterion A4 symptoms (*Marked tensing or tightening of the pelvic floor muscles during attempted vaginal penetration*) can also be measured (e.g., via electromyographic amplitude, dynamometer, 4D ultrasound by a qualified physical therapist). There are no validated physiological measures of component symptoms for Criterion A1 or A3. Validated psychometric inventories may be used to formally assess the pain and anxiety components related to genito-pelvic pain/penetration disorder.

Functional Consequences of Genito-Pelvic Pain/Penetration Disorder

Functional difficulties in genito-pelvic pain/penetration disorder are often associated with interference in various aspects of the romantic relationship —including the initiation of such relationships—and sometimes with the ability to conceive via penile/vaginal intercourse.

Differential Diagnosis

Another medical condition. In many instances, females with genito-pelvic pain/penetration disorder will also be diagnosed with another medical condition (e.g., lichen sclerosus, endometriosis, pelvic inflammatory disease, genitourinary syndrome of menopause). In some cases, treating the medical condition may alleviate the genito-pelvic pain/penetration disorder. Much of the time, this is not the case. There are no reliable tools or diagnostic methods to allow clinicians to know whether the medical condition or genito-pelvic pain/penetration disorder is primary. Often, the associated medical conditions are difficult to diagnose and treat. For example, the increased incidence of postmenopausal pain during intercourse may sometimes be attributable to vaginal dryness or irritation associated with declining estrogen levels. The relationship, however, between genital symptoms, estrogen, and pain is not well understood.

Somatic symptom and related disorders. Some females with genito-pelvic pain/penetration disorder may also be diagnosable with somatic symptom disorder. Since both genito-pelvic pain/penetration disorder and the somatic symptom and related disorders are new diagnoses in DSM-5, it is not yet clear whether they can be reliably differentiated. Some females diagnosed with genito-pelvic pain/penetration disorder will also be diagnosed with a specific phobia.

Inadequate sexual stimuli. It is important that the clinician, in considering differential diagnoses, assess the adequacy of sexual stimuli within the female's sexual experience. Sexual situations in which there is inadequate foreplay or arousal may lead to difficulties in penetration, pain, or

avoidance. Erectile dysfunction or premature (early) ejaculation in the male partner may result in difficulties with penetration. These conditions should be carefully assessed. In some situations, a diagnosis of genito-pelvic pain/penetration disorder may not be appropriate.

Comorbidity

Comorbidity between genito-pelvic pain/penetration disorder and other sexual difficulties appears to be common. Comorbidity with relationship distress is also common and typically related to the lack of sexual intimacy rather than (solely) the pain itself. This is not surprising, because the inability to have (pain-free) intercourse with a desired partner and the avoidance of sexual opportunities may be either a contributing factor to or the result of other sexual or relationship problems. Because pelvic floor symptoms are implicated in the diagnosis of genito-pelvic pain/penetration disorder, there is likely to be a higher prevalence of other disorders related to the pelvic floor or reproductive organs (e.g., interstitial cystitis, constipation, vaginal infection, endometriosis, irritable bowel syndrome). Females with genito-pelvic pain/penetration disorder frequently experience comorbid chronic pain conditions (e.g., fibromyalgia, chronic headaches), and the prevalence of these comorbidities increases with the severity of vulvar pain symptoms.

Lesbian women also report genito-pelvic pain and penetration difficulties during sexual activities; the frequency of genito-pelvic pain/penetration symptoms among nonheterosexual women has been shown to be less than or the same as among heterosexual women.

Male Hypoactive Sexual Desire Disorder

Diagnostic Criteria	F52.0

A. Persistently or recurrently deficient (or absent) sexual/erotic thoughts or fantasies and desire for sexual activity. The judgment of deficiency is made by the clinician, taking into

account factors that affect sexual functioning, such as age and general and sociocultural contexts of the individual's life.

B. The symptoms in Criterion A have persisted for a minimum duration of approximately 6 months.

C. The symptoms in Criterion A cause clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Evidence of mild distress over the symptoms in Criterion A.

Moderate: Evidence of moderate distress over the symptoms in Criterion A.

Severe: Evidence of severe or extreme distress over the symptoms in Criterion A.

Diagnostic Features

When an assessment for male hypoactive sexual desire disorder is being made, interpersonal context must be taken into account. A “desire discrepancy,” in which a man has lower desire for sexual activity than his partner, is not sufficient to diagnose male hypoactive sexual desire disorder. Both low/absent desire for sex and deficient/absent sexual thoughts or fantasies (Criterion A) are required for a diagnosis of the disorder. There may be variation across men in how sexual desire is expressed.

The lack of desire for sex and deficient/absent erotic thoughts or fantasies must be persistent or recurrent and must occur for a minimum duration of approximately 6 months. The inclusion of this duration criterion is meant to safeguard against making a diagnosis in cases in which a man’s low sexual desire may represent a reactive but temporary response to adverse life conditions. For example, the man’s low sexual desire may be related to an acute stressor or loss of self-esteem (e.g., being fired from a job or experiencing financial difficulty such as business failure). If these stressors persist beyond 6 months along with low sexual desire, then clinician judgment determines the appropriateness of the male hypoactive sexual desire disorder diagnosis.

Associated Features

Male hypoactive sexual desire disorder is sometimes associated with erectile and/or ejaculatory concerns. For example, persistent difficulties obtaining an erection may lead a man to lose interest in sexual activity. Men with hypoactive sexual desire disorder often report that they no longer initiate sexual activity and that they are minimally receptive to a partner’s attempt to initiate. Sexual activities (e.g., masturbation or partnered sexual activity) may sometimes occur even in the presence of low sexual desire. Relationship-specific preferences regarding patterns of sexual initiation must be taken into account when making a diagnosis of male hypoactive sexual desire disorder. Although men are more likely to initiate sexual activity, and thus low desire may be characterized by a pattern of noninitiation, many men may prefer to have their partner initiate sexual activity. In such situations, the man’s lack of receptivity to a partner’s initiation should be considered when evaluating low desire.

In addition to the subtypes "lifelong/acquired" and "generalized/situational," the following five factors must be considered during assessment and diagnosis of male hypoactive sexual desire disorder given that they may be relevant to etiology and/or treatment: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., poor body image, history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), or stressors (e.g., job loss, bereavement); 4) cultural/religious factors (e.g., inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment. Each of these factors may contribute differently to the presenting symptoms of different men with this disorder.

Prevalence

The prevalence of male hypoactive sexual desire disorder varies depending on country of origin and method of assessment. Estimates of prevalence in representative samples range

from 3% to 17%. Sexual desire problems are less common in younger men (ages 16–24), with prevalence rates between 3% and 14%, compared with older men (ages 60–74 years), with prevalence rates between 16% and 28%. However, a persistent lack of interest in sex, lasting 6 months or more, affects a smaller proportion of men (6%). Moreover, less than 2% of men report clinically significant distress associated with low desire. Studies on help-seeking behavior indicate that only 10.5% of men with sexual problems in the previous year sought help.

Development and Course

By definition, lifelong male hypoactive sexual desire disorder indicates that low or no sexual desire has always been present, whereas the acquired subtype would be assigned if the man's low desire developed after a period of normal sexual desire. There is a requirement that low desire persist for

approximately 6 months or more; thus, short-term changes in sexual desire should not be diagnosed as male hypoactive sexual desire disorder.

There is a normative age-related decline in sexual desire. The prevalence of low sexual desire in men increases with age, from approximately 5.2% prevalence at age 27 years to 18.5% at age 50 years. Like women, men identify a variety of triggers for their sexual desire, and they describe a wide range of reasons that they choose to engage in sexual activity. Although erotic visual cues may be more potent elicitors of desire in younger men, the potency of sexual cues may decrease with age and must be considered when evaluating men for hypoactive sexual desire disorder.

Risk and Prognostic Factors

Temperamental. Mood and anxiety symptoms appear to be strong predictors of low desire in men. Up to half of men with a past history of psychiatric symptoms may have moderate or severe loss of desire, compared with only 15% of those without such a history. A man's feelings about himself, his perception of his partner's sexual desire toward him, feelings of being emotionally connected, and contextual variables may all negatively (as well as positively) affect sexual desire.

Beliefs about sexuality (particularly restrictive sexual attitudes and conservative beliefs) are commonly associated with low sexual desire in men. Moreover, lack of erotic thoughts and concerns about erection during sexual activity are significant predictors of low sexual desire, as well as low confidence levels in erectile function.

Environmental. Alcohol use may increase the occurrence of low desire. Other environmental determinants of low sexual desire include problematic dyadic relationships, reduced attraction toward the partner, living in a long-term relationship, sexual boredom, and professional stress. At the larger societal level, cohort studies in a few high-income countries indicate a trend toward the decrease of sexual desire in men in recent decades.

Genetic and physiological. Endocrine disorders such as hyperprolactinemia and hypogonadism significantly affect sexual desire in men. Age is a significant risk factor for low desire in men. It is unclear whether or not men with low desire also have abnormally low levels of testosterone;

however, among hypogonadal men, low desire is common. There also may be a critical threshold below which testosterone will affect sexual desire in men and above which there is little effect of testosterone on men's desire.

Culture-Related Diagnostic Issues

There is marked variability in prevalence rates of low desire across world regions, ranging from 12.5% in Northern European men to 28% in Southeast Asian men ages 40–80 years. Distress about lack of sexual desire was significantly associated with sociocultural contexts (e.g., occupational stress) in a web-based survey across three European countries (Portugal, Croatia, and Norway).

Sex- and Gender-Related Diagnostic Issues

In contrast to the classification of sexual dysfunctions in women, desire and arousal disorders have been retained as separate constructs in men. Despite some similarities in the experience of desire across men and women, and the fact that desire fluctuates over time and is dependent on contextual factors, men do report a significantly higher intensity and frequency of sexual desire compared with women. However, preliminary data suggest that the overlap between sexual desire and sexual arousal (erectile function) is also very common in men, particularly when they present for help regarding sexual problems. Regarding sexual orientation, data suggest that low sexual desire is more commonly reported by gay men (19%) than by heterosexual men (9%).

Differential Diagnosis

Nonsexual mental disorders. Nonsexual mental disorders, such as major depressive disorder, which is characterized by "markedly diminished interest or pleasure in all, or almost all, activities," may explain the lack of sexual desire. If the lack of desire is better explained by another mental disorder, then a diagnosis of male hypoactive sexual desire disorder would not be made.

Substance/medication use.

An onset of male hypoactive sexual desire that coincides with the beginning of substance/medication use and that dissipates with discontinuation of the substance/medication or dose reduction is suggestive of a substance/medication-induced sexual dysfunction, which should be diagnosed instead of male hypoactive sexual desire disorder.

Another medical condition. If the low/absent desire and deficient/absent erotic thoughts or fantasies are better explained by the effects of another medical condition (e.g., hypogonadism, diabetes mellitus, thyroid dysfunction, central nervous system disease), then a diagnosis of male hypoactive sexual desire disorder would not be made.

Interpersonal factors. If interpersonal or significant contextual factors, such as severe relationship distress or other significant stressors, are associated with the loss of desire in the man, then a diagnosis of male hypoactive sexual desire disorder would not be made.

Other sexual dysfunctions. The presence of another sexual dysfunction does not rule out a diagnosis of male hypoactive sexual desire disorder; there is some evidence that up to one-half of men with low sexual desire also have erectile difficulties, and slightly fewer may also have early ejaculation difficulties. If the man's low desire is explained by self-identification as an asexual, then a diagnosis of male hypoactive sexual desire disorder is not made.

Comorbidity

Male hypoactive sexual desire disorder is rarely the sole sexual diagnosis in men. Erectile dysfunction, delayed ejaculation, and premature (early) ejaculation are often comorbid diagnoses. Depression and other mental disorders, as well as endocrinological factors, are often comorbid with male hypoactive sexual desire disorder.

Premature (Early) Ejaculation

Diagnostic Criteria	F52.4

A. A persistent or recurrent pattern of ejaculation occurring during partnered sexual activity within approximately 1 minute following vaginal penetration and before the individual wishes it.

Note: Although the diagnosis of premature (early) ejaculation may be applied to individuals engaged in nonvaginal sexual activities, specific duration criteria have not been established for these activities.

B. The symptom in Criterion A must have been present for at least 6 months and must be experienced on almost all or all (approximately 75%–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts).

C. The symptom in Criterion A causes clinically significant distress in the individual.

D. The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

Specify whether:

Lifelong: The disturbance has been present since the individual became sexually active.

Acquired: The disturbance began after a period of relatively normal sexual function.

Specify whether:

Generalized: Not limited to certain types of stimulation, situations, or partners.

Situational: Only occurs with certain types of stimulation, situations, or partners.

Specify current severity:

Mild: Ejaculation occurring within approximately 30 seconds to 1 minute of vaginal penetration.

Moderate: Ejaculation occurring within approximately 15–30 seconds of vaginal penetration.

Severe: Ejaculation occurring prior to sexual activity, at the start of sexual activity, or within approximately 15 seconds of vaginal penetration.

Diagnostic Features

Premature (early) ejaculation is manifested by ejaculation that occurs prior to or shortly after vaginal penetration, operationalized by an individual's estimate of ejaculatory latency (i.e., elapsed time before ejaculation) after vaginal penetration. Although the diagnostic criteria specify penile-vaginal sex, it is reasonable to assume that similar estimates of ejaculatory latency apply to males having sex with males, as well as to other sexual behaviors. Estimated and measured intravaginal ejaculatory latencies are highly correlated as long as the ejaculatory latency is of short duration; therefore, self-reported estimates of ejaculatory latency are sufficient for diagnostic purposes. A 60-second intravaginal ejaculatory latency time was previously considered to be an appropriate cutoff for the diagnosis of lifelong premature (early) ejaculation in men; however, expert consensus now considers this latency time to be too brief and instead recommends a 120-second threshold.

Associated Features

Many males with premature (early) ejaculation complain of a sense of lack of control over ejaculation and report apprehension about their anticipated inability to delay ejaculation on future sexual encounters.

The following factors may be relevant in the evaluation of any sexual dysfunction: 1) partner factors (e.g., partner's sexual problems, partner's health status); 2) relationship factors (e.g., poor communication, discrepancies in desire for sexual activity); 3) individual vulnerability factors (e.g., history of sexual or emotional abuse), psychiatric comorbidity (e.g., depression, anxiety), and stressors (e.g., job loss, bereavement); 4)

cultural/religious factors (e.g., lack of privacy, inhibitions related to prohibitions against sexual activity; attitudes toward sexuality); and 5) medical factors relevant to prognosis, course, or treatment.

Prevalence

Estimates of the prevalence of premature (early) ejaculation vary widely depending on the definition utilized. Internationally, a prevalence range of 8%–30% has been reported across all ages, with even lower and higher rates in other studies. Prevalence of premature (early) ejaculation may increase with age. For example, the prevalence among males ages 18–30 in Switzerland and Turkey is about 9%–11%, while the reported prevalence of concern among males ages 50–59 in the United States about how rapidly they ejaculate may be as high as 55%. When premature (early) ejaculation is defined as ejaculation occurring within approximately 1 minute of vaginal penetration, only 1%–3% of males would be diagnosed with the disorder.

Development and Course

By definition, lifelong premature (early) ejaculation starts during a male's initial sexual experiences and persists thereafter. Some males may experience premature (early) ejaculation during their initial sexual encounters but gain ejaculatory control over time. It is the persistence of ejaculatory problems for longer than 6 months that determines the diagnosis of premature (early) ejaculation. In contrast, some males develop the disorder after a period of having a normal ejaculatory latency, known as *acquired premature (early) ejaculation.* There is far less known about acquired premature (early) ejaculation than about lifelong premature (early) ejaculation. The acquired form likely has a later onset, usually appearing during or after the fourth decade of life. Lifelong is relatively stable throughout life.

Risk and Prognostic Factors

Temperamental.

Premature (early) ejaculation may be more common in males with anxiety disorders, especially social anxiety disorder.

Genetic and physiological. There is a moderate genetic contribution to lifelong premature (early) ejaculation. Premature (early) ejaculation may be associated with dopamine transporter gene polymorphism or serotonin transporter gene polymorphism. Thyroid disease, prostatitis, and drug withdrawal are associated with acquired premature (early) ejaculation. Positron emission tomography measures of regional cerebral blood flow during ejaculation have shown primary activation in the mesocephalic transition zone, including the ventral tegmental area.

Culture-Related Diagnostic Issues

Perception of what constitutes a normal ejaculatory latency differs cross-culturally and may be related to varying awareness of sexual dysfunction, concern about sexual failure, and perceptions about the importance of sex. Measured ejaculatory latencies may differ in some countries. Cultural or religious factors may contribute to these differences. For example, reports of premature (early) ejaculation were more common in arranged marriages, because of factors such as anxiety over family pressures and lack of premarital sexual experience.

Sex- and Gender-Related Diagnostic Issues

Premature (early) ejaculation is a sexual dysfunction in men. Men and their sexual partners may differ in their perception of what constitutes an acceptable ejaculatory latency. There may be increasing concerns in women about early ejaculation in their sexual partners, which may be a reflection of changing societal attitudes concerning women's sexual activity.

Diagnostic Markers

Ejaculatory latency is usually monitored in research settings by the sexual partner utilizing a timing device (e.g., stopwatch), though this is not ideal in real-life sexual situations. In clinical settings, the man's estimate of the time

between intravaginal penetration and ejaculation should be accepted in lieu of stopwatch measurements.

Association With Suicidal Thoughts or Behavior

Among males receiving treatment for premature (early) ejaculation with comorbid depression, elevated rates of suicidal thoughts or behavior have been observed; although the affected males attributed the suicidal symptoms to their premature (early) ejaculation, the presence of depression was also a likely contributing factor.

Functional Consequences of Premature (Early) Ejaculation

A pattern of premature (early) ejaculation may be associated with decreased self-esteem and self-confidence, a sense of lack of control, and adverse consequences for partner relationships. It may also cause personal distress and decreased sexual satisfaction in the sexual partner. Single males are more bothered than partnered males by premature (early) ejaculation because of its interference with seeking and maintaining new relationships. Ejaculation prior to penetration may be associated with difficulties in conception.

Differential Diagnosis

Substance/medication-induced sexual dysfunction. When problems with premature (early) ejaculation are attributable exclusively to substance use, intoxication, or withdrawal, substance/medication-induced sexual dysfunction should be diagnosed.

Ejaculatory concerns that do not meet diagnostic criteria. It is necessary to identify males with normal ejaculatory latencies who desire longer ejaculatory latencies and males who have episodic premature (early) ejaculation (e.g., during the first sexual encounter with a new partner when a short ejaculatory latency may be common or normative). Neither of these situations would lead to a diagnosis of premature (early) ejaculation, even though these situations may be distressing to some males.

Comorbidity

Premature (early) ejaculation may be associated with erectile problems. In many cases, it may be difficult to determine which difficulty preceded the other. Lifelong premature (early) ejaculation may be associated with certain anxiety disorders. Acquired premature (early) ejaculation may be associated with prostatitis, thyroid disease, or drug withdrawal (e.g., during opioid withdrawal).

Substance/Medication-Induced Sexual Dysfunction

Diagnostic Criteria

A. A clinically significant disturbance in sexual function is predominant in the clinical picture.

B. There is evidence from the history, physical examination, or laboratory findings of both (1) and (2):

1. The symptoms in Criterion A developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication.
2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by a sexual dysfunction that is not substance/medication-induced. Such evidence of an independent sexual dysfunction could include the following:

   The symptoms precede the onset of the substance/medication use; the symptoms persist for a substantial period of time (e.g., about 1 month) after the

cessation of acute withdrawal or severe intoxication; or there is other evidence suggesting the existence of an independent non-substance/medication-induced sexual dysfunction (e.g., a history of recurrent non-substance/medication-related episodes).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress in the individual.

Note: This diagnosis should be made instead of a diagnosis of substance intoxication or substance withdrawal only when the symptoms in Criterion A predominate in the clinical picture and are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance/medication]-induced sexual dysfunctions are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given. If a mild substance use disorder is comorbid with the substance-induced sexual dysfunction, the 4th position character is “1,” and the clinician should record “mild [substance] use disorder” before the substance-induced sexual dysfunction (e.g., “mild cocaine use disorder with cocaine-induced sexual dysfunction”). If a moderate or severe substance use disorder is comorbid with the substance-induced sexual dysfunction, the 4th position character is “2,” and the clinician should record “moderate [substance] use disorder” or “severe [substance] use disorder,” depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is “9,” and the clinician should record only the substance-induced sexual dysfunction.

	ICD-10-CM		
	With mild use disorder	**With moderate or severe use disorder**	**Without use disorder**
Alcohol	F10.181	F10.281	F10.981
Opioid	F11.181	F11.281	F11.981
Sedative, hypnotic, or anxiolytic	F13.181	F13.281	F13.981
Amphetamine-type substance (or other stimulant)	F15.181	F15.281	F15.981
Cocaine	F14.181	F14.281	F14.981
Other (or unknown) substance	F19.181	F19.281	F19.981

Specify (see Table 1 in the chapter "Substance-Related and Addictive Disorders," which indicates whether "with onset during intoxication" and/or "with onset during withdrawal" applies to a given substance class; or *specify* "with onset after medication use"):

With onset during intoxication: If criteria are met for intoxication with the substance and the symptoms develop during intoxication.

With onset during withdrawal: If criteria are met for withdrawal from the substance and the symptoms develop during, or shortly after, withdrawal.

With onset after medication use: If symptoms developed at initiation of medication, with a change in use of medication, or during withdrawal of medication.

Specify current severity:

Mild: Occurs on 25%–50% of occasions of sexual activity.

Moderate: Occurs on 50%–75% of occasions of sexual activity.

Severe: Occurs on 75% or more of occasions of sexual activity.

Recording Procedures

The name of the substance/medication-induced sexual dysfunction begins with the specific substance (e.g., alcohol) that is presumed to be causing the sexual dysfunction. The ICD-10-CM code that corresponds to the applicable drug class is selected from the table included in the criteria set. For substances that do not fit into any of the classes (e.g., fluoxetine), the ICD-10-CM code for the other (or unknown) substance class should be used and the name of the specific substance recorded (e.g., F19.981 fluoxetine-induced sexual dysfunction). In cases in which a substance is judged to be an etiological factor but the specific substance is unknown, the ICD-10-CM code for the other (or unknown) substance class is used and the fact that the substance is unknown is recorded (e.g., F19.981 unknown substance-induced sexual dysfunction).

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance-induced sexual dysfunction, followed by the specification of onset (i.e., onset during intoxication, onset during withdrawal, with onset after medication use), followed by the severity specifier (e.g., mild, moderate, severe). For example, in the case of erectile dysfunction occurring during intoxication in a man with a severe alcohol use disorder, the diagnosis is F10.281 severe alcohol use disorder with alcohol-induced sexual dysfunction, with onset during intoxication, moderate. A separate diagnosis of the comorbid severe alcohol use disorder is not given. If the substance-induced sexual dysfunction occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F15.981 amphetamine-induced sexual dysfunction, with onset during intoxication). When more than one substance is judged to play a significant role in the development of the sexual dysfunction, each should be listed separately (e.g., F14.181 mild cocaine use disorder with cocaine-induced sexual dysfunction, with onset during intoxication, moderate; F19.981 fluoxetine-induced sexual dysfunction, with onset after medication use, moderate).

Diagnostic Features

The essential features of substance/medication-induced sexual dysfunction are clinically significant disturbances in sexual function that are predominant in the clinical picture (Criterion A) that are judged to be due to the effects of a substance (e.g., a drug of abuse or medication). The sexual dysfunction must have developed during or soon after substance intoxication or withdrawal or after exposure to or withdrawal from a medication, and the substances or medications must be capable of producing the symptoms (Criterion B2). Substance/medication-induced sexual dysfunction due to a prescribed treatment for a

mental disorder or another medical condition must have its onset while the individual is receiving the medication (or during withdrawal, if a withdrawal is associated with the medication). Once the treatment is discontinued, the sexual dysfunction will usually improve or remit within days to several weeks (depending on the half-life of the substance/medication and the presence of withdrawal). The diagnosis of substance/medication-induced sexual dysfunction should not be given if the onset of the sexual dysfunction precedes the substance/medication intoxication or withdrawal, or if the symptoms persist for a substantial period of time (i.e., usually longer than 1 month) from the time of severe intoxication or withdrawal.

Associated Features

Sexual dysfunctions can occur in association with intoxication with the following classes of substances: alcohol; opioids; sedatives, hypnotics, or anxiolytics; stimulants (including cocaine); and other (or unknown) substances. Sexual dysfunctions can occur in association with withdrawal from the following classes of substances: alcohol; opioids; sedatives, hypnotics, or anxiolytics; and other (or unknown) substances. Medications that can induce sexual dysfunctions include antidepressants, antipsychotics, and hormonal contraceptives.

The most commonly reported side effect of antidepressant drugs is difficulty with orgasm or ejaculation in men, and with arousal in women.

Problems with desire and erection are less frequent. There is evidence that the effects of antidepressant medications on sexual dysfunction occur regardless of levels of depression. Approximately 30% of sexual complaints are clinically significant. Certain agents (i.e., bupropion, mirtazapine, nefazodone, and vilazodone) appear to have lower rates of sexual side effects than other antidepressants.

The sexual problems associated with antipsychotic drugs, including problems with sexual desire, erection, lubrication, ejaculation, or orgasm, have occurred with typical as well as atypical agents. However, problems are less common with prolactin-sparing antipsychotics or those that do not block dopamine receptors.

Although the effects of mood stabilizers on sexual function are unclear, it is possible that lithium and anticonvulsants, with the possible exception of lamotrigine, have adverse effects on sexual desire. Problems with orgasm may occur with gabapentin. Similarly, there may be a higher prevalence of erectile and orgasm problems associated with benzodiazepines. There have not been such reports with buspirone.

Many nonpsychiatric medications, such as cardiovascular, cytotoxic, gastrointestinal, and hormonal agents, are associated with disturbances in sexual function. Use of 5-α-reductase inhibitors (e.g., dutasteride, finasteride) may reduce erectile function, ejaculatory function, and libido in men.

Illicit substance use is associated with decreased sexual desire, erectile dysfunction, and difficulty reaching orgasm. Sexual dysfunctions are also seen in individuals receiving methadone but are seldom reported by individuals receiving buprenorphine. Chronic nicotine or chronic alcohol abuse are associated with erectile problems. Cannabis, like alcohol, is a central nervous system depressant, and its use may be a risk factor for sexual dysfunction; however, it has also been suggested to potentially improve satisfaction with orgasm.

Prevalence

The prevalence and the incidence of substance/medication-induced sexual dysfunction are unclear, likely because of underreporting of treatment-emergent sexual side effects. Data on substance/medication-induced sexual

dysfunction typically concern the effects of antidepressant drugs. The prevalence of antidepressant-induced sexual dysfunction varies in part depending on the specific agent. Approximately 25%–80% of individuals taking monoamine oxidase inhibitors, tricyclic antidepressants, serotonergic antidepressants, and combined serotonergic-adrenergic antidepressants report sexual side effects. There

are differences in the incidence of sexual side effects between some serotonergic and combined adrenergic-serotonergic antidepressants, with medications such as citalopram, fluoxetine, fluvoxamine, paroxetine, sertraline, and venlafaxine having the highest rates of sexual dysfunction.

Approximately 50% of individuals taking antipsychotic medications will experience adverse sexual side effects, including problems with sexual desire, erection, lubrication, ejaculation, or orgasm. The incidence of these side effects among different antipsychotic agents is unclear.

Exact prevalence and incidence of sexual dysfunctions among users of nonpsychiatric medications such as cardiovascular, cytotoxic, gastrointestinal, and hormonal agents are unknown. Elevated rates of sexual dysfunction have been reported high-dose opioid drugs for pain. There are increased rates of decreased sexual desire, erectile dysfunction, and difficulty reaching orgasm associated with illicit substance use. The prevalence of sexual problems appears related to chronic drug abuse and appears higher in individuals who abuse heroin (approximately 60%–70%) than in individuals who abuse amphetamine-type substances or 3,4-methylenedioxymethamphetamine (i.e., MDMA, ecstasy). Elevated rates of sexual dysfunction are also seen in individuals receiving methadone but are seldom reported by individuals receiving buprenorphine. Chronic alcohol abuse and chronic nicotine abuse are related to higher rates of erectile problems.

Development and Course

The onset of antidepressant-induced sexual dysfunction may be as early as 8 days after the agent is first taken. Approximately 30% of individuals with

mild to moderate orgasm delay will experience spontaneous remission of the dysfunction within 6 months. In some cases, serotonin reuptake inhibitor–induced sexual dysfunction may persist after the agent is discontinued. The time to onset of sexual dysfunction after initiation of antipsychotic drugs or drugs of abuse is unknown. It is probable that the adverse effects of nicotine and alcohol may not appear until after years of use. Premature (early) ejaculation can sometimes occur after cessation of opioid use. There is some evidence that disturbances in sexual function related to substance/medication use increase with age.

Culture-Related Diagnostic Issues

There may be an interaction among cultural factors, the influence of medications on sexual functioning, and the response of the individual to those changes.

Sex- and Gender-Related Diagnostic Issues

Some gender differences in sexual side effects from substances and medications may exist, such that men may more often report impairments with desire and orgasm following antidepressant use, and women may more often report difficulties with sexual arousal.

Functional Consequences of Substance/Medication-Induced Sexual Dysfunction

Medication-induced sexual dysfunction may result in medication noncompliance, such as stopping medications or using them irregularly, which could contribute to the lack of efficacy for antidepressants.

Differential Diagnosis

Non-substance/medication-induced sexual dysfunctions. Many mental disorders, such as depressive, bipolar, anxiety, and psychotic disorders, are associated with disturbances of sexual function. Thus, differentiating a substance/medication-induced sexual

dysfunction from a manifestation of the underlying mental disorder can be quite difficult. The diagnosis is usually established if a close relationship between substance/medication initiation or discontinuation is observed. A clear diagnosis can be established if the problem occurs after substance/medication initiation, dissipates with substance/medication discontinuation, and recurs with introduction of the same agent. Most substance/medication-induced side effects occur shortly after initiation or discontinuation. Sexual side effects that only occur after chronic use of a substance/medication may be extremely difficult to diagnose with certainty.

Other Specified Sexual Dysfunction

F52.8

This category applies to presentations in which symptoms characteristic of a sexual dysfunction that cause clinically significant distress in the individual predominate but do not meet the full criteria for any of the disorders in the sexual dysfunctions diagnostic class. The other specified sexual dysfunction category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific sexual dysfunction. This is done by recording “other specified sexual dysfunction” followed by the specific reason (e.g., “sexual aversion”).

Unspecified Sexual Dysfunction

F52.9

This category applies to presentations in which symptoms characteristic of a sexual dysfunction that cause clinically significant distress in the individual predominate but do not meet the full criteria for any of the disorders in the sexual dysfunctions

diagnostic class. The unspecified sexual dysfunction category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific sexual dysfunction, and includes presentations for which there is insufficient information to make a more specific diagnosis.

Gender Dysphoria

In this chapter, there is one overarching diagnosis of gender dysphoria, with separate developmentally appropriate criteria sets for children and for adolescents and adults. The area of sex and gender is highly controversial and has led to a proliferation of terms whose meanings vary over time and within and between disciplines. An additional source of confusion is that in English "sex" connotes both male/female and sexuality. This chapter employs constructs and terms as they are widely used by clinicians from various disciplines with specialization in treating gender dysphoria. In this chapter, *sex* and *sexual* refer to the biological indicators of male and female (understood in the context of reproductive capacity), such as in sex chromosomes, gonads, sex hormones, and nonambiguous internal and external genitalia. Disorders of sex development or differences of sex development (DSDs) included the historical terms *hermaphroditism* and *pseudohermaphroditism*. DSDs include somatic intersex conditions such as congenital development of ambiguous genitalia (e.g., clitoromegaly, micropenis), congenital disjunction of internal and external sex anatomy (e.g., complete androgen insensitivity syndrome), incomplete development of sex anatomy (e.g., gonadal agenesis), sex chromosome anomalies (e.g., Turner syndrome; Klinefelter syndrome), or disorders of gonadal development (e.g., ovotestes).

Gender is used to denote the public, sociocultural (and usually legally recognized) lived role as boy or girl, man or woman, or other gender. Biological factors are seen as contributing, in interaction with social and psychological factors, to gender development. *Gender assignment* refers to the assignment as male or female. This occurs usually at birth based on phenotypic sex and, thereby, yields the *birth-assigned gender*, historically referred to as "biological sex" or, more recently, "natal gender." *Birth-assigned sex* is often used interchangeably with birth-assigned gender. The

terms *assigned sex* and *assigned gender* encompass birth-assigned sex/gender but also include gender/sex assignments and reassignments made after birth but during infancy or early childhood, usually in the case of intersex conditions. *Gender-atypical* refers to somatic features or behaviors that are not typical (in a statistical sense) of individuals with the same assigned gender in a given society and historical era; *gender-nonconforming*, *gender variant*, and *gender diverse* are alternative nondiagnostic terms. *Gender reassignment* denotes an official (and sometimes legal) change of gender. *Gender-affirming treatments* are medical procedures (hormones or surgeries or both) that aim to align an individual's physical characteristics with their *experienced gender*. *Gender identity* is a category of social identity and refers to an individual's identification as male, female, some category in between (i.e., *gender fluid*), or a category other than male or female (i.e., *gender neutral*). There has been a proliferation of gender identities in recent years. *Gender dysphoria* as a general descriptive term refers to the distress that may accompany the incongruence between one's experienced or expressed gender and one's assigned gender. However, it is more specifically defined when used as a diagnostic category. It does not refer to distress related to stigma, a distinct although possibly co-occurring source of distress. *Transgender* refers to the broad spectrum of individuals whose gender identity is different from their birth-assigned gender. *Cisgender* describes individuals whose gender expression is congruent with their birth-assigned gender (also *nontransgender*). *Transsexual*, a historic term, denotes an individual who seeks, is undergoing,

or has undergone a social transition from male to female or female to male, which in many, but not all, cases also involves a somatic transition by gender-affirming hormone treatment and genital, breast, or other gender-affirming surgery (historically referred to as *sex reassignment surgery*).

Although not all individuals will experience distress from incongruence, many are distressed if the desired physical interventions using hormones and/or surgery are not available. The current term is more descriptive than

the previous DSM-IV term *gender identity disorder* and focuses on dysphoria as the clinical problem, not identity per se.

Gender Dysphoria

Diagnostic Criteria

Gender Dysphoria in Children — F64.2

A. A marked incongruence between one's experienced/expressed gender and assigned gender, of at least 6 months' duration, as manifested by at least six of the following (one of which must be Criterion A1):

1. A strong desire to be of the other gender or an insistence that one is the other gender (or some alternative gender different from one's assigned gender).
2. In boys (assigned gender), a strong preference for cross-dressing or simulating female attire; or in girls (assigned gender), a strong preference for wearing only typical masculine clothing and a strong resistance to the wearing of typical feminine clothing.
3. A strong preference for cross-gender roles in make-believe play or fantasy play.
4. A strong preference for the toys, games, or activities stereotypically used or engaged in by the other gender.
5. A strong preference for playmates of the other gender.
6. In boys (assigned gender), a strong rejection of typically masculine toys, games, and activities and a strong avoidance of rough-and-tumble play; or in girls (assigned gender), a strong rejection of typically feminine toys, games, and activities.
7. A strong dislike of one's sexual anatomy.

8. A strong desire for the primary and/or secondary sex characteristics that match one's experienced gender.

B. The condition is associated with clinically significant distress or impairment in social, school, or other important areas of functioning.

Specify if:

With a disorder/difference of sex development (e.g., a congenital adrenogenital disorder such as E25.0 congenital adrenal hyperplasia or E34.50 androgen insensitivity syndrome).

Coding note: Code the disorder/difference of sex development as well as gender dysphoria.

Gender Dysphoria in Adolescents and Adults F64.0

A. A marked incongruence between one's experienced/expressed gender and assigned gender, of at least 6 months' duration, as manifested by at least two of the following:

1. A marked incongruence between one's experienced/expressed gender and primary and/or secondary sex characteristics (or in young adolescents, the anticipated secondary sex characteristics).

2. A strong desire to be rid of one's primary and/or secondary sex characteristics because of a marked incongruence with one's experienced/expressed gender (or in young adolescents, a desire to prevent the development of the anticipated secondary sex characteristics).
3. A strong desire for the primary and/or secondary sex characteristics of the other gender.

4. A strong desire to be of the other gender (or some alternative gender different from one's assigned gender).
5. A strong desire to be treated as the other gender (or some alternative gender different from one's assigned gender).
6. A strong conviction that one has the typical feelings and reactions of the other gender (or some alternative gender different from one's assigned gender).

B. The condition is associated with clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

With a disorder/difference of sex development (e.g., a congenital adrenogenital disorder such as E25.0 congenital adrenal hyperplasia or E34.50 androgen insensitivity syndrome).

Coding note: Code the disorder/difference of sex development as well as gender dysphoria.

Specify if:

Posttransition: The individual has transitioned to full-time living in the experienced gender (with or without legalization of gender change) and has undergone (or is preparing to have) at least one gender-affirming medical procedure or treatment regimen—namely, regular gender-affirming hormone treatment or gender reassignment surgery confirming the experienced gender (e.g., breast augmentation surgery and/or vulvovaginoplasty in an individual assigned male at birth; transmasculine chest surgery and/or phalloplasty or metoidioplasty in an individual assigned female at birth).

Specifiers

The specifier "with a disorder/difference of sex development" should be used in the context of individuals who have a specific and codable

disorder/difference of sex development documented in their medical record.

The “posttransition” specifier may be used in the context of continuing treatment procedures that serve to support the new gender assignment.

Diagnostic Features

Individuals with gender dysphoria have a marked incongruence between the gender to which they have been assigned (usually based on phenotypic sex at birth, referred to as *birth-assigned gender*) and their experienced/expressed gender. This discrepancy is the core component of the diagnosis. There must also be evidence of distress about this incongruence. Experienced gender may include alternative gender identities beyond binary stereotypes. Consequently, distress may involve not only the experience that the individual is a male or female gender other than the one assigned at birth but also an experience that the individual is an intermediate or alternative gender that differs from the individual’s birth-assigned gender.

Gender dysphoria manifests itself differently in different age groups. The following examples may be less prominent in children raised in surroundings with fewer gender stereotypes.

Prepubertal individuals assigned female at birth with gender dysphoria may express a marked, persistent feeling or conviction that they are a boy, express aversion to the idea of

being a girl, or assert they will grow up to be a man. They often prefer boys’ clothing and hairstyles, may be perceived by strangers as boys, and may ask to be called by a boy’s name. Sometimes they display intense negative reactions to parental attempts to have them wear dresses or other feminine attire. Some may refuse to attend school or social events where such clothes are required. These children may demonstrate marked gender nonconformity in role-playing, dreams, gender-typed play and toy preferences, styles, mannerisms, fantasies, and peer preferences. Contact sports, rough-and-tumble play, traditional boyhood games, and boys as playmates are most often preferred. They show little interest in

stereotypically feminine toys (e.g., dolls) or activities (e.g., feminine dress-up or role-play). Occasionally, they refuse to urinate in a sitting position. Some may express a desire to have a penis or claim to have a penis or that they will grow one when older. They may also state that they do not want to develop breasts or menstruate.

Prepubertal individuals assigned male at birth with gender dysphoria may express a marked, persistent feeling or conviction that they are a girl or assert that they will grow up to be a woman. They may express aversion to the idea of being a boy. They often prefer dressing in girls' or women's clothes or may improvise clothing from available materials (e.g., using towels, aprons, and scarves for long hair or skirts). These children may demonstrate marked gender nonconformity in gender-typed play and toy preferences, styles, mannerisms, and peer preferences. They may role-play female figures (e.g., playing "mother") and may be intensely interested in female fantasy figures. Traditional feminine activities, stereotypical games, and pastimes (e.g., "playing house"; drawing feminine pictures; watching television or videos of favorite female characters) may be preferred. Stereotypical female-type dolls (e.g., Barbie) may be favorite toys, and girls are their preferred playmates. They avoid rough-and-tumble play and have little interest in stereotypically masculine toys (e.g., cars, trucks). They may state that they find their penis or testes disgusting, that they wish them removed, or that they have, or wish to have, a vagina.

Increasingly, parents are presenting to specialized clinics after their child with gender dysphoria has already socially transitioned.

As the onset of puberty for individuals assigned female at birth is somewhere between ages 9 and 13, and between 11 and 14 for individuals assigned male at birth, their symptoms and concerns may arise in a developmental phase somewhere between childhood and adolescence. As secondary sex characteristics of younger adolescents are not yet fully developed, these individuals may not state dislike of them, but they may be markedly distressed by imminent physical changes.

In adolescents and adults with gender dysphoria, the discrepancy between experienced gender and physical sex characteristics is often, but not always, accompanied by a desire to be rid of primary and/or secondary sex characteristics and/or a strong desire to acquire some primary and/or secondary sex characteristics of another gender. To varying degrees, older

adolescents and adults with gender dysphoria may adopt the behavior, clothing, and mannerisms of their experienced gender. They feel uncomfortable being regarded by others, or functioning in society, as members of their assigned gender. Some adults and adolescents may have a strong desire to be of a different gender and treated as such, and they may have an inner certainty to feel and respond as their experienced gender without seeking medical treatment to alter body characteristics. They may find other ways to resolve the incongruence between experienced/expressed and assigned gender by partially living in the desired role or by adopting a gender role neither conventionally male nor conventionally female.

Associated Features

When visible signs of puberty develop, individuals assigned male at birth may shave their facial, body, and leg hair at the first signs of growth. They sometimes bind their genitals to make erections less visible. Individuals assigned female at birth may bind their breasts,

walk with a stoop, or use loose sweaters to make breasts less visible. Increasingly, adolescents request, or may obtain without medical prescription and supervision, drugs that suppress production of gonadal steroids (e.g., gonadotropin-releasing hormone [GnRH] agonists) or that block gonadal hormone actions (e.g., spironolactone). Clinically referred adolescents often want hormone treatment and many also wish for gender-affirming surgery. Adolescents living in an accepting environment may openly express the desire to be and be treated as their experienced gender and dress partly or completely as their experienced gender, have a hairstyle typical of their experienced gender, preferentially seek friendships with peers of another gender, and/or adopt a new first name consistent with their experienced gender. Older adolescents, when sexually active, often do not show or allow partners to touch their sexual organs. For adults with an aversion toward their genitals, sexual activity is constrained by the preference that their genitals not be seen or touched by their partners. Not infrequently, adults may seek hormone treatment (sometimes without

medical prescription and supervision) and gender-affirming surgery. Others are satisfied with either hormone treatment or surgery alone, or without any gender-affirming medical treatment.

In children, adolescents, and adults with gender dysphoria, an overrepresentation of autism spectrum traits has been observed. Also, individuals with autism spectrum disorder are more likely to exhibit gender diversity.

Adolescents and adults with gender dysphoria before gender-affirming treatment and legal gender change are at increased risk for mental health problems including suicidal ideation, suicide attempts, and suicides. After gender reassignment, adjustment may vary, and suicide risk and mental health problems may persist.

In prepubertal children, increasing age is associated with having more behavioral or emotional problems; this is related to the increasing nonacceptance of gender-nonconforming behavior by others. Children and adolescents who feel supported and accepted in their gender nonconformity may show less or even no psychological problems.

Prevalence

There are no large-scale population studies of gender dysphoria. Based on gender-affirming treatment–seeking populations, the prevalence for gender dysphoria diagnosis across populations has been assessed to be less than 1/1,000 (i.e., < 0.1%) for both individuals assigned male at birth and individuals assigned female at birth. Because many adults with gender dysphoria do not seek care at specialty treatment programs, prevalence rates are likely underestimates. Prevalence estimates based on surveys of self-reporting general population samples in the United States and Europe suggest higher numbers, although varied methods of assessment make comparisons difficult across studies. Self-identification as transgender ranges from 0.5% to 0.6%; experiencing oneself as having an incongruent gender identity ranges from 0.6% to 1.1%; feeling that one is a person of a different sex ranges from 2.1% to 2.6%; and the desire to undergo medical treatment ranges from 0.2% to 0.6%.

Development and Course

Because expression of gender dysphoria varies with age, there are separate criteria sets for children versus those for adolescents and adults. Criteria for children are defined in a more concrete, behavioral manner than those for adolescents and adults. Young children are less likely than older children, adolescents, and adults to express extreme and persistent anatomic dysphoria. In adolescents and adults, incongruence between experienced gender and assigned gender is a central feature of the diagnosis. Factors related to distress and impairment also vary with age. A very young child may show signs of distress (e.g., intense crying) only when parents tell the child that he or she is "really" not a member of another gender but only "desires" to be. Distress may not be manifest in social environments supportive of the child's gender nonconformity and may emerge only if there is

parental/social interference with the child's gender variance. In adolescents and adults, distress may manifest because of strong incongruence between experienced gender and birth-assigned gender. Such distress may, however, be mitigated by supportive environments and knowledge that biomedical treatments exist to reduce incongruence. Impairment (e.g., school refusal, development of depression, anxiety, peer and behavioral problems, and substance abuse) may be a correlate of gender dysphoria.

Gender dysphoria without a disorder of sex development. For clinic-referred children studied in Canada and the Netherlands, onset of gender-nonconforming behaviors is usually between ages 2 and 4 years. This corresponds to the developmental time period in which most children begin expressing gendered behaviors and interests. For some preschool-age children, both marked, persistent gender-atypical behaviors and the expressed desire to be another gender may be present, or labeling themselves as a member of another gender may occur. In other cases, the gender expression appears later, usually at entry into elementary school. Children may sometimes express discomfort with their sexual anatomy or will state the desire to have a sexual anatomy corresponding to their experienced gender ("anatomic dysphoria"). Expressions of anatomic

dysphoria become more common as children with gender dysphoria approach and anticipate puberty.

No general population studies exist of adolescent or adult outcomes of childhood gender variance. Some prepubescent children expressing a desire to be another gender will not seek gender-affirming somatic treatments when they reach puberty. They frequently report nonheterosexual orientations and frequently marked gender-nonconforming behavior, although not necessarily a transgender identity in adolescence/young adulthood. Some children with gender dysphoria in childhood that remits in adolescence may experience a recurrence in adulthood.

In individuals assigned male at birth, studies from North America and the Netherlands found persistence ranged from 2% to 39%. In individuals assigned female at birth, persistence ranged from 12% to 50%. Persistence of gender dysphoria is modestly correlated with dimensional measures of severity ascertained at the time of a childhood baseline assessment. Early social transition may also be a factor in persistence of gender dysphoria in adolescence.

Studies have shown a high incidence of sexual attraction to those of the individual's birth-assigned gender, regardless of the trajectory of the prepubescent child's gender dysphoria. For individuals whose gender dysphoria continues into adolescence and beyond, most self-identify as heterosexual. In those who no longer have gender dysphoria by the time of adolescence, a majority self-identify as gay, lesbian, or bisexual.

Two broad trajectories have been described for development of gender dysphoria in individuals who identify as either male or female.

As opposed to gender-nonconforming children, individuals with prepubertal-onset gender dysphoria have symptoms that meet diagnostic criteria for gender dysphoria in childhood. The dysphoria can continue into adolescence and adulthood; alternatively, some individuals go through a period in which the gender dysphoria either desists or is denied. At such times, these individuals may self-identify as being gay or lesbian. Some may identify as heterosexual and cisgender. However, it is possible that some of these individuals may experience a recurrence of gender dysphoria later in life.

Regardless of whether the individual's gender dysphoria persists or desists at a later date, either the onset of puberty or the realization that

puberty will begin with development of secondary sex characteristics can prompt distressing feelings of gender incongruence that can exacerbate the individual's gender dysphoria.

The early/prepubertal-onset group often present for clinical, gender-affirming care during childhood, during adolescence, or in young adulthood. This may reflect a more intense gender dysphoria compared with individuals with late/postpubertal-onset gender dysphoria, whose distress may be more variable and less intense.

Late-onset or pubertal/postpubertal-onset gender dysphoria occurs around puberty or even much later in life. Some of these individuals report having had a desire to be of another gender in childhood that was not expressed verbally to others or had gender-nonconforming behavior that did not meet full criteria for gender dysphoria in childhood. Others have no recollection of any signs of childhood gender dysphoria. Parents of individuals with gender dysphoria of pubertal/postpubertal-onset often report surprise, as they saw no signs of gender dysphoria during childhood.

Gender dysphoria in association with a disorder of sex development. Individuals with DSDs who require early medical intervention or decisions about gender assignment come to clinical attention at an early age. Depending on the condition, they may have been gonadectomized (often because of risk of future malignancy) before puberty so that administration of exogenous hormones is part of routine care to induce puberty. Infertility is common whether due to the condition itself or to gonadectomy, and genital surgery may have been done in infancy or childhood with the intent of affirming the assigned gender to both the affected individual and caregivers.

Affected individuals may exhibit gender-nonconforming behavior starting in early childhood in a manner that is predictable depending on the specific DSD syndrome and the gender assignment, and thresholds for supporting social and medical gender transition in minors have traditionally been much lower for those with compared to those without DSDs. As individuals with some DSD syndromes become aware of their condition and medical history, many experience uncertainty about their gender, as opposed to developing a firm conviction that they are of another gender.

The proportion who develop gender dysphoria and progress to gender transition varies markedly depending on the particular syndrome and gender assignment.

Risk and Prognostic Factors

Temperamental. Gender-variant behavior among individuals with prepubertal-onset gender dysphoria can develop in early preschool age. Studies suggest that a greater intensity of gender nonconformity and an older age at presentation make persistence of gender dysphoria into adolescence and adulthood more likely. A predisposing factor under consideration, especially in individuals with postpubertal-onset gender dysphoria (adolescence, adulthood), includes history of transvestism that may develop into autogynephilia (i.e., sexual arousal associated with the thought or image of oneself as a woman).

Environmental. Individuals assigned male at birth with gender dysphoria without a DSD (in both childhood and adolescence) more commonly have older brothers when compared with cisgender males.

Genetic and physiological. For individuals with gender dysphoria without a DSD, some genetic contribution is suggested by evidence for (weak) familiality of gender dysphoria among nontwin siblings, increased concordance for gender dysphoria in monozygotic compared with dizygotic same-sex twins, and some degree of heritability of gender dysphoria. Research suggests that gender dysphoria has a polygenetic basis involving interactions of several genes and polymorphisms that may affect in utero sexual differentiation of the brain, contributing to gender dysphoria in individuals assigned male at birth.

As to endocrine findings in individuals with gender dysphoria, no endogenous systemic abnormalities in sex-hormone levels have been found in 46,XY individuals, whereas there appear to be increased androgen levels (in the range found in hirsute women but far below normal male levels) in 46,XX individuals. Overall, current evidence is insufficient to label gender dysphoria without a DSD as a form of intersexuality limited to the central nervous system.

In gender dysphoria associated with a DSD, the likelihood of later gender dysphoria is increased if prenatal production and utilization (via

receptor sensitivity) of androgens are grossly variant relative to what is usually seen in individuals with the same assigned

gender. Examples include 46,XY individuals with a history of normal male prenatal hormone milieu but inborn nonhormonal genital defects (as in cloacal bladder exstrophy or penile agenesis) and who have been assigned to the female gender. The likelihood of gender dysphoria is further enhanced by additional, prolonged, highly gender-variant postnatal androgen exposure with somatic virilization as may occur in female-raised and noncastrated 46,XY individuals with 5-alpha reductase-2 deficiency or 17-beta-hydroxysteroid dehydrogenase-3 deficiency or in female-raised 46,XX individuals with classical congenital adrenal hyperplasia with prolonged periods of nonadherence to glucocorticoid replacement therapy. However, the prenatal androgen milieu is more closely related to gendered behavior than to gender identity. Many individuals with DSDs and markedly gender-variant behavior do not develop gender dysphoria. Thus, gender-nonconforming behavior by itself should not be interpreted as an indicator of current or future gender dysphoria. There appears to be a higher rate of gender dysphoria and patient-initiated gender change from assigned female to male than from assigned male to female in individuals prenatally exposed to a full complement of masculinizing hormonal influences.

Culture-Related Diagnostic Issues

Individuals with gender dysphoria have been reported across many countries and cultural contexts around the world. The equivalent of gender dysphoria has also been reported in individuals living in cultural contexts with institutionalized gender identity categories other than men/boys or women/girls that sanction gender nonconforming development. These include India, Sri Lanka, Myanmar, Oman, Samoa, Thailand, and Indigenous Peoples of North America. It is unclear however, in such cultural contexts, whether the diagnostic criteria for gender dysphoria would be met with these individuals.

The prevalence of coexisting mental health problems differs among cultures; these differences may also be related to differences in attitudes toward gender nonconformity in children, adolescents, and adults. However, also in some non-Western cultures, anxiety has been found to be relatively common in individuals with gender dysphoria, even in cultures with accepting attitudes toward gender-variant behavior.

Sex- and Gender-Related Diagnostic Issues

Sex differences in rate of referrals to specialty clinics vary by age group. In children, sex ratios of individuals assigned male at birth to individuals assigned female at birth range from 1.25:1 to 4.3:1. Studies show increasing numbers of children and adolescents presenting to specialty clinics, presentation at younger ages, more frequent early social transition, and a shift to a greater number of individuals assigned female at birth in adolescents and young adults than individuals assigned male at birth. In adults, estimates generally suggest more individuals assigned male at birth seek gender-affirming treatment, with ratios ranging from 1:1 to 6.1:1 in most studies in the United States and Europe.

Association With Suicidal Thoughts or Behavior

Rates of suicidality and suicide attempts for transgender individuals are reported to range from 30% to 80%, with risk factors including past maltreatment, gender victimization, depression, substance abuse, and younger age. Transgender adolescents referred to gender clinics have substantially higher rates of suicidal thoughts and behaviors when compared with nonreferred adolescents. Prior to receiving gender-affirming treatment and legal gender reassignment, adolescents and adults with gender dysphoria are at increased risk for suicidal thoughts and suicide attempts. After gender-affirming treatment, adjustment varies, and while improvement in coexisting symptoms is often seen, some individuals continue to experience prominent anxiety and affective symptoms and remain at increased risk for suicide.

A study of 572 children referred for gender identity concerns in Canada and several comparison groups (siblings, other referred children, and nonreferred children) largely from other high-income countries found that gender-referred children were 8.6 times more likely to self-harm or attempt suicide than comparison children, even after adjustment for overall behavior and peer relationship problems, and particularly in the second half of childhood. Among adolescents, the highest rate of suicide attempt is among transgender young men, followed by those defining themselves as neither male nor female.

Functional Consequences of Gender Dysphoria

Gender nonconformity may appear at all ages after the first 2–3 years of childhood and may interfere with daily activities. In older children, gender nonconformity may affect peer relationships and may lead to isolation from peer groups and to distress. Many children experience teasing and harassment or pressure to dress in attire associated with their birth-assigned sex, especially when growing up in a nonsupportive and nonaccepting environment. Also in adolescents and adults, the distress resulting from gender incongruence often interferes with daily activities. Relationship difficulties, including sexual relationship problems, are common, and functioning at school or at work may be impaired. Gender dysphoria is associated with high levels of stigmatization, discrimination, and victimization, leading to negative self-concept, increased rates of depression, suicidality, and other mental disorder co-occurrence, school dropout, and economic marginalization, including unemployment, with attendant social and mental health risks, especially in individuals who lack family or social support. In addition, these individuals' access to health services and mental health services may be impeded by structural barriers, such as institutional discomfort about, inexperience with, or hostility toward working with this patient population.

Differential Diagnosis

Nonconformity to gender roles. Gender dysphoria should be distinguished from simple nonconformity to stereotypical gender role behavior by the strong desire to be of another gender than the assigned one and by the

extent and pervasiveness of gender-variant activities and interests. The diagnosis is not meant to merely describe nonconformity to stereotypical gender role behavior (e.g., “tomboyism” in girls, “girly-boy” behavior in boys, occasional cross-dressing in adult men). Given the increased openness of gender-diverse expressions by individuals across the entire range of the transgender spectrum, it is important that the clinical diagnosis be limited to those individuals whose distress and impairment meet the specified criteria.

Transvestic disorder. Transvestic disorder is diagnosed in heterosexual (or bisexual) adolescent and adult males (rarely in females) for whom women’s clothing generates sexual excitement and causes distress and/or impairment without drawing their assigned gender into question. It is occasionally accompanied by gender dysphoria. An individual with transvestic disorder who also has clinically significant gender dysphoria can be given both diagnoses. In some cases of postpubertal-onset gender dysphoria in individuals assigned male at birth who are attracted to women, cross-dressing with sexual excitement is a precursor to the diagnosis of gender dysphoria.

Body dysmorphic disorder. An individual with body dysmorphic disorder focuses on the alteration or removal of a specific body part because it is perceived as abnormally formed, not because it represents a repudiated assigned gender. When an individual’s presentation meets criteria for both gender dysphoria and body dysmorphic disorder, both diagnoses can be given. Individuals wishing to have a healthy limb amputated (termed by some *body integrity identity disorder*) because it makes them feel more “complete” usually do not wish to change gender, but rather desire to live as an amputee or a disabled person.

Autism spectrum disorder. In individuals with autism spectrum disorder, diagnosing gender dysphoria can be challenging. It can be difficult to differentiate potential co-occurring

gender dysphoria from an autistic preoccupation because of the concrete and rigid thinking around gender roles and/or poor understanding of social relationships characteristic of autism spectrum disorder.

Schizophrenia and other psychotic disorders. In schizophrenia, there may rarely be delusions of belonging to some other gender. In the absence of psychotic symptoms, insistence by an individual with gender dysphoria that he or she is another gender is not considered a delusion. Schizophrenia (or other psychotic disorders) and gender dysphoria may co-occur. Gender-themed delusions may occur in up to 20% of individuals with schizophrenia. They can usually be differentiated from gender dysphoria by their bizarre content and by waxing and waning with remissions and exacerbations of psychotic episodes.

Other clinical presentations. Some individuals with an emasculinization desire who develop an alternative, nonmale/nonfemale gender identity do have a presentation that meets criteria for gender dysphoria. However, some males seek genital surgery for either aesthetic reasons or to remove psychological effects of androgens without changing male identity; in these cases, the criteria for gender dysphoria are not met.

Comorbidity

Clinically referred children with gender dysphoria show elevated levels of anxiety, disruptive, impulse-control, and depressive disorders. Autism spectrum disorder is more prevalent in clinically referred adolescents and adults with gender dysphoria than in the general population. Clinically referred adolescents and adults with gender dysphoria often have high rates of associated mental disorders, with anxiety and depressive disorders being the most common. Individuals who have experienced harassment and violence may also develop posttraumatic stress disorder.

Other Specified Gender Dysphoria

F64.8

This category applies to presentations in which symptoms characteristic of gender dysphoria that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for gender

dysphoria. The other specified gender dysphoria category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for gender dysphoria. This is done by recording "other specified gender dysphoria" followed by the specific reason (e.g., "brief gender dysphoria," in which symptoms meet full criteria for gender dysphoria but the duration is less than the required 6 months).

Unspecified Gender Dysphoria

F64.9

This category applies to presentations in which symptoms characteristic of gender dysphoria that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for gender dysphoria. The unspecified gender dysphoria category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for gender dysphoria, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Disruptive, Impulse-Control, and Conduct Disorders

Disruptive, impulse-control, and conduct disorders include conditions involving problems in the self-control of emotions and behaviors. While other disorders in DSM-5 may also involve problems in emotional and/or behavioral regulation, the disorders in this chapter are unique in that these problems are manifested in behaviors that violate the rights of others (e.g., aggression, destruction of property) and/or that bring the individual into significant conflict with societal norms or authority figures. The underlying causes of the problems in the self-control of emotions and behaviors can vary greatly across the disorders in this chapter and among individuals within a given diagnostic category.

The chapter includes oppositional defiant disorder, intermittent explosive disorder, conduct disorder, antisocial personality disorder (which is described in the chapter "Personality Disorders"), pyromania, kleptomania, and other specified and unspecified disruptive, impulse-control, and conduct disorders. Although all the disorders in the chapter involve problems in both emotional and behavioral regulation, the source of variation among the disorders is the relative emphasis on problems in the two types of self-control. For example, the criteria for conduct disorder focus largely on poorly controlled behaviors that violate the rights of others or that violate major societal norms. These behaviors may or may not result from poorly controlled emotions. Some symptoms of conduct disorder (e.g., certain forms of aggression) can be attributable to constricted emotional responses. At the other extreme, the criteria for intermittent explosive disorder focus largely on poorly controlled emotion, outbursts of anger that are disproportionate to the interpersonal or other provocation or to other psychosocial stressors.

Intermediate in impact to these two disorders is oppositional defiant disorder, in which the criteria are more evenly distributed between emotions (anger and irritation) and behaviors (argumentativeness and defiance). Pyromania and kleptomania are characterized by poor impulse control related to specific behaviors (fire setting or stealing) that relieve internal tension. Other specified disruptive, impulse-control, and conduct disorder is a category for conditions in which there are symptoms of conduct disorder, oppositional defiant disorder, or other disruptive, impulse-control, and conduct disorders, but the number or type of symptoms does not meet the diagnostic threshold for any of the disorders in this chapter, even though there is evidence of clinically significant impairment associated with the symptoms.

The disruptive, impulse-control, and conduct disorders all tend to be more common in boys and men than in girls and women, although the relative degree of male predominance may differ both across disorders and within a disorder at different ages. The disorders in this chapter tend to have first onset in childhood or adolescence. In fact, it is very rare for either conduct disorder or oppositional defiant disorder to first emerge in adulthood. There is a developmental relationship between oppositional defiant disorder and conduct disorder, in that most cases of conduct disorder previously would have had symptoms that met criteria for oppositional defiant disorder, at least in those cases in which conduct disorder emerges prior to adolescence. However, most children with oppositional defiant disorder do not eventually develop conduct disorder. Furthermore, children with

oppositional defiant disorder are at risk for eventually developing other problems besides conduct disorder, including anxiety and depressive disorders.

Many of the symptoms that define the disruptive, impulse-control, and conduct disorders are behaviors that can occur to some degree in typically developing persons. Thus, it is critical that the frequency, persistence, pervasiveness across situations, and impairment associated with the behaviors indicative of the diagnosis be considered relative to what is

normative for a person's age, gender, and culture when determining if they are symptomatic of a disorder.

The disruptive, impulse-control, and conduct disorders have been linked to a common externalizing spectrum associated with the personality dimensions of *disinhibition* and *negative emotionality* (some facets); and inversely with *constraint* and *agreeableness*. These shared personality dimensions could account for the high level of comorbidity among these disorders and their frequent comorbidity with substance use disorders and antisocial personality disorder. However, the specific nature of the shared diathesis that constitutes the externalizing spectrum remains unknown.

Oppositional Defiant Disorder

Diagnostic Criteria F91.3

A. A pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness lasting at least 6 months as evidenced by at least four symptoms from any of the following categories, and exhibited during interaction with at least one individual who is not a sibling.

Angry/Irritable Mood

1. Often loses temper.
2. Is often touchy or easily annoyed.
3. Is often angry and resentful.

Argumentative/Defiant Behavior

4. Often argues with authority figures or, for children and adolescents, with adults.
5. Often actively defies or refuses to comply with requests from authority figures or with rules.
6. Often deliberately annoys others.
7. Often blames others for his or her mistakes or misbehavior.

Vindictiveness

8. Has been spiteful or vindictive at least twice within the past 6 months.

Note: The persistence and frequency of these behaviors should be used to distinguish a behavior that is within normal limits from a behavior that is symptomatic. For children younger than 5 years, the behavior should occur on most days for a period of at least 6 months unless otherwise noted (Criterion A8). For individuals 5 years or older, the behavior should occur at least once per week for at least 6 months, unless otherwise noted (Criterion A8). While these frequency criteria provide guidance on a minimal level of frequency to define symptoms, other factors should also be considered, such as whether the frequency and intensity of the behaviors are outside a range that is normative for the individual's developmental level, gender, and culture.

B. The disturbance in behavior is associated with distress in the individual or others in his or her immediate social context (e.g., family, peer group, work colleagues), or it impacts negatively on social, educational, occupational, or other important areas of functioning.

C. The behaviors do not occur exclusively during the course of a psychotic, substance use, depressive, or bipolar disorder. Also, the criteria are not met for disruptive mood dysregulation disorder.

Specify current severity:

Mild: Symptoms are confined to only one setting (e.g., at home, at school, at work, with peers).

Moderate: Some symptoms are present in at least two settings.

Severe: Some symptoms are present in three or more settings.

Specifiers

It is not uncommon for individuals with oppositional defiant disorder to show symptoms only at home and only with family members. However, the pervasiveness of the symptoms is an indicator of the severity of the disorder.

Diagnostic Features

The essential feature of oppositional defiant disorder is a frequent and persistent pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness (Criterion A). It is not unusual for individuals with oppositional defiant disorder to show the behavioral features of the disorder without problems of negative mood. However, individuals with the disorder who show the angry/irritable mood symptoms typically show the behavioral features as well.

The symptoms of oppositional defiant disorder may be confined to only one setting, and this is most frequently the home. Individuals who show enough symptoms to meet the diagnostic threshold, even if it is only at home, may be significantly impaired in their social functioning. However, in more severe cases, the symptoms of the disorder are present in multiple settings. Given that the pervasiveness of symptoms is an indicator of the severity of the disorder, it is critical that the individual's behavior be assessed across multiple settings and relationships. Because these behaviors are common among siblings, they must be observed during interactions with persons other than siblings. Also, because symptoms of the disorder are typically more evident in interactions with adults or peers whom the individual knows well, they may not be apparent during a clinical examination.

The symptoms of oppositional defiant disorder can occur to some degree in persons without this disorder. There are several key considerations for determining if the behaviors are symptomatic of oppositional defiant disorder. First, the diagnostic threshold of four or more

symptoms within the preceding 6 months must be met. Second, the persistence and frequency of the symptoms should exceed what is normative for an individual's age, gender, and culture. Temper outbursts for a preschool child would be considered a symptom of oppositional defiant disorder only if they occurred on most days for the preceding 6 months, if they occurred with at least three other symptoms of the disorder, and if the temper outbursts contributed to the significant impairment associated with the disorder (e.g., led to destruction of property during outbursts, resulted in the child being asked to leave a preschool). It should be noted that temper loss need not always involve tantrum behavior and can be displayed by angry facial expressions, verbal expressions of anger, and subjective feelings of anger that would not typically be considered a tantrum.

The symptoms of the disorder often are part of a pattern of problematic interactions with others. Furthermore, individuals with this disorder typically do not regard themselves as angry, oppositional, or defiant. Instead, they often justify their behavior as a response to unreasonable demands or circumstances. Thus, it can be difficult to disentangle the relative contribution of the individual with the disorder to the problematic interactions he or she experiences. For example, children with oppositional defiant disorder may have

experienced a history of hostile parenting, and it is often impossible to determine if the child's behavior caused the parents to act in a more hostile manner toward the child, if the parents' hostility led to the child's problematic behavior, or if there was some combination of both. Whether or not the clinician can separate the relative contributions of potential causal factors should not influence whether the diagnosis is made. In the event that the child may be living in particularly poor conditions where neglect or mistreatment may occur (e.g., in institutional settings), clinical attention to reducing the contribution of the environment may be helpful.

Associated Features

Two of the most common co-occurring conditions with oppositional defiant disorder are attention-deficit/hyperactivity disorder (ADHD) and conduct disorder (see the section “Comorbidity” for this disorder). Oppositional defiant disorder has been associated with increased risk for suicide attempts, even after comorbid disorders are controlled for.

Prevalence

The cross-national prevalence of oppositional defiant disorder ranges from 1% to 11%, with an average prevalence estimate of around 3.3%. The rate of oppositional defiant disorder may vary depending on the age and gender of the child. The disorder appears to be somewhat more prevalent in boys than in girls (1.59:1) prior to adolescence. This male predominance is not consistently found in samples of adolescents or adults.

Development and Course

The first symptoms of oppositional defiant disorder usually appear during the preschool years and rarely later than early adolescence. Oppositional defiant disorder often precedes the development of conduct disorder, especially for those with the childhood-onset type of conduct disorder. However, many children and adolescents with oppositional defiant disorder do not subsequently develop conduct disorder. Oppositional defiant disorder also conveys risk for the development of anxiety disorders and major depressive disorder, even in the absence of conduct disorder. The defiant, argumentative, and vindictive symptoms carry most of the risk for conduct disorder, whereas the angry/irritable mood symptoms carry most of the risk for mood and anxiety disorders.

Manifestations of the disorder across development appear consistent. Children and adolescents with oppositional defiant disorder are at increased risk for a number of problems in adjustment as adults, including functional impairments (e.g., problems in relationships with family, peers, and romantic partners; lower educational attainment; more workplace stress), the persistence of oppositional defiant disorder, and other psychopathology, such as antisocial behavior, impulse-control problems, substance misuse, anxiety, and depression.

Many of the behaviors associated with oppositional defiant disorder increase in frequency during the preschool period and in adolescence. Thus, it is especially critical during these development periods that the frequency and intensity of these behaviors be evaluated against normative levels before it is decided that they are symptoms of oppositional defiant disorder. For example, it is not unusual for preschool children to show temper tantrums on a weekly basis, but daily tantrums occur in only about 10% of preschool children.

Risk and Prognostic Factors

Temperamental. Temperamental factors related to problems in emotional regulation (e.g., high levels of emotional reactivity, poor frustration tolerance) have been predictive of the disorder.

Environmental. Children with oppositional defiant disorder influence their environments, which in turn can influence them. For example, harsh, inconsistent, or neglectful child-rearing practices predict increases in symptoms, and oppositional symptoms predict increases in harsh and inconsistent parenting. In children and adolescents, oppositional defiant disorder is more prevalent in families in which childcare is disrupted by a succession of different caregivers. Children with oppositional defiant disorder are also at greater risk for both bullying peers and being bullied by peers.

Genetic and physiological. A number of neurobiological markers (e.g., lower heart rate and skin conductance reactivity; reduced basal cortisol reactivity; abnormalities in the prefrontal cortex and amygdala) have been associated with oppositional defiant disorder. Studies have demonstrated overlapping genetic influences for the irritability and anger symptoms of oppositional defiant disorder with depression and generalized anxiety disorder. To date, the vast majority of studies have not separated children with oppositional defiant disorder from those with conduct disorder. Further study of markers specific to oppositional defiant disorder is needed.

Culture-Related Diagnostic Issues

The reported prevalence of oppositional defiant disorder or other disruptive disorders may be affected by misdiagnosis or overdiagnosis of individuals from some cultural backgrounds. Social norms may affect the prevalence of the disorder and its male gender predominance in children and adolescents. A meta-analysis of prevalence rates in middle childhood found that the disorder is more common in boys compared with girls in Western cultures, but that the prevalence is similar across genders in non-Western cultures. Also, despite adverse experiences, first-generation migrants and refugees may be at decreased risk of developing oppositional defiant disorder symptoms.

Sex- and Gender-Related Diagnostic Issues

Some studies find few sex or gender differences for this disorder compared with, for example, conduct disorder. There may be slight differences in risk factors with harsh parenting more highly associated with oppositional defiant disorder in girls but not boys.

Functional Consequences of Oppositional Defiant Disorder

When oppositional defiant disorder is persistent throughout development, individuals with the disorder experience frequent conflicts with parents, teachers, supervisors, peers, and romantic partners. Such problems often result in significant impairments in the individual's emotional, social, academic, and occupational adjustment.

Differential Diagnosis

Conduct disorder. Conduct disorder and oppositional defiant disorder are both related to conduct problems that bring the individual in conflict with adults and other authority figures (e.g., teachers, work supervisors). The behaviors of oppositional defiant disorder are typically of a less severe nature than those of conduct disorder and do not include aggression toward people or animals, destruction of property, or a pattern of theft or deceit. However, evidence suggests that oppositional defiant disorder is associated

with equivalent or even greater levels of impairment than conduct disorder. Furthermore, oppositional defiant disorder includes problems of emotion dysregulation (i.e., angry and irritable mood) that are not included in the definition of conduct disorder.

Adjustment disorder. Environmental and family stressors may be associated with externalizing manifestations of emotion dysregulation. In children, these may manifest as

tantrums and oppositional behavior; and in adolescents, as aggressive behaviors (e.g., rebellion and defiance). Temporal association with a stressor and symptom duration of less than 6 months after the resolution of the stressor may help distinguish adjustment disorder from oppositional defiant disorder.

Posttraumatic stress disorder. In children younger than 6 years, posttraumatic stress disorder may manifest initially as dysregulated behaviors, opposition, and tantrums; the association with a traumatic event and with other specific symptoms (traumatic play) are key to establishing the diagnosis. In adolescents, traumatic reenactment and risk-taking may be misinterpreted as defiance and opposition or as conduct problems.

Attention-deficit/hyperactivity disorder. ADHD is often comorbid with oppositional defiant disorder. To make the additional diagnosis of oppositional defiant disorder, it is important to determine that the individual's failure to conform to requests of others is not solely in situations that demand sustained effort and attention or demand that the individual sit still.

Depressive and bipolar disorders. Depressive and bipolar disorders often involve negative affect and irritability. As a result, a diagnosis of oppositional defiant disorder should not be made if the symptoms occur exclusively during the course of a mood disorder.

Disruptive mood dysregulation disorder. Oppositional defiant disorder shares with disruptive mood dysregulation disorder the symptoms of chronic irritable mood and temper outbursts. However, if the irritable mood and other symptoms meet criteria for disruptive mood dysregulation disorder, a

diagnosis of oppositional defiant disorder is not given, even if all criteria for oppositional defiant disorder are met.

Intermittent explosive disorder. Intermittent explosive disorder also involves high rates of anger. However, individuals with this disorder show serious aggression toward others that is not part of the definition of oppositional defiant disorder.

Intellectual developmental disorder (intellectual disability). In individuals with intellectual developmental disorder, a diagnosis of oppositional defiant disorder is given only if the oppositional behavior is markedly greater than is commonly observed among individuals of comparable mental age and with comparable severity of intellectual disability.

Language disorder. Oppositional defiant disorder must also be distinguished from a failure to follow directions that is the result of impaired language comprehension (e.g., hearing loss).

Social anxiety disorder. Oppositional defiant disorder must also be distinguished from defiance because of fear of negative evaluation associated with social anxiety disorder.

Comorbidity

Rates of oppositional defiant disorder are much higher in samples of children, adolescents, and adults with ADHD, and this may be the result of shared temperamental risk factors. Also, oppositional defiant disorder often precedes conduct disorder, although this appears to be most common in children with the childhood-onset subtype. Individuals with oppositional defiant disorder are also at increased risk for anxiety disorders and major depressive disorder, and this seems largely attributable to the presence of the angry-irritable mood symptoms. Extremely high rates of comorbidity between disruptive mood dysregulation disorder and symptoms characteristic of oppositional defiant disorder have been reported, with most individuals with disruptive mood dysregulation disorder having symptoms that meet criteria for oppositional defiant disorder (such as showing argumentative/defiant symptoms); but because oppositional defiant disorder cannot be diagnosed if criteria are also met for disruptive mood dysregulation disorder, only disruptive mood dysregulation disorder would

be diagnosed in such cases. Adolescents and adults with oppositional defiant disorder also show a higher rate of substance use disorders,

although it is unclear if this association is independent of the comorbidity with conduct disorder.

Intermittent Explosive Disorder

Diagnostic Criteria F63.81

A. Recurrent behavioral outbursts representing a failure to control aggressive impulses as manifested by either of the following:
   1. Verbal aggression (e.g., temper tantrums, tirades, verbal arguments or fights) or physical aggression toward property, animals, or other individuals, occurring twice weekly, on average, for a period of 3 months. The physical aggression does not result in damage or destruction of property and does not result in physical injury to animals or other individuals.
   2. Three behavioral outbursts involving damage or destruction of property and/or physical assault involving physical injury against animals or other individuals occurring within a 12-month period.

B. The magnitude of aggressiveness expressed during the recurrent outbursts is grossly out of proportion to the provocation or to any precipitating psychosocial stressors.

C. The recurrent aggressive outbursts are not premeditated (i.e., they are impulsive and/or anger-based) and are not committed to achieve some tangible objective (e.g., money, power, intimidation).

D. The recurrent aggressive outbursts cause either marked distress in the individual or impairment in occupational or interpersonal functioning, or are associated with financial or legal consequences.

E. Chronological age is at least 6 years (or equivalent developmental level).

F. The recurrent aggressive outbursts are not better explained by another mental disorder (e.g., major depressive disorder, bipolar disorder, disruptive mood dysregulation disorder, a psychotic disorder, antisocial personality disorder, borderline personality disorder) and are not attributable to another medical condition (e.g., head trauma, Alzheimer's disease) or to the physiological effects of a substance (e.g., a drug of abuse, a medication). For children ages 6–18 years, aggressive behavior that occurs as part of an adjustment disorder should not be considered for this diagnosis.

Note: This diagnosis can be made in addition to the diagnosis of attention-deficit/hyperactivity disorder, conduct disorder, oppositional defiant disorder, or autism spectrum disorder when recurrent impulsive aggressive outbursts are in excess of those usually seen in these disorders and warrant independent clinical attention.

Diagnostic Features

The impulsive (or anger-based) aggressive outbursts in intermittent explosive disorder have a rapid onset and, typically, little or no prodromal period. Outbursts typically last for less than 30 minutes and commonly occur in response to a minor provocation by a close intimate or associate. Individuals with intermittent explosive disorder often have less severe episodes of verbal or nondamaging, nondestructive, or noninjurious physical assault (Criterion A1) in between more severe destructive/assaultive episodes (Criterion A2). Criterion A1 defines frequent (i.e., twice weekly, on average, for a period of 3 months) aggressive outbursts characterized by temper tantrums, tirades, verbal

arguments or fights, or assault without damage to objects or without injury to animals or other individuals. Criterion A2 defines infrequent (i.e., three in a 1-year period) impulsive aggressive outbursts characterized by damaging or destroying an object, regardless of its tangible value, or by

assaulting/striking or otherwise causing physical injury to an animal or to another individual. Regardless of the nature of the impulsive aggressive outburst, the core feature of intermittent explosive disorder is failure to control impulsive aggressive behavior in response to subjectively experienced provocation (i.e., psychosocial stressor) that would not typically result in an aggressive outburst (Criterion B). The aggressive outbursts are generally impulsive or anger-based rather than premeditated or instrumental (Criterion C) and cause significant distress or impairment in occupational or interpersonal functioning or are associated with financial or legal consequences (Criterion D). A diagnosis of intermittent explosive disorder should not be given to individuals younger than 6 years, or the equivalent developmental level (Criterion E), or to individuals whose aggressive outbursts are better explained by another mental disorder (Criterion F). A diagnosis of intermittent explosive disorder should not be given to individuals with disruptive mood dysregulation disorder or to individuals whose impulsive aggressive outbursts are attributable to another medical condition or to the physiological effects of a substance (Criterion F). In addition, children ages 6–18 years should not receive this diagnosis when impulsive aggressive outbursts occur in the context of an adjustment disorder (Criterion F).

Associated Features

Depressive disorders, anxiety disorders, and substance use disorders are associated with intermittent explosive disorder, although onset of these disorders is typically later than that of intermittent explosive disorder.

Research provides neurobiological support for the presence of serotonergic abnormalities, globally and in the brain, specifically in areas of the limbic system (anterior cingulate) and orbitofrontal cortex in individuals

with intermittent explosive disorder. Amygdala responses to anger stimuli, during functional magnetic resonance imaging scanning, are greater in individuals with intermittent explosive disorder compared with healthy persons. In addition, the volume of gray matter in several frontolimbic regions is reduced and correlates inversely with measures of aggression in individuals with intermittent explosive disorder, although these brain differences are not always seen.

Prevalence

The 1-year prevalence for intermittent explosive disorder in the United States is about 2.6%, with a lifetime prevalence of 4.0%. Higher 1-year prevalences of 3.9% and 6.9% (narrow definition) are present among African Americans and Caribbean Black adolescents, respectively, in the United States, especially among males. This is consistent with higher 12-month rates of psychiatric disorder among immigrant Caribbean Black men and their second- and third-generation offspring, possibly associated with downward social mobility and the effects of racism. However, the reported prevalence of conduct disorder or other disruptive disorders may be affected by misdiagnosis or overdiagnosis of individuals from some cultural backgrounds. Intermittent explosive disorder is more prevalent among younger individuals (e.g., younger than 35–40 years), compared with individuals older than 50 years, and individuals with a high school education or less. In some studies, the prevalence of intermittent explosive disorder is greater in men and boys than in women and girls; other studies have found no sex or gender differences.

Development and Course

The onset of recurrent, problematic, impulsive aggressive behavior is most common in late childhood or adolescence and rarely begins for the first time after age 40 years. The course of the disorder may be episodic, with recurrent periods of impulsive aggressive outbursts. Intermittent explosive disorder appears to follow a chronic and persistent course over many years. It also appears to be quite common regardless of the presence or absence of

attention-deficit/hyperactivity disorder (ADHD) or other disruptive, impulse-control, and conduct disorders (e.g., conduct disorder, oppositional defiant disorder).

Risk and Prognostic Factors

Environmental. Individuals with a history of physical and emotional trauma during the first 20 years of life are at increased risk for intermittent explosive disorder. Long-term displacement from home and separation from family members are risk factors in some refugee population settings.

Genetic and physiological. First-degree relatives of individuals with intermittent explosive disorder are at increased risk for intermittent explosive disorder, and twin studies have demonstrated a substantial genetic influence for impulsive aggression.

Culture-Related Diagnostic Issues

The lower prevalence of intermittent explosive disorder in some regions (Asia, Middle East) or countries (Romania, Nigeria), compared with the United States, suggests that information about recurrent, problematic, impulsive aggressive behaviors either is not elicited on questioning or is less likely to be present, because of cultural factors.

Association With Suicidal Thoughts or Behavior

A study of 1,460 research volunteers found that intermittent explosive disorder comorbid with posttraumatic stress disorder was associated with a markedly elevated rate of lifetime suicide attempt (41%). Posttraumatic stress disorder and intermittent explosive disorder were the only disorders associated with suicide attempt among soldiers with suicidal ideation, although the role of intermittent explosive disorder was less clear in multivariate analyses.

Functional Consequences of Intermittent Explosive Disorder

Social (e.g., loss of friends, relatives, marital instability), occupational (e.g., demotion, loss of employment), financial (e.g., because of value of objects

destroyed), and legal (e.g., civil suits as a result of aggressive behavior against person or property; criminal charges for assault) problems often develop as a result of intermittent explosive disorder.

Differential Diagnosis

A diagnosis of intermittent explosive disorder should not be made when Criteria A1 and/or A2 are only met during an episode of another mental disorder (e.g., major depressive disorder, bipolar disorder, psychotic disorder), or when impulsive aggressive outbursts are attributable to another medical condition or to the physiological effects of a substance or medication. This diagnosis also should not be made, particularly in children and adolescents ages 6–18 years, when the impulsive aggressive outbursts occur in the context of an adjustment disorder.

Disruptive mood dysregulation disorder. In contrast to intermittent explosive disorder, disruptive mood dysregulation disorder is characterized by a persistently negative mood state (i.e., irritability, anger) most of the day, nearly every day, between impulsive aggressive outbursts. A diagnosis of disruptive mood dysregulation disorder can only be given when the onset of recurrent, problematic, impulsive aggressive outbursts is before age 10 years. Finally, a diagnosis of disruptive mood dysregulation disorder should not be made for the first time after age 18 years. Otherwise, these diagnoses are mutually exclusive.

Antisocial personality disorder or borderline personality disorder. Individuals with antisocial personality disorder or borderline personality disorder often display recurrent,

problematic impulsive aggressive outbursts. However, the level of impulsive aggression in individuals with antisocial personality disorder or borderline personality disorder is lower than that in individuals with intermittent explosive disorder.

Delirium, major neurocognitive disorder, and personality change due to another medical condition, aggressive type.

A diagnosis of intermittent explosive disorder should not be made when aggressive outbursts are judged to result from the physiological effects of another diagnosable medical condition (e.g., brain injury associated with a change in personality characterized by aggressive outbursts; complex partial epilepsy). Nonspecific abnormalities on neurological examination (e.g., “soft signs”) and nonspecific electroencephalographic changes are compatible with a diagnosis of intermittent explosive disorder unless there is a diagnosable medical condition that better explains the impulsive aggressive outbursts.

Substance intoxication or substance withdrawal. A diagnosis of intermittent explosive disorder should not be made when impulsive aggressive outbursts are nearly always associated with intoxication with or withdrawal from substances (e.g., alcohol, phencyclidine, cocaine and other stimulants, barbiturates, inhalants). However, when a sufficient number of impulsive aggressive outbursts also occur in the absence of substance intoxication or withdrawal, and these warrant independent clinical attention, a diagnosis of intermittent explosive disorder may be given.

Attention-deficit/hyperactivity disorder, conduct disorder, oppositional defiant disorder, or autism spectrum disorder. Individuals with any of these childhood-onset disorders may exhibit impulsive aggressive outbursts. Individuals with ADHD are typically impulsive and, as a result, may also exhibit impulsive aggressive outbursts. While individuals with conduct disorder can exhibit impulsive aggressive outbursts, the form of aggression characterized by the diagnostic criteria is proactive and predatory. Aggression in oppositional defiant disorder is typically characterized by temper tantrums and verbal arguments with authority figures, whereas impulsive aggressive outbursts in intermittent explosive disorder are in response to a broader array of provocation and include physical assault. The level of impulsive aggression in individuals with a history of one or more of these disorders has been reported as lower than that in comparable individuals whose symptoms also meet intermittent explosive disorder Criteria A through E. Accordingly, if Criteria A through E are also met, and the impulsive aggressive outbursts warrant independent clinical attention, a diagnosis of intermittent explosive disorder may be given.

Comorbidity

Depressive disorders, anxiety disorders, posttraumatic stress disorder, bulimia nervosa, binge-eating disorder, and substance use disorders are most commonly comorbid with intermittent explosive disorder in community samples. In addition, individuals with antisocial personality disorder or borderline personality disorder, and individuals with a history of disorders with disruptive behaviors (e.g., ADHD, conduct disorder, oppositional defiant disorder), are at greater risk for comorbid intermittent explosive disorder.

Conduct Disorder

Diagnostic Criteria

A. A repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated, as manifested by the presence of at least three of the following 15 criteria in the past 12 months from any of the categories below, with at least one criterion present in the past 6 months:

Aggression to People and Animals

1. Often bullies, threatens, or intimidates others.
2. Often initiates physical fights.
3. Has used a weapon that can cause serious physical harm to others (e.g., a bat, brick, broken bottle, knife, gun).
4. Has been physically cruel to people.
5. Has been physically cruel to animals.
6. Has stolen while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery).

7. Has forced someone into sexual activity.

Destruction of Property

8. Has deliberately engaged in fire setting with the intention of causing serious damage.
9. Has deliberately destroyed others' property (other than by fire setting).

Deceitfulness or Theft

10. Has broken into someone else's house, building, or car.
11. Often lies to obtain goods or favors or to avoid obligations (i.e., "cons" others).
12. Has stolen items of nontrivial value without confronting a victim (e.g., shoplifting, but without breaking and entering; forgery).

Serious Violations of Rules

13. Often stays out at night despite parental prohibitions, beginning before age 13 years.
14. Has run away from home overnight at least twice while living in the parental or parental surrogate home, or once without returning for a lengthy period.
15. Is often truant from school, beginning before age 13 years.

B. The disturbance in behavior causes clinically significant impairment in social, academic, or occupational functioning.

C. If the individual is age 18 years or older, criteria are not met for antisocial personality disorder.

Specify whether:

F91.1 Childhood-onset type: Individuals show at least one symptom characteristic of conduct disorder prior to age 10 years.

F91.2 Adolescent-onset type: Individuals show no symptom characteristic of conduct disorder prior to age 10 years.

F91.9 Unspecified onset: Criteria for a diagnosis of conduct disorder are met, but there is not enough information

available to determine whether the onset of the first symptom was before or after age 10 years.

Specify if:

With limited prosocial emotions: To qualify for this specifier, an individual must have displayed at least two of the following characteristics persistently over at least 12 months and in multiple relationships and settings. These characteristics reflect the individual's typical pattern of interpersonal and emotional functioning over this period and not just occasional occurrences in some situations. Thus, to assess the criteria for the specifier, multiple information sources are necessary. In addition to the individual's self-report, it is necessary to consider reports by others who have known the individual for extended periods of time (e.g., parents, teachers, co-workers, extended family members, peers).

Lack of remorse or guilt: Does not feel bad or guilty when he or she does something wrong (exclude remorse when expressed only when caught and/or facing punishment). The individual shows a general lack of concern about the negative consequences of his or her actions. For example, the individual is not remorseful after hurting someone or does not care about the consequences of breaking rules.

Callous—lack of empathy: Disregards and is unconcerned about the feelings of others. The individual is described as cold and uncaring. The individual appears more concerned about the effects of his or her actions on himself or herself, rather than their effects on others, even when they result in substantial harm to others.

Unconcerned about performance: Does not show concern about poor/problematic performance at school, at work, or in other important activities. The individual does

not put forth the effort necessary to perform well, even when expectations are clear, and typically blames others for his or her poor performance.

Shallow or deficient affect: Does not express feelings or show emotions to others, except in ways that seem shallow, insincere, or superficial (e.g., actions contradict the emotion displayed; can turn emotions "on" or "off" quickly) or when emotional expressions are used for gain (e.g., emotions displayed to manipulate or intimidate others).

Specify current severity:

Mild: Few if any conduct problems in excess of those required to make the diagnosis are present, and conduct problems cause relatively minor harm to others (e.g., lying, truancy, staying out after dark without permission, other rule breaking).

Moderate: The number of conduct problems and the effect on others are intermediate between those specified in "mild" and those in "severe" (e.g., stealing without confronting a victim, vandalism).

Severe: Many conduct problems in excess of those required to make the diagnosis are present, or conduct problems cause considerable harm to others (e.g., forced sex, physical cruelty, use of a weapon, stealing while confronting a victim, breaking and entering).

Subtypes

Three subtypes of conduct disorder are provided based on the age at onset of the disorder. Both childhood-onset and adolescent-onset subtypes can occur in a mild, moderate, or severe form. An unspecified-onset subtype is designated when there is insufficient information to determine age at onset.

In childhood-onset conduct disorder, individuals are usually male, have disturbed peer relationships, may have had oppositional defiant disorder

during early childhood, and usually have symptoms that meet full criteria for conduct disorder prior to puberty. Individuals with the childhood-onset type may be more likely to display aggression toward others than individuals with the adolescent-onset type. Many children with this subtype also have concurrent attention-deficit/hyperactivity disorder (ADHD) or other neurodevelopmental difficulties. Individuals with childhood-onset type are more likely to have persistent conduct disorder into adulthood than are those with adolescent-onset type. Individuals with adolescent-onset conduct disorder tend to have more normative peer relationships (although they often display conduct problems in the company of others).

Specifiers

A minority of individuals with conduct disorder exhibit characteristics that qualify for the “with limited prosocial emotions” specifier. The indicators of this specifier are those that have often been labeled as callous and unemotional traits in research. Other personality features, such as thrill seeking, fearlessness, and insensitivity to punishment, may also distinguish those with characteristics described in the specifier. Individuals with

characteristics described in this specifier may be more likely than other individuals with conduct disorder to engage in aggression that is planned for instrumental gain. Individuals with conduct disorder of any subtype or any level of severity can have characteristics that qualify for the specifier “with limited prosocial emotions,” although individuals with the specifier are more likely to have childhood-onset type and a severity specifier rating of severe.

Although the validity of self-report to assess the presence of the specifier has been supported in some research contexts, individuals with conduct disorder with this specifier may not readily admit to the traits in a clinical interview. Thus, to assess the criteria for the specifier, multiple information sources are necessary. Also, because the indicators of the specifier are characteristics that reflect the individual’s typical pattern of interpersonal and emotional functioning, it is important to consider reports

by others who have known the individual for extended periods of time and across relationships and settings (e.g., parents, teachers, co-workers, extended family members, peers).

Diagnostic Features

The essential feature of conduct disorder is a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated (Criterion A). These behaviors fall into four main groupings: aggressive conduct that causes or threatens physical harm to other people or animals (Criteria A1–A7); nonaggressive conduct that causes property loss or damage (Criteria A8–A9); deceitfulness or theft (Criteria A10–A12); and serious violations of rules (Criteria A13–A15). Three or more characteristic behaviors must have been present during the past 12 months, with at least one behavior present in the past 6 months. The disturbance in behavior causes clinically significant impairment in social, academic, or occupational functioning (Criterion B). The behavior pattern is usually present in a variety of settings, such as home, at school, or in the community. Because individuals with conduct disorder are likely to minimize their conduct problems, the clinician often must rely on additional informants. However, informants' knowledge of the individual's conduct problems may be limited if they have inadequately supervised the individual or the individual has concealed symptom behaviors.

Individuals with conduct disorder often initiate aggressive behavior and react aggressively to others. They may display bullying, threatening, or intimidating behavior (including bullying via messaging on web-based social media) (Criterion A1); initiate frequent physical fights (Criterion A2); use a weapon that can cause serious physical harm (e.g., a bat, brick, broken bottle, knife, gun) (Criterion A3); be physically cruel to people (Criterion A4) or animals (Criterion A5); steal while confronting a victim (e.g., mugging, purse snatching, extortion, armed robbery) (Criterion A6); or force someone into sexual activity (Criterion A7). Physical violence may take the form of rape, assault, or, in rare cases, homicide. Deliberate destruction of others' property may include deliberate fire setting with the intention of causing serious damage (Criterion A8) or deliberate destroying

of other people's property in other ways (e.g., smashing car windows, vandalizing school property) (Criterion A9). Acts of deceitfulness or theft may include breaking into someone else's house, building, or car (Criterion A10); frequently lying or breaking promises to obtain goods or favors or to avoid debts or obligations (e.g., "conning" other individuals) (Criterion A11); or stealing items of nontrivial value without confronting the victim (e.g., shoplifting, forgery, fraud) (Criterion A12).

Individuals with conduct disorder may also frequently commit serious violations of rules (e.g., school, parental, workplace). Children with conduct disorder often have a pattern, beginning before age 13 years, of staying out late at night despite parental prohibitions (Criterion A13). Children may also show a pattern of running away from home overnight (Criterion A14). To be considered a symptom of conduct disorder, the running away must have occurred at least twice (or only once if the individual did not return for a lengthy period). Runaway episodes that occur as a direct consequence of physical or sexual abuse do not typically qualify for this criterion. Children with conduct disorder may often be truant from school, beginning prior to age 13 years (Criterion A15).

Associated Features

Especially in ambiguous situations, aggressive individuals with conduct disorder frequently misperceive the intentions of others as more hostile and threatening than is the case and respond with aggression that they then feel is reasonable and justified. Personality features of trait negative emotionality and poor self-control, including poor frustration tolerance, irritability, temper outbursts, suspiciousness, insensitivity to punishment, thrill seeking, and recklessness, frequently co-occur with conduct disorder. Substance misuse is often an associated feature, particularly in adolescent girls.

Prevalence

One-year population prevalence estimates in the United States and other largely high-income countries range from 2% to more than 10%, with a

median of 4%. In the United States, the lifetime prevalence was found to be 12.0% among men and 7.1% among women. The prevalence of conduct disorder in largely Western samples appears to be fairly consistent across various countries. Prevalence rates rise from childhood to adolescence. Prevalence of adolescent-onset conduct disorder is more frequently associated with psychosocial stressors—for example, being a member of a socially oppressed ethnic group facing discrimination. Few children with impairing conduct disorder receive treatment.

Development and Course

The onset of conduct disorder may occur as early as the preschool years, but the first significant symptoms usually emerge during the period from middle childhood through middle adolescence. Oppositional defiant disorder is a common precursor to the childhood-onset type of conduct disorder. Physically aggressive symptoms are more common than nonaggressive symptoms during childhood, but nonaggressive symptoms become more common than aggressive symptoms during adolescence.

Conduct disorder may be diagnosed in adults; however, symptoms of conduct disorder usually emerge in childhood or adolescence, and onset is rare after age 16 years. The course of conduct disorder after onset is variable. In a majority of individuals, the disorder remits by adulthood. Many individuals with conduct disorder—particularly those with adolescent-onset type and those with few and milder symptoms—achieve adequate social and occupational adjustment as adults. However, the childhood-onset type predicts a worse prognosis and an increased risk of criminal behavior, conduct disorder, and substance-related disorders in adulthood. Individuals with conduct disorder are at risk for later mood disorders, anxiety disorders, posttraumatic stress disorder, impulse-control disorders, psychotic disorders, somatic symptom disorders, and substance-related disorders as adults.

Symptoms of the disorder vary with age as the individual develops increased physical strength, cognitive abilities, and sexual maturity. Symptom behaviors that emerge first tend to be less serious (e.g., lying, shoplifting), whereas conduct problems that emerge last tend to be more severe (e.g., rape, theft while confronting a victim). However, there are

wide differences among individuals, with some engaging in the more damaging behaviors at an early age (which is predictive of a worse prognosis). When individuals with conduct disorder reach adulthood, symptoms of aggression, property destruction, deceitfulness, and rule violation, including violence against co-workers, partners, and children, may be exhibited in the workplace and the home, such that antisocial personality disorder may be considered.

Risk and Prognostic Factors

Temperamental. Temperamental risk factors include a difficult undercontrolled infant temperament and lower-than-average intelligence, particularly with regard to verbal IQ.

Environmental. Family-level risk factors include parental rejection and neglect, inconsistent child-rearing practices, harsh discipline, physical or sexual abuse, lack of supervision,

early institutional living, frequent changes of caregivers, large family size, parental criminality, and certain kinds of familial psychopathology (e.g., substance-related disorders). Community-level risk factors include peer rejection, association with a delinquent peer group, neighborhood disadvantage, and exposure to violence. Both types of risk factors tend to be more common and severe among individuals with the childhood-onset subtype of conduct disorder. On the other hand, parental migration is a risk factor for children who are left in the country of origin as well as for those who migrated with their parents, with conduct problems being attributable to acculturation processes. Nevertheless, first-generation immigrants and refugees often have fewer conduct problems than their peers.

Genetic and physiological. Conduct disorder is influenced by both genetic and environmental factors. Genetic associations may be stronger for aggressive symptoms. The risk is increased in children with a biological or adoptive parent or a sibling with conduct disorder. The disorder also appears to be more common in children of biological parents with severe alcohol use disorder, depressive and bipolar disorders, or schizophrenia or

biological parents who have a history of ADHD or conduct disorder. Family history particularly characterizes individuals with the childhood-onset subtype of conduct disorder. Slower resting heart rate has been reliably noted in individuals with conduct disorder compared with those without the disorder, and this marker is not characteristic of any other mental disorder. Reduced autonomic fear conditioning, particularly low skin conductance, is also well documented. However, these psychophysiological findings are not diagnostic of the disorder. Structural and functional differences in brain areas associated with affect regulation and affect processing, particularly frontotemporal-limbic connections involving the brain's ventral prefrontal cortex and amygdala, have been consistently noted in individuals with conduct disorder compared with those without the disorder. However, neuroimaging findings are not diagnostic of the disorder.

Course modifiers. Persistence is more likely for individuals with behaviors that meet criteria for the childhood-onset subtype and qualify for the specifier "with limited prosocial emotions." The risk that conduct disorder will persist is also increased by co-occurring ADHD and by substance abuse.

Culture-Related Diagnostic Issues

Conduct disorder diagnosis may at times be misapplied to individuals in settings where patterns of disruptive behavior are viewed as near-normative (e.g., in very threatening, high-crime areas or war zones). Therefore, the context in which the undesirable behaviors have occurred should be considered. In youth from underserved ethnic and racialized groups, reactions to racism that involve anger and resistance-based coping may be misdiagnosed as conduct disorder by uninformed practitioners, as suggested by the association between experiences of discrimination and adolescent-onset conduct disorder in these groups.

Sex- and Gender-Related Diagnostic Issues

Boys and men with a diagnosis of conduct disorder frequently exhibit fighting, stealing, vandalism, and school discipline problems. Girls and women with a diagnosis of conduct disorder are more likely to exhibit lying, truancy, running away, and prostitution. Whereas boys and men and

girls and women exhibit relational aggression (behavior that harms social relationships of others), girls and women exhibit considerably less physical aggression than do boys and men.

Association With Suicidal Thoughts or Behavior

Suicidal thoughts, suicide attempts, and suicide occur at a higher-than-expected rate in individuals with conduct disorder. A large study conducted in Taiwan that followed adolescents with conduct disorder over 10 years found that conduct disorder was associated

with a higher rate of suicide attempts even after adjustment for comorbid mood, anxiety, and substance use disorders.

Functional Consequences of Conduct Disorder

Conduct disorder behaviors may lead to school suspension or expulsion, problems in work adjustment, legal difficulties, sexually transmitted diseases, unplanned pregnancy, and physical injury from accidents or fights. These problems may preclude attendance in ordinary schools or living in a parental or foster home. Conduct disorder is often associated with an early onset of sexual behavior, alcohol use, tobacco smoking, use of illegal substances, and reckless and risk-taking acts. Accident rates appear to be higher among individuals with conduct disorder compared with those without the disorder. These functional consequences of conduct disorder may increase the risk for health difficulties when individuals reach midlife. It is not uncommon for individuals with conduct disorder to come into contact with the criminal justice system for engaging in illegal behavior. Conduct disorder is a common reason for treatment referral and is frequently diagnosed in mental health facilities for children, especially in forensic practice. It is associated with impairment that is more severe and chronic than that experienced by other clinic-referred children.

Differential Diagnosis

Oppositional defiant disorder.

Conduct disorder and oppositional defiant disorder are both related to symptoms that bring the individual in conflict with adults and other authority figures (e.g., parents, teachers, work supervisors). The behaviors of oppositional defiant disorder are typically of a less severe nature than those of individuals with conduct disorder and do not include aggression toward people or animals, destruction of property, or a pattern of theft or deceit. Furthermore, oppositional defiant disorder includes problems of emotion dysregulation (i.e., angry and irritable mood) that are not included in the definition of conduct disorder. When criteria are met for both oppositional defiant disorder and conduct disorder, both diagnoses can be given.

Attention-deficit/hyperactivity disorder. Although children with ADHD often exhibit hyperactive and impulsive behavior that may be disruptive, this behavior does not by itself violate societal norms or the rights of others and therefore does not usually meet criteria for conduct disorder. When criteria are met for both ADHD and conduct disorder, both diagnoses should be given.

Depressive and bipolar disorders. Irritability, aggression, and conduct problems can occur in children or adolescents with major depressive disorder, bipolar disorder, or disruptive mood dysregulation disorder. The behavioral problems associated with these mood disorders can usually be distinguished from the pattern of conduct problems seen in conduct disorder based on their course. Specifically, individuals with conduct disorder will display substantial levels of aggressive or nonaggressive conduct problems during periods in which there is no mood disturbance, either historically (i.e., a history of conduct problems predating the onset of the mood disturbance) or concurrently (i.e., display of some conduct problems that are premeditated and do not occur during periods of intense emotional arousal). In those cases in which criteria for conduct disorder and a mood disorder are met, both diagnoses can be given.

Intermittent explosive disorder. Both conduct disorder and intermittent explosive disorder involve high rates of aggression. However, the aggression in individuals with intermittent explosive disorder is limited to impulsive aggression and is not premeditated, and it is not committed in order to achieve some tangible objective (e.g., money, power, intimidation).

Also, the definition of intermittent explosive disorder does not include the non-aggressive symptoms of conduct disorder. If criteria for both disorders are met, the diagnosis of intermittent explosive disorder should be given only when the recurrent impulsive aggressive outbursts warrant independent clinical attention.

Adjustment disorders. The diagnosis of an adjustment disorder (with disturbance of conduct or with mixed disturbance of emotions and conduct) should be considered if clinically significant conduct problems that do not meet the criteria for another specific disorder develop in clear association with the onset of a psychosocial stressor and do not resolve within 6 months of the termination of the stressor (or its consequences). Conduct disorder is diagnosed only when the conduct problems represent a repetitive and persistent pattern that is associated with impairment in social, academic, or occupational functioning.

Comorbidity

ADHD and oppositional defiant disorder are both common in individuals with conduct disorder, and this comorbid presentation predicts worse outcomes. Individuals who show the personality features associated with antisocial personality disorder often violate the basic rights of others or violate major age-appropriate societal norms, and as a result their pattern of behavior often meets criteria for conduct disorder. Conduct disorder may also co-occur with one or more of the following mental disorders: specific learning disorder, anxiety disorders, depressive or bipolar disorders, and substance-related disorders. Academic achievement, particularly in reading and other verbal skills, is often below the level expected on the basis of age and intelligence and may justify the additional diagnosis of specific learning disorder or a communication disorder.

Antisocial Personality Disorder

Criteria and text for antisocial personality disorder can be found in the chapter “Personality Disorders.” Because this disorder is closely connected to the spectrum of “externalizing” conduct disorders in this chapter, as well as to the disorders in the adjoining chapter “Substance-Related and Addictive Disorders,” it is listed here as well as in the chapter “Personality Disorders.”

Pyromania

Diagnostic Criteria	**F63.1**

A. Deliberate and purposeful fire setting on more than one occasion.
B. Tension or affective arousal before the act.
C. Fascination with, interest in, curiosity about, or attraction to fire and its situational contexts (e.g., paraphernalia, uses, consequences).
D. Pleasure, gratification, or relief when setting fires or when witnessing or participating in their aftermath.
E. The fire setting is not done for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, to improve one’s living circumstances, in response to a delusion or hallucination, or as a result of impaired judgment (e.g., in major neurocognitive disorder, intellectual developmental disorder [intellectual disability], substance intoxication).
F. The fire setting is not better explained by conduct disorder, a manic episode, or antisocial personality disorder.

Diagnostic Features

The essential feature of pyromania is the presence of multiple episodes of deliberate and purposeful fire setting (Criterion A). Individuals with this

disorder experience tension or affective arousal before setting a fire (Criterion B). There is a fascination with, interest in,

curiosity about, or attraction to fire and its situational contexts (e.g., paraphernalia, uses, consequences) (Criterion C). Individuals with this disorder are often regular "watchers" at fires in their neighborhoods, may set off false alarms, and derive pleasure from institutions, equipment, and personnel associated with fire. They may spend time at the local fire department, set fires to be affiliated with the fire department, or even become firefighters. Individuals with this disorder experience pleasure, gratification, or relief when setting the fire, witnessing its effects, or participating in its aftermath (Criterion D). The fire setting is not done for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, to improve one's living circumstances, or in response to a delusion or a hallucination (Criterion E). The fire setting does not result from impaired judgment (e.g., in major neurocognitive disorder or intellectual developmental disorder [intellectual disability]). The diagnosis is not made if the fire setting is better explained by conduct disorder, a manic episode, or antisocial personality disorder (Criterion F).

Associated Features

Individuals with pyromania may make considerable advance preparation for starting a fire. They may be indifferent to the consequences to life or property caused by the fire, or they may derive satisfaction from the resulting property destruction. The behaviors may lead to property damage, legal consequences, or injury or loss of life to the fire setter or to others. Individuals who impulsively set fires (who may or may not have pyromania) often have a current or past history of alcohol use disorder.

Prevalence

The population prevalence of pyromania is not known. The lifetime prevalence of fire-setting behavior, which is just one component of

pyromania and not sufficient for a diagnosis by itself, was reported as 1.0%–1.1% in a population sample. Fire-setting behavior occurs more often in men than in women (lifetime prevalence 1.7% vs. 0.4%); however, whether this also holds true for pyromania is unknown. The most common comorbidities of fire-setting behavior were antisocial personality disorder, substance use disorder, bipolar disorder, and gambling disorder. In contrast to fire setting, pyromania as a primary diagnosis appears to be very rare. Among a sample of persons in a Finnish hospital reaching the criminal system because of repeated fire setting, only 3.3% had symptoms that met full criteria for pyromania. In a U.S. study, 3.4% of a sample of adults hospitalized for psychiatric reasons had symptoms that met full criteria for current pyromania.

Development and Course

Although data are limited, some research suggests that late adolescence may be the typical age at onset of pyromania. The relationship between fire setting in childhood and pyromania in adulthood has not been documented. In individuals with pyromania, fire-setting incidents are episodic and may wax and wane in frequency. Longitudinal course is unknown. Although fire setting is a major problem in children and adolescents (over 40% of those arrested for arson offenses in the United States are younger than 18 years), pyromania in childhood appears to be rare. Juvenile fire setting is usually associated with conduct disorder, attention-deficit/hyperactivity disorder, or an adjustment disorder.

Sex- and Gender-Related Diagnostic Issues

While fire setting is associated with antisocial behavior in men and women, they differ on some of the antisocial behaviors that accompany fire setting. Whether this holds for pyromania, which is a subset of those with fire setting, is unknown.

Association With Suicidal Thoughts or Behavior

A study of a consecutive sample of male fire setters who had a forensic assessment compared each case with four age-, sex-, and place of birth–

matched controls and found that

fire setting was associated during follow-up with higher rates of suicide and also suicide attempt. Whether these differences apply to pyromania is unknown.

Differential Diagnosis

Other causes of intentional fire setting. It is important to rule out other causes of fire setting before giving the diagnosis of pyromania. Intentional fire setting may occur for profit, sabotage, or revenge; to conceal a crime; to make a political statement (e.g., an act of terrorism or protest); or to attract attention or recognition (e.g., setting a fire in order to discover it and save the day). Fire setting may also occur as part of developmental experimentation in childhood (e.g., playing with matches, lighters, or fire).

Other mental disorders. A separate diagnosis of pyromania is not given when fire setting occurs as part of conduct disorder, a manic episode, or antisocial personality disorder, or if it occurs in response to a delusion or a hallucination (e.g., in schizophrenia) or is attributable to the physiological effects of another medical condition (e.g., epilepsy). The diagnosis of pyromania should also not be given when fire setting results from impaired judgment associated with major neurocognitive disorder, intellectual developmental disorder, or substance intoxication.

Comorbidity

There appears to be a high co-occurrence of substance use disorders, gambling disorder, depressive and bipolar disorders, and other disruptive, impulse-control, and conduct disorders with pyromania.

Kleptomania

Diagnostic Criteria	F63.2

A. Recurrent failure to resist impulses to steal objects that are not needed for personal use or for their monetary value.
B. Increasing sense of tension immediately before committing the theft.
C. Pleasure, gratification, or relief at the time of committing the theft.
D. The stealing is not committed to express anger or vengeance and is not in response to a delusion or a hallucination.
E. The stealing is not better explained by conduct disorder, a manic episode, or antisocial personality disorder.

Diagnostic Features

The essential feature of kleptomania is the recurrent failure to resist impulses to steal items even though the items are not needed for personal use or for their monetary value (Criterion A). The individual experiences a rising subjective sense of tension before the theft (Criterion B) and feels pleasure, gratification, or relief when committing the theft (Criterion C). The stealing is not committed to express anger or vengeance, is not done in response to a delusion or hallucination (Criterion D), and is not better explained by conduct disorder, a manic episode, or antisocial personality disorder (Criterion E). The objects are stolen despite the fact that they are typically of little value to the individual, who could have afforded to pay for them and often gives them away or discards them. Occasionally the individual may hoard the stolen objects or surreptitiously return them. Although individuals with this disorder will generally avoid stealing when immediate arrest is probable (e.g., in full view of a police officer), they usually do not preplan the thefts or fully take

into account the chances of apprehension. The stealing is done without assistance from, or collaboration with, others.

Associated Features

Individuals with kleptomania typically attempt to resist the impulse to steal, and they are aware that the act is wrong and senseless. The individual frequently fears being apprehended and often feels depressed or guilty about the thefts. Neurotransmitter pathways associated with behavioral addictions, including those associated with the serotonin, dopamine, and opioid systems, appear to play a role in kleptomania as well.

Prevalence

In the United States and Canada, kleptomania occurs in about 4%–24% of individuals arrested for shoplifting. Its prevalence in the U.S. general population is very rare, at approximately 0.3%–0.6%. Women outnumber men at a ratio of 3:1.

Development and Course

Age at onset of kleptomania is variable, but the disorder often begins in adolescence. However, the disorder may begin in childhood, adolescence, or adulthood, and in rare cases in late adulthood. There is little systematic information on the course of kleptomania, but three typical courses have been described: sporadic with brief episodes and long periods of remission; episodic with protracted periods of stealing and periods of remission; and chronic with some degree of fluctuation. The disorder may continue for years, despite multiple convictions for shoplifting.

Risk and Prognostic Factors

Genetic and physiological. There appears to be a higher rate of alcohol use disorders in first-degree relatives of individuals with kleptomania than in the general population.

Association With Suicidal Thoughts or Behavior

Kleptomania has been associated with an increased risk for suicide attempts.

Functional Consequences of Kleptomania

The disorder may cause legal, family, career, and personal difficulties.

Differential Diagnosis

Ordinary theft. Kleptomania should be distinguished from ordinary acts of theft or shoplifting. Ordinary theft (whether planned or impulsive) is deliberate and is motivated by the usefulness of the object or its monetary worth. Some persons, especially adolescents, may also steal on a dare, as an act of rebellion, or as a rite of passage. The diagnosis is not made unless other characteristic features of kleptomania are also present. Kleptomania is rare, whereas shoplifting is relatively common.

Malingering. In malingering, individuals may simulate the symptoms of kleptomania to avoid criminal prosecution.

Antisocial personality disorder and conduct disorder. Antisocial personality disorder and conduct disorder are distinguished from kleptomania by a general pattern of antisocial behavior.

Manic episodes, psychotic episodes, and major neurocognitive disorder. Kleptomania should be distinguished from intentional or inadvertent stealing that may occur during a

manic episode, in response to delusions or hallucinations (e.g., in schizophrenia), or as a result of a major neurocognitive disorder.

Comorbidity

Kleptomania may be associated with compulsive buying as well as with depressive and bipolar disorders (especially major depressive disorder), anxiety disorders, eating disorders (particularly bulimia nervosa), personality disorders, substance use disorders (especially alcohol use disorder), and other disruptive, impulse-control, and conduct disorders.

Other Specified Disruptive, Impulse-Control, and Conduct Disorder

F91.8

This category applies to presentations in which symptoms characteristic of a disruptive, impulse-control, and conduct disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the disruptive, impulse-control, and conduct disorders diagnostic class. The other specified disruptive, impulse-control, and conduct disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific disruptive, impulse-control, and conduct disorder. This is done by recording “other specified disruptive, impulse-control, and conduct disorder” followed by the specific reason (e.g., “recurrent behavioral outbursts of insufficient frequency”).

Unspecified Disruptive, Impulse-Control, and Conduct Disorder

F91.9

This category applies to presentations in which symptoms characteristic of a disruptive, impulse-control, and conduct disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the disruptive, impulse-control, and conduct disorders diagnostic class. The unspecified disruptive, impulse-control, and conduct disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific disruptive, impulse-control, and conduct disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Substance-Related and Addictive Disorders

The substance-related disorders encompass 10 separate classes of drugs: alcohol; caffeine; cannabis; hallucinogens (with separate categories for phencyclidine [or similarly acting arylcyclohexylamines] and other hallucinogens); inhalants; opioids; sedatives, hypnotics, or anxiolytics; stimulants (amphetamine-type substances, cocaine, and other stimulants); tobacco; and other (or unknown) substances. These 10 classes are not fully distinct. All drugs that are taken in excess have in common the ability to directly activate the brain reward systems, which are involved in the reinforcement of behaviors and establishment of memories. Instead of achieving reward system activation through adaptive behaviors, these substances produce such an intense activation of the reward system that normal activities may be neglected. The pharmacological mechanisms by which each class of drugs produces reward are different, but the drugs typically activate the system and produce feelings of pleasure, often referred to as a "high." Furthermore, studies suggest that the neurobiological roots of substance use disorders for some individuals can be seen in their behaviors long before the onset of actual substance use (e.g., lower levels of self-control may reflect impairments of brain inhibitory mechanisms); research also suggests the negative impact of substance use itself on brain inhibitory mechanisms.

Note that the phrase "drug addiction" is not applied as a diagnostic term in this classification, although it is in common usage in many countries to describe severe problems related to compulsive and habitual use of substances. The more neutral term *substance use disorder* is used to describe the wide range of the disorder, from a mild form to a severe state of chronically relapsing, compulsive pattern of drug taking. Some clinicians will choose to use the phrase "drug addiction" to describe more severe presentations, but that wording is omitted from the official DSM-5 substance use disorder diagnostic terminology because of its uncertain definition and its potentially negative connotation.

In addition to the substance-related disorders, this chapter also includes gambling disorder, reflecting evidence that gambling behaviors activate reward systems similar to those activated by drugs of abuse and that produce some behavioral symptoms that appear comparable to those produced by the substance use disorders. Other excessive behavioral patterns, such as Internet gaming (see "Conditions for Further Study"), have also been described, but the research on these and other behavioral syndromes is less clear. Thus, groups of repetitive behaviors, sometimes termed *behavioral addictions* (with subcategories such as "sex addiction," "exercise addiction," and "shopping addiction"), are not included because there is insufficient peer-reviewed evidence to establish the diagnostic criteria and course descriptions needed to identify these behaviors as mental disorders.

The substance-related disorders are divided into two groups: substance use disorders and substance-induced disorders. The following conditions may be classified as substance-induced: substance intoxication, substance withdrawal, and substance/medication-induced mental disorders (diagnostic criteria and text are provided in this manual for substance/medication-induced psychotic disorders, bipolar and related disorders, depressive disorders, anxiety disorders, obsessive-compulsive

and related disorders, sleep disorders, sexual dysfunctions, delirium, and neurocognitive disorders in their respective chapters). The term

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substance/medication-induced mental disorder refers to symptomatic presentations that are due to the physiological effects of an exogenous substance on the central nervous system and includes typical intoxicants (e.g., alcohol, inhalants, cocaine), psychotropic medications (e.g., stimulants, sedative-hypnotics), other medications, (e.g., steroids), and environmental toxins (e.g., organophosphate insecticides).

The current section begins with a general discussion of criteria sets for substance use disorder, substance intoxication, substance withdrawal, and substance/medication-induced mental disorders, at least some of which are applicable across classes of substances. Reflecting some unique aspects of the 10 substance classes relevant to this chapter, the remainder of the chapter is organized by substance class. To facilitate differential diagnosis, the diagnostic criteria and text for the substance/medication-induced mental disorders are included with disorders with which they share phenomenology (e.g., substance/medication-induced depressive disorder is in the chapter "Depressive Disorders"). Note that only certain classes of drugs are capable of causing particular types of substance-induced disorders. The substance-related diagnostic categories associated with specific drug classes are shown in Table 1.

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TABLE 1 Diagnoses associated with substance class

	Psychotic disorders	Bipolar and related disorders	Depressive disorders	Anxiety disorders	Obsessive-compulsive and related disorders	Sleep disorders	Sexual dysfunctions	Del
Alcohol	I/W	I/W	I/W	I/W		I/W	I/W	I
Caffeine				I		I/W		
Cannabis	I			I		I/W		
Hallucinogens								
Phencyclidine	I	I	I	I				
Other hallucinogens	I*	I	I	I				
Inhalants	I		I	I				
Opioids			I/W	W		I/W	I/W	I
Sedatives, hypnotics, or anxiolytics	I/W	I/W	I/W	W		I/W	I/W	I
Stimulants**	I	I/W	I/W	I/W	I/W	I/W	I	
Tobacco						W		
Other (or unknown)	I/W	I/W	I/W	I/W	I/W	I/W	I/W	I

Note. X = The category is recognized in DSM-5.
I = The specifier "with onset during intoxication" may be noted for the category.
W = The specifier "with onset during withdrawal" may be noted for the category.
I/W = Either "with onset during intoxication" or "with onset during withdrawal" may be noted for the category.
Major = major neurocognitive disorder; mild = mild neurocognitive disorder.
*Also hallucinogen persisting perception disorder (flashbacks).
**Includes amphetamine-type substances, cocaine, and other or unspecified stimulants.

Substance-Related Disorders

Substance Use Disorders

Diagnostic Features

The essential feature of a substance use disorder is a cluster of cognitive, behavioral, and physiological symptoms indicating that the individual continues using the substance despite significant substance-related problems. As seen in Table 1, the diagnosis of a substance use disorder can be applied to all 10 substance classes included in this chapter except caffeine. For certain classes, some symptoms are less salient, and in a few instances not all symptoms apply (e.g., withdrawal symptoms are not specified for phencyclidine use disorder, other hallucinogen use disorder, or inhalant use disorder). Of note, the consumption of substances, including prescribed medications, may depend in part on cultural background, substance availability, and specific local drug regulations. Thus, there can be significant local or cultural variation in exposure (e.g., countries with cultural prohibitions against alcohol or other substance use may have a lower prevalence of substance-related disorders).

An important characteristic of substance use disorders is an underlying change in brain circuits that may persist beyond detoxification, particularly in individuals with severe disorders. The behavioral effects of these brain changes may be exhibited in the repeated relapses and intense drug craving when the individuals are exposed to drug-related stimuli. These persistent drug effects may benefit from long-term approaches to treatment.

Overall, the diagnosis of a substance use disorder is based on a pathological pattern of behaviors related to use of the substance. To assist with organization, the diagnostic items making up Criterion A can be considered to fit within overall groupings of *impaired control, social impairment, risky use,* and *pharmacological criteria.* Impaired control over substance use is the first criteria grouping (Criteria 1–4). The individual may take the substance in larger amounts or over a longer period than was originally intended (Criterion 1). The individual may express a persistent desire to cut down or regulate substance use and may report

multiple unsuccessful efforts to decrease or discontinue use (Criterion 2). The individual may spend a great deal of time obtaining the substance, using the substance, or recovering from its effects (Criterion 3). In some instances of more severe substance use disorders, virtually all of the individual's daily activities revolve around the substance. Craving (Criterion 4) is manifested by an intense desire or urge for the drug that may occur at any time but is more likely when in an environment where the drug previously was obtained or used. Craving has also been shown to involve classical conditioning and is associated with activation of specific reward structures in the brain. Craving might be queried by asking if there has ever been a time when there were such strong urges to take the drug that the individual could not think of anything else. Current craving is often used as a treatment outcome measure because it may be a signal of impending relapse.

Social impairment is the second grouping of criteria (Criteria 5–7). Recurrent substance use may result in a failure to fulfill major role obligations at work, school, or home (Criterion 5). The individual may continue substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (Criterion 6). Important social, occupational, or recreational

activities may be given up or reduced because of substance use (Criterion 7). The individual may withdraw from family activities and hobbies in order to use the substance.

Risky use of the substance is the third grouping of criteria (Criteria 8–9). This may take the form of recurrent substance use in situations in which it is physically hazardous (Criterion 8). The individual may continue substance use despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance (Criterion 9). The key issue in evaluating this criterion is not the existence of the problem, but rather the individual's failure to abstain from using the substance despite the difficulty it is causing.

Pharmacological criteria are the final grouping (Criteria 10 and 11). Tolerance (Criterion 10) is signaled by requiring a markedly increased dose of the substance to achieve the desired effect or a markedly reduced effect when the usual dose is consumed. The degree to which tolerance develops varies greatly across different individuals as well as across substances and may involve a variety of central nervous system effects. For example, tolerance to respiratory depression and tolerance to sedating and motor coordination may develop at different rates, depending on the substance. Tolerance may be difficult to determine by history alone, and laboratory tests may be helpful (e.g., high blood levels of the substance coupled with little evidence of intoxication suggest that tolerance is likely). Tolerance must also be distinguished from individual variability in the initial sensitivity to the effects of particular substances. For example, some first-time alcohol drinkers show very little evidence of intoxication with three or four drinks, whereas others of similar weight and drinking histories have slurred speech and incoordination.

Withdrawal (Criterion 11) is a syndrome that occurs when blood or tissue concentrations of a substance decline in an individual who had maintained prolonged, heavy use of the substance. After developing withdrawal symptoms, the individual is likely to consume the substance to relieve the symptoms. Withdrawal symptoms vary greatly across the classes of substances, and separate criteria sets for withdrawal are provided for the drug classes. Marked and generally easily measured physiological signs of withdrawal are common with alcohol, opioids, and sedatives, hypnotics, and anxiolytics. Withdrawal signs and symptoms with stimulants (amphetamine-type substances, cocaine, other or unspecified stimulants), as well as tobacco and cannabis, are often present but may be less apparent. Significant withdrawal has *not* been documented in humans after repeated use of phencyclidine, other hallucinogens, and inhalants; therefore, this criterion is not included for these substances. Neither tolerance nor withdrawal is necessary for a diagnosis of a substance use disorder. However, for most classes of substances, a past history of withdrawal is associated with a

more severe clinical course (i.e., an earlier onset of a substance use disorder, higher levels of substance intake, and a greater number of substance-related problems).

Symptoms of tolerance and withdrawal occurring during appropriate use of prescribed medications given as part of medical treatment (e.g., opioid analgesics, sedatives, stimulants) are specifically *not* counted when diagnosing a substance use disorder. The appearance of normal, expected pharmacological tolerance and withdrawal during the course of medical treatment has been known to lead to an erroneous diagnosis of "addiction" even when these were the only symptoms present. Individuals whose *only* symptoms are those that occur as a result of medical treatment (i.e., tolerance and withdrawal as part of medical care when the medications are taken as prescribed) should not receive a diagnosis solely on the basis of these symptoms. However, prescription medications can be used inappropriately, and a substance use

disorder can be correctly diagnosed when there are other symptoms of compulsive, drug-seeking behavior.

Severity and Specifiers

Substance use disorders occur in a broad range of severity, from mild to severe, with severity based on the number of symptom criteria endorsed. As a general estimate of severity, a *mild* substance use disorder is suggested by the presence of two to three symptoms, *moderate* by four to five symptoms, and *severe* by six or more symptoms. Changing severity across time is also reflected by reductions or increases in the frequency and/or dose of substance use, as assessed by the individual's own report, report of knowledgeable others, clinician's observations, and biological testing. The following course specifiers and descriptive features specifiers are also available for substance use disorders: "in early remission," "in sustained remission," "on maintenance therapy," and "in a controlled environment." Definitions of each are provided within respective criteria sets.

Recording Procedures

The clinician should use the code that applies to the substance class but record the name of the *specific substance.* For example, the clinician should record F13.20 moderate alprazolam use disorder (rather than moderate sedative, hypnotic, or anxiolytic use disorder) or F15.10 mild methamphetamine use disorder (rather than mild amphetamine-type substance use disorder). For substances that do not fit into any of the classes (e.g., anabolic steroids), the ICD-10-CM code for other (or unknown) substance use disorder should be used and the specific substance indicated (e.g., F19.10 mild anabolic steroid use disorder). If the substance taken by the individual is unknown, the same ICD-10-CM code (i.e., for "other [or unknown] substance use disorder") should be used (e.g., F19.20 severe unknown substance use disorder). If criteria are met for more than one substance use disorder, each should be diagnosed (e.g., F11.20 severe heroin use disorder; F14.20 moderate cocaine use disorder).

The appropriate ICD-10-CM code for a substance use disorder depends on whether there is a comorbid substance-induced disorder (including substance intoxication and substance withdrawal). In the first example in the paragraph above, the diagnostic code for moderate alprazolam use disorder, F13.20, reflects the absence of a comorbid alprazolam-induced mental disorder. Because ICD-10-CM codes for substance-induced disorders indicate both the presence (or absence) and the severity of the substance use disorder, ICD-10-CM codes for substance use disorders can be used only in the absence of a substance-induced disorder. See the individual substance-specific sections for additional coding information.

Substance-Induced Disorders

The overall category of substance-induced disorders includes substance intoxication, substance withdrawal, and substance/medication-induced mental disorders (e.g., substance-induced psychotic disorder, substance-induced depressive disorder). While substance intoxication and substance withdrawal are recognized as mental disorders, for purposes of clarity of reference in discussions across this chapter, the term *substance/medication-induced mental disorder* (e.g., alcohol-induced depressive disorder, methamphetamine-induced anxiety disorder) is used to distinguish these disorders from substance intoxication and substance withdrawal.

Substance Intoxication and Substance Withdrawal

Criteria for the substance-specific intoxication syndromes are included within the substance-specific sections of this chapter. The essential feature is the development of a reversible substance-specific syndrome due to the recent ingestion of a substance (Criterion A). The clinically significant problematic behavioral or psychological changes associated with intoxication (e.g., belligerence, mood lability, impaired judgment) are attributable to the physiological effects of the substance on the central nervous system (CNS) and develop during or shortly after use of the substance (Criterion B) and are accompanied by substance-specific signs and symptoms (Criterion C). The symptoms are not attributable to another medical condition and are not better explained by another mental disorder (Criterion D). Substance intoxication is common among individuals with a substance use disorder but also occurs frequently in persons who use substances but do not have a substance use disorder. This category does *not* apply to tobacco.

The most common changes in substance intoxication involve disturbances of perception, wakefulness, attention, thinking, judgment, psychomotor behavior, and interpersonal behavior. Short-term, or "acute," substance intoxications may have different signs and symptoms from sustained, or "chronic," substance intoxications. For example, moderate cocaine doses may initially produce gregariousness, but social withdrawal may develop if such doses are frequently repeated over days or weeks.

When used in the physiological sense, the term *intoxication* is broader than the diagnosis of substance intoxication as defined in this manual. Many substances may produce physiological or psychological changes that are not necessarily problematic. For example, an individual with tachycardia from substance use is experiencing a physiological effect from the substance, but if this is the only symptom in the absence of problematic behavior, the diagnosis of substance intoxication would not apply. Intoxication may sometimes persist beyond the time when the substance is detectable in the body. This may be attributable to enduring CNS effects, from which the recovery takes longer than the time for elimination of the substance. These longer-term effects of intoxication must be distinguished from *withdrawal* (i.e., symptoms initiated by a decline in blood or tissue concentrations of a substance).

Criteria for substance withdrawal are also included within the substance-specific sections of this chapter. The essential feature is the development of a substance-specific problematic behavioral change, with physiological and cognitive concomitants, that is due to the cessation of, or reduction in, heavy and prolonged substance use (Criterion A). The substance-specific syndrome (Criterion B) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C). The symptoms are not due to another medical condition and are not better explained by another mental disorder (Criterion D). Withdrawal is usually, but not always, associated with a substance use disorder. Also, it is important to emphasize that symptoms of withdrawal occurring during appropriate use of medications given as part of medical treatment with

prescribed medications (e.g., opioid analgesics, sedatives, stimulants) are specifically *not* counted when diagnosing a substance use disorder. Most individuals with withdrawal have an urge to readminister the substance to reduce the symptoms.

Route of Administration and Speed of Substance Effects

Routes of administration that produce more rapid and efficient absorption into the bloodstream (e.g., intravenous, smoking, intranasal "snorting") tend to result in a more intense intoxication and an increased likelihood of an escalating pattern of substance

use leading to withdrawal. Similarly, rapidly acting substances are more likely than slower-acting substances to produce immediate intoxication.

Duration of Effects

Within the same drug category, relatively short-acting substances tend to have a higher potential for the development of withdrawal than do those with a longer duration of action. However, longer-acting substances tend to have longer duration of withdrawal symptoms. The half-life of the substance parallels aspects of withdrawal: the longer the duration of action, the longer the time between cessation and the onset of withdrawal symptoms and the longer the withdrawal duration. In general, the longer the acute withdrawal period, the less intense the syndrome tends to be.

Use of Multiple Substances

Substance intoxication and withdrawal often involve several substances used simultaneously or sequentially. In these cases, each diagnosis should be recorded separately.

Associated Laboratory Findings

Laboratory analyses of blood and urine samples can help determine recent use and the specific substances involved. However, a positive laboratory test result does not by itself indicate that the individual has a pattern of substance use that meets criteria for a substance-induced or substance use disorder, and a negative test result does not by itself rule out a diagnosis.

Laboratory tests can be useful in identifying withdrawal. If the individual presents with withdrawal from an unknown substance, laboratory tests may help identify the substance and may also be helpful in differentiating withdrawal from other mental disorders. In addition, normal functioning in the presence of high blood levels of a substance suggests considerable tolerance.

Development and Course

Individuals ages 18–24 years have relatively high prevalence rates for the use of virtually every substance. Intoxication is usually the initial substance-related disorder and often begins in the teens. Withdrawal can occur at any age as long as the relevant drug has been taken in sufficient doses over an extended period of time.

Recording Procedures for Substance Intoxication and Substance Withdrawal

The clinician should use the code that applies to the class of substances but record the name of the *specific substance.* For example, the clinician should record F13.230 secobarbital withdrawal (rather than sedative, hypnotic, or anxiolytic withdrawal) or F15.120 methamphetamine intoxication (rather than amphetamine-type substance intoxication). Note that

the appropriate ICD-10-CM diagnostic codes for substance intoxication and substance withdrawal depend on whether there is a comorbid substance use disorder. In this case, the F15.120 code for methamphetamine intoxication indicates the presence of a comorbid mild methamphetamine use disorder. If there had been no comorbid methamphetamine use disorder (and no perceptual disturbances), the diagnostic code would have been F15.920. See the coding note for the substance-specific intoxication and withdrawal syndromes for the actual coding options.

For substances that do not fit into any of the classes (e.g., anabolic steroids), the ICD-10-CM code for other (or unknown) substance intoxication or other (or unknown) substance withdrawal should be used and the specific substance indicated (e.g., F19.920 anabolic steroid intoxication). If the substance taken by the individual is unknown, the same code (i.e., for the class "other [or unknown] substance") should be used (e.g., F19.920 unknown substance intoxication). If there are symptoms or problems associated with a particular substance but criteria are not met for any of the substance-specific disorders, the unspecified category can be used (e.g., F12.99 unspecified cannabis-related disorder).

As noted above, the substance-related codes in ICD-10-CM combine the substance use disorder aspect of the clinical picture and the substance-induced aspect into a single combined code. Thus, if both heroin withdrawal and moderate heroin use disorder are present, the single code F11.23 for heroin withdrawal is given to cover both presentations. See the individual substance-specific sections for additional coding information.

Substance/Medication-Induced Mental Disorders

The substance/medication-induced mental disorders are potentially severe, usually temporary, but sometimes persisting CNS syndromes that develop in the context of the effects of substances of abuse, medications, and some toxins. They are distinguished from the substance use disorders, in which a cluster of cognitive, behavioral, and physiological symptoms contribute to the continued use of a substance despite significant substance-related problems. The substance/medication-induced mental disorders may be induced by any of the 10 classes of substances that produce substance use disorders, or by a great variety of other medications used in medical treatment. Each substance/medication-induced mental disorder is described in the relevant chapter (e.g., substance/medication-induced depressive disorder is located in "Depressive Disorders"), and therefore, only a brief description is offered here. All substance/medication-induced disorders share common characteristics. It is important to recognize these common features to aid in the detection of these disorders. These features are described as follows:

A. A clinically significant presentation of symptoms characteristic of disorders in the relevant diagnostic class predominates in the clinical picture.

B. There is evidence from the history, physical examination, or laboratory findings of both of the following:

   1. The symptoms in Criterion A developed during or soon after substance intoxication, substance withdrawal, or exposure to or withdrawal from a medication; and
   2. The involved substance/medication is capable of producing the symptoms in Criterion A.

C. The disturbance is not better explained by an independent mental disorder (i.e., one that is not substance- or medication-induced). Such evidence of an independent mental disorder could include the following:

   1. The disturbance preceded the onset of severe intoxication or withdrawal or exposure to the medication; or
   2. The disturbance persisted for a substantial period of time (e.g., at least 1 month) after the cessation of acute withdrawal or severe intoxication or taking the

medication. This criterion does not apply to substance-induced neurocognitive disorders or hallucinogen persisting perception disorder, which persist beyond the cessation of acute intoxication or withdrawal.

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Diagnostic and Associated Features

Some generalizations can be made regarding the categories of substances capable of producing clinically relevant substance-induced mental disorders. In general, the more sedating drugs (sedative, hypnotics, or anxiolytics, and alcohol) can produce prominent and clinically significant depressive disorders during intoxication, while anxiety conditions are likely to be observed during withdrawal syndromes from these substances. Also, during intoxication, the more stimulating substances (e.g., amphetamines and cocaine) are likely to be associated with substance-induced psychotic disorders and substance-induced anxiety disorders, and with substance-induced major depressive episodes observed during withdrawal. Both the more sedating and the more stimulating drugs are likely to produce significant but temporary sleep and sexual disturbances. An overview of the relationship between specific categories of substances and specific psychiatric syndromes is presented in Table 1.

The medication-induced conditions include what are often idiosyncratic CNS reactions or relatively extreme examples of side effects for a wide range of medications taken for a variety of medical concerns. These include neurocognitive complications of anesthetics, antihistamines, antihypertensives, and a variety of other medications and toxins (e.g., organophosphates, insecticides, carbon monoxide), as described in the chapter on neurocognitive disorders. Psychotic syndromes may be temporarily experienced in the context of anticholinergic, cardiovascular, and steroid drugs, as well as during use of stimulant-like and depressant-like prescription or over-the-counter drugs. Temporary but severe mood disturbances can be observed with a wide range of medications, including steroids, antihypertensives, disulfiram, and any prescription or over-the-counter depressant or stimulant-like substances. A similar range of medications can be associated with temporary anxiety syndromes, sexual dysfunctions, and conditions of disturbed sleep.

In general, to be considered a substance/medication-induced mental disorder, there must be evidence that the symptoms being observed are not likely to be better explained by an independent mental disorder. The latter is more likely to be the case if the symptoms were present before the severe intoxication or withdrawal or medication administration, or, with the exception of several substance-induced persisting disorders listed in 1, continued more than 1 month after cessation of acute withdrawal, severe intoxication, or use of the medications. When symptoms are only observed during a substance-induced delirium (e.g., alcohol withdrawal delirium), only the delirium should be diagnosed, and other psychiatric symptoms occurring during the delirium should not also be diagnosed separately, as many of these symptoms (e.g., disturbances in mood, anxiety, reality testing) are commonly seen during agitated, confused states. The features associated with each relevant major mental disorder are similar whether observed with independent or substance/medication-induced mental disorders. However, individuals with substance/medication-induced mental disorders are likely to also demonstrate the associated features seen with the specific category of substance or medication, as listed in other subsections of this chapter.

Development and Course

Substance-induced mental disorders develop in the context of intoxication with or withdrawal from substances of abuse, whereas medication-induced mental disorders can be seen with prescribed or over-the-counter medications that are taken at the suggested doses.

Both conditions are usually temporary and likely to disappear within 1 month or so of cessation of acute withdrawal, severe intoxication, or use of the medication. Exceptions to these generalizations occur for certain long-duration substance-induced disorders: substance-associated neurocognitive disorders that relate to conditions such as alcohol-induced neurocognitive disorder, inhalant-induced neurocognitive disorder, and sedative-, hypnotic-, or anxiolytic-induced neurocognitive disorder; and hallucinogen persisting perception disorder ("flashbacks"; see the section "Hallucinogen-Related Disorders" later in this chapter). However, most substance/medication-induced mental disorders, regardless of the severity of the symptoms, are likely to improve relatively quickly with abstinence and unlikely to remain clinically relevant for more than 1 month after complete cessation of use.

As is true of many consequences of heavy substance use, some individuals are more and others less prone toward developing specific substance-induced disorders. Similar types of predispositions may make some individuals more likely to develop psychiatric side effects of some types of medications, but not others. However, it is unclear whether individuals with family histories or personal prior histories of independent psychiatric syndromes are more likely to develop the induced syndrome once the consideration is made as to whether the quantity and frequency of the substance were sufficient to lead to the development of a substance-induced syndrome.

There are indications that the intake of substances of abuse or some medications with psychiatric side effects in the context of a preexisting mental disorder is likely to result in an intensification of the symptoms of the preexisting mental disorder. The risk for substance/medication-induced mental disorders is likely to increase with both the quantity and the frequency of consumption of the relevant substance.

The symptom profiles for the substance/medication-induced mental disorders resemble independent mental disorders. While the symptoms of substance/medication-induced mental disorders can be identical to those of independent mental disorders (e.g., delusions, hallucinations, psychoses, major depressive episodes, anxiety syndromes), and although they can have the same severe consequences (e.g., suicide), most induced mental disorders are likely to improve in a matter of days to weeks of abstinence.

The substance/medication-induced mental disorders are an important part of the differential diagnoses for the independent psychiatric conditions. The importance of recognizing an induced mental disorder is similar to the relevance of identifying the possible role of some medical conditions and medication reactions before diagnosing an independent mental disorder. Symptoms of substance- and medication-induced mental disorders may be identical cross-sectionally to those of independent mental disorders but have different treatments and prognoses from the independent condition.

Functional Consequences of Substance/Medication-Induced Mental Disorders

The same consequences related to the relevant independent mental disorder (e.g., suicide attempts) are likely to apply to the substance/medication-induced mental disorders, but these are likely to disappear within 1 month after abstinence. Similarly, the same functional consequences associated with the relevant substance use disorder are likely to be seen for the substance-induced mental disorders.

Recording Procedures for Substance/Medication-Induced Mental Disorders

Diagnostic criteria, coding notes, and recording procedures for the specific substance/medication-induced mental disorders are provided in chapters of the manual corresponding with disorders of shared phenomenology (see the substance/medication-induced

mental disorders in these chapters: “Schizophrenia Spectrum and Other Psychotic Disorders,” “Bipolar and Related Disorders,” “Depressive Disorders,” “Anxiety Disorders,” “Obsessive-Compulsive and Related Disorders,” “Sleep-Wake Disorders,” “Sexual Dysfunctions,” and “Neurocognitive Disorders”). When recording a substance/medication-induced mental disorder that is comorbid with a substance use disorder, only a single diagnosis is given that reflects both the type of substance and the type of mental disorder induced by the substance, as well as the severity of the comorbid substance use disorder (e.g., cocaine-induced psychotic disorder with severe cocaine use disorder). For a substance-induced mental disorder occurring in the absence of comorbid substance use disorder (e.g., when the disorder is induced by one-time use of a substance or medication), only the substance/medication-induced mental disorder is recorded (e.g., corticosteroid-induced depressive disorder). Additional information needed to record the diagnostic name of the substance/medication-induced mental disorder is provided in the section “Recording Procedures” for each substance/medication-induced mental disorder in its respective chapter.

Alcohol-Related Disorders

Alcohol Use Disorder

Alcohol Intoxication

Alcohol Withdrawal

Alcohol-Induced Mental Disorders

Unspecified Alcohol-Related Disorder

Alcohol Use Disorder

Diagnostic Criteria

A. A problematic pattern of alcohol use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. Alcohol is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control alcohol use.
3. A great deal of time is spent in activities necessary to obtain alcohol, use alcohol, or recover from its effects.
4. Craving, or a strong desire or urge to use alcohol.

5. Recurrent alcohol use resulting in a failure to fulfill major role obligations at work, school, or home.
6. Continued alcohol use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of alcohol.
7. Important social, occupational, or recreational activities are given up or reduced because of alcohol use.
8. Recurrent alcohol use in situations in which it is physically hazardous.

9. Alcohol use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by alcohol.
10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of alcohol to achieve intoxication or desired effect.
    b. A markedly diminished effect with continued use of the same amount of alcohol.
11. Withdrawal, as manifested by either of the following:
    a. The characteristic withdrawal syndrome for alcohol (refer to Criteria A and B of the criteria set for alcohol withdrawal).
    b. Alcohol (or a closely related substance, such as a benzodiazepine) is taken to relieve or avoid withdrawal symptoms.

Specify if:

In early remission: After full criteria for alcohol use disorder were previously met, none of the criteria for alcohol use disorder have been met for at least 3 months but for less than 12 months (with the exception that Criterion A4, “Craving, or a strong desire or urge to use alcohol,” may be met).

In sustained remission: After full criteria for alcohol use disorder were previously met, none of the criteria for alcohol use disorder have been met at any time during a period of 12 months or longer (with the exception that Criterion A4, “Craving, or a strong desire or urge to use alcohol,” may be met).

Specify if:

In a controlled environment: This additional specifier is used if the individual is in an environment where access to alcohol is restricted.

Code based on current severity/remission: If an alcohol intoxication, alcohol withdrawal, or another alcohol-induced mental disorder is also present, do not use the codes below for alcohol use disorder. Instead, the comorbid alcohol use disorder is indicated in the 4th character of the alcohol-induced disorder code (see the coding note for alcohol intoxication, alcohol withdrawal, or a specific alcohol-induced mental disorder). For example, if there is comorbid alcohol intoxication and alcohol use disorder, only the alcohol intoxication code is given, with the 4th character indicating whether the comorbid alcohol use disorder is mild, moderate, or severe: F10.129 for mild alcohol use disorder with alcohol intoxication or F10.229 for a moderate or severe alcohol use disorder with alcohol intoxication.

Specify current severity/remission:

F10.10 Mild: Presence of 2–3 symptoms.

F10.11 Mild, In early remission

F10.11 Mild, In sustained remission

F10.20 Moderate: Presence of 4–5 symptoms.

F10.21 Moderate, In early remission

F10.21 Moderate, In sustained remission

F10.20 Severe: Presence of 6 or more symptoms.

F10.21 Severe, In early remission

F10.21 Severe, In sustained remission

Specifiers

"In a controlled environment" applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

Severity of the disorder is based on the number of diagnostic criteria endorsed. For a given individual, changes in severity of alcohol use disorder across time are also reflected by reductions in the frequency (e.g., days of use per month) or dose (e.g., number of standard drinks consumed per day) of alcohol used, as assessed by the individual's self-report, report of knowledgeable others, clinician observations, and, when practical, biological testing (e.g., elevations in blood tests as described in the section "Diagnostic Markers" for this disorder).

Diagnostic Features

Alcohol use disorder is defined by a cluster of behavioral and physical symptoms, such as withdrawal, tolerance, and craving. Alcohol withdrawal is characterized by withdrawal symptoms that develop approximately 4–12 hours after the reduction of intake following prolonged, heavy alcohol ingestion. Because withdrawal from alcohol can be unpleasant and intense, individuals may continue to consume alcohol despite adverse consequences, often to avoid or to relieve withdrawal symptoms. Some withdrawal symptoms (e.g., sleep problems) can persist at lower intensities for months and can contribute to relapse. Once a pattern of repetitive and intense use develops, individuals with alcohol use disorder may devote substantial periods of their time to obtaining and consuming alcoholic beverages.

Craving for alcohol is indicated by a strong desire to drink that makes it difficult to think of anything else and that often results in the onset of drinking. School and job performance may also suffer either from the aftereffects of drinking or from actual intoxication at school or on the job; child care or household responsibilities may be neglected; and alcohol-related absences may occur from school or work. The individual may use alcohol in physically hazardous circumstances (e.g., driving an automobile, swimming, operating machinery while intoxicated). Finally, individuals with an alcohol use disorder may continue to consume alcohol despite the knowledge that continued consumption poses significant physical (e.g., blackouts, liver disease), psychological (e.g., depression), social, or interpersonal problems (e.g., violent arguments with spouse while intoxicated, child abuse).

Associated Features

Alcohol use disorder is often associated with problems similar to those associated with other substances (e.g., cannabis; cocaine; heroin; amphetamines; sedatives, hypnotics, or anxiolytics). Alcohol may be used to alleviate the unwanted effects of these other substances or to substitute for them when they are not available. Symptoms of conduct problems, depression, anxiety, and insomnia frequently accompany heavy drinking and sometimes precede it.

Repeated intake of high doses of alcohol can affect nearly every organ system, especially the gastrointestinal tract, cardiovascular system, and the central and peripheral nervous systems. Gastrointestinal effects include gastritis, stomach or duodenal ulcers, and, in about 15% of individuals who use alcohol heavily, liver cirrhosis and/or pancreatitis. There is also an increased rate of cancer of the esophagus, stomach, and other parts of the gastrointestinal tract. One of the most commonly associated conditions is low-grade hypertension. Cardiomyopathy and other myopathies are less common but occur at an increased rate among those who drink very heavily. These factors, along with marked

increases in levels of triglycerides and low-density lipoprotein cholesterol, contribute to an elevated risk of heart disease. Peripheral neuropathy may be evidenced by muscular weakness, paresthesias, and decreased peripheral sensation. More persistent central nervous system effects include cognitive deficits, such as severe memory impairment and degenerative changes in the cerebellum. These effects are related to the direct effects of alcohol, trauma, or vitamin deficiencies (particularly of the B vitamins, including thiamine). One devastating central nervous system effect is the relatively rare alcohol-induced persisting amnestic disorder, or Wernicke-Korsakoff syndrome, in which the ability to encode new memory is severely impaired. This condition would now be described within the chapter “Neurocognitive Disorders” and would be termed a *substance/medication-induced neurocognitive disorder.*

Alcohol use disorder is an important contributor to suicide risk during severe intoxication and in the context of a temporary alcohol-induced depressive or bipolar disorder. There is an increased rate of suicidal behavior as well as of suicide among individuals with the disorder.

Prevalence

Alcohol use disorder is common. In the United States, lifetime prevalence rates of DSM-5 alcohol use disorder among adults were estimated to be 29.1% overall with severity specified as follows: 8.6% mild, 6.6% moderate, and 13.9% severe. Among Australian adults, the estimated lifetime prevalence of DSM-5 alcohol use disorder was 31.0%.

Rates of disorder vary by gender and age. In the United States, rates were greater among men (36.0% lifetime prevalence) than among women (22.7%). Twelve-month prevalence of DSM-IV alcohol use disorders in the United States was 4.6% among individuals ages 12–17 years, 16.2% among individuals ages 18–29 years, and 1.5% among individuals 65 years and older.

Twelve-month prevalence of alcohol use disorders varies across U.S. ethnoracial groups as well. For individuals ages 12–17 years, prevalence of DSM-IV alcohol use disorders was greatest among Native Americans (2.8%) and non-Latinx Whites (2.2%), relative to Asian Americans (1.6%), individuals reporting two or more racialized backgrounds (1.6%), Hispanics (1.5%), and African Americans (0.8%). Among adults, data from a large U.S. population-based study indicated that the 12-month prevalence of DSM-5 alcohol use disorder was 14.4% in African Americans,

14.0% in non-Hispanic Whites, 13.6% in Hispanics, and 10.6% in Asian Americans and Pacific Islanders. Data from a large community-based survey of Native Americans from Southwestern and Northern Plains tribal nations showed that the 12-month prevalence of DSM-IV alcohol abuse and dependence ranged from 4.1% to 9.8%. There is extensive diversity in the rates and patterns of alcohol abuse and dependence across the more than 570 American Indian and Alaska Native communities in the United States, as well as high rates of abstinence from alcohol use in some of these communities. Historical experiences of dispossession and subjugation and ongoing discrimination have been associated with increased risk of symptom onset. Given the diversity of tribal communities, prevalence estimates for alcohol use disorder among Native Americans should be interpreted with caution.

Development and Course

The first episode of alcohol intoxication is likely to occur during the mid-teens. Alcohol-related problems that do not meet full criteria for a use disorder or isolated problems may occur before age 20 years, but the age at onset of an alcohol use disorder with two or more of the criteria clustered together peaks in the late teens or early to mid 20s. The large majority of individuals who develop alcohol-related disorders do so by their late 30s. The first evidence of withdrawal is not likely to appear until after many other aspects of an alcohol use disorder have developed. An earlier onset of alcohol use disorder is observed in adolescents with preexisting conduct problems and those with an earlier onset of intoxication.

Alcohol use disorder has a variable course that is characterized by periods of remission and relapse. A decision to stop drinking, often in response to a crisis, is likely to be followed by a period of weeks or more of abstinence, which is often followed by limited periods of controlled or nonproblematic drinking. However, once alcohol intake resumes, it is highly likely that consumption will rapidly escalate and that severe problems will once again develop.

Alcohol use disorder is often erroneously perceived as an intractable condition, perhaps based on the fact that individuals who present for treatment typically have a history of many years of severe alcohol-related problems. However, these most severe cases represent only a minority of individuals with this disorder, and the typical individual with the disorder has a much more promising prognosis.

Among adolescents, conduct disorder and repeated antisocial behavior often co-occur with alcohol- and with other substance-related disorders. While most individuals with alcohol use disorder develop the condition before age 40 years, perhaps 10% have later onset, as suggested by a prospective study in California. Age-related physical changes in older individuals result in increased brain susceptibility to the depressant effects of alcohol; decreased rates of liver metabolism of a variety of substances, including alcohol; and decreased percentages of body water. These changes can cause older people to develop more severe intoxication and subsequent problems at lower levels of consumption. Alcohol-related problems in older people are also especially likely to be associated with other medical complications.

Risk and Prognostic Factors

Environmental. Environmental risk and prognostic factors may include poverty and discrimination (including structural inequities such as differential incarceration rates and differential access to medications for addiction treatment), unemployment and low levels of education, cultural attitudes toward drinking and intoxication, the availability of alcohol (including price), acquired personal experiences with alcohol, and stress

levels. Additional potential mediators of how alcohol problems develop in predisposed individuals include heavier peer substance use, exaggerated positive expectations of the effects of alcohol, and suboptimal ways of coping with stress.

Genetic and physiological. Alcohol use disorder runs in families, with 40%–60% of the variance of risk explained by genetic influences. The rate of this condition is three to four times higher in close relatives of individuals with alcohol use disorder, with values highest for individuals with a greater number of affected relatives, closer genetic relationships to the affected individual, and higher severity of the alcohol-related problems in those relatives. A significantly higher rate of alcohol use disorder exists in the monozygotic twin than in the dizygotic twin of an individual with the condition. A three- to fourfold increase in risk has been observed in children of individuals with alcohol use disorder, even when these children were given up for adoption at birth and raised by adoptive parents who did not have the disorder.

Advances in understanding the genes that operate through intermediate characteristics (or phenotypes) to affect the risk of alcohol use disorder can help to identify individuals who might be at particularly low or high risk for alcohol use disorder. Among the low-risk phenotypes is the acute alcohol-related skin flush (seen more commonly in persons of Asian descent). High vulnerability is associated with preexisting schizophrenia or bipolar disorder, as well as impulsivity (producing enhanced rates of all substance use disorders and gambling disorder), and a high risk specifically for alcohol use disorder is associated with a low level of response (low sensitivity) to alcohol. A number of gene variations may account for low response to alcohol or modulate the dopamine reward systems; however, any single gene variant is likely to explain only 1%–2% of the risk for these disorders. Gene-environment interactions modulate the impact of genetic variations; for

example, genetic effects on alcohol use are more pronounced when social constraints are minimized (e.g., low parental monitoring) or when the environment permits easy access to alcohol or encourages its use (e.g., high peer deviance).

Course modifiers. In general, high levels of impulsivity are associated with an earlier onset and more severe alcohol use disorder.

Culture-Related Diagnostic Issues

In most cultures, alcohol is the most frequently used intoxicating substance and contributes to considerable morbidity and mortality. Globally, 2.8 million deaths were attributed to alcohol use, which corresponds to 2.2% of total age-standardized deaths among women and 6.8% among men. Globally, an estimated 237 million men and 46 million women have alcohol use disorder, with the highest prevalence being among men and women in the European Region (14.8% and 3.5%) and the Region of the Americas (11.5% and 5.1%); in general, high-income countries have the highest prevalence. Greater acculturation to U.S. society among immigrants is associated with rising prevalence of alcohol use disorder, especially among women. Ethnic density (greater proportion of people from the same background) may decrease the risk of alcohol use disorder because of greater social support and buffering against the effects of discrimination. However, neighborhood segregation may increase the risk for disorders because of the association with other risk factors, such as higher concentration of alcohol advertising and retail outlets in low-income areas.

Genetic polymorphisms for the alcohol-metabolizing enzymes alcohol dehydrogenase and aldehyde dehydrogenase may affect the response to alcohol. When consuming alcohol, individuals with certain polymorphisms can experience a flushed face and palpitations, reactions that can be so severe as to limit or preclude future

alcohol consumption and diminish the risk for alcohol use disorder. For example, these gene variations are seen in as many as 40% of Japanese, Chinese, and Korean individuals and are related to lower risks for the disorder. However, this protective effect may be modulated by sociocultural factors, as shown by rising prevalence of alcohol use disorder in Japan, China, and South Korea over the last decades associated with increasing westernization and changing cultural attitudes about women's drinking.

Despite small variations regarding individual criterion items, the diagnostic criteria perform equally well across most race/ethnicity groups.

Sex- and Gender-Related Diagnostic Issues

Men have higher rates of drinking and alcohol use disorder than women, although the gender gap is narrowing as women are initiating alcohol use at a younger age. Because females generally weigh less than males, have more fat and less water in their bodies, and metabolize less alcohol in their esophagus and stomach, they are likely to develop higher blood alcohol levels per drink than males. Females who drink heavily may also be more vulnerable than males to some of the physical consequences associated with alcohol, including alcohol-related blackouts and liver disease. Additionally, while genetic-related mechanisms for alcohol risk overlap for males and females, the specific environmental components that add to the risk may differ across sexes, especially during adolescence. Drinking during pregnancy, which tends to decrease overall, may be a sign of an alcohol use disorder.

Diagnostic Markers

Individuals whose heavier drinking places them at elevated risk for alcohol use disorder can be identified both through standardized questionnaires and by elevations in blood test results likely to be seen with regular heavier drinking. These measures do not establish a diagnosis of an alcohol-related disorder but can be useful in highlighting individuals for

whom more information should be gathered. The most direct test available to measure alcohol consumption cross-sectionally is *blood alcohol concentration*, which can also be used to judge tolerance to alcohol. For example, an individual with a concentration of 150 mg of ethanol per deciliter (dL) of blood who does not show signs of intoxication can be presumed to have acquired at least some degree of tolerance to alcohol. At 200 mg/dL, most nontolerant individuals demonstrate severe intoxication.

Regarding laboratory tests, one sensitive laboratory indicator of heavy drinking is a modest elevation or high-normal levels (>35 units) of gamma-glutamyltransferase (GGT). This may be the only laboratory finding. At least 70% of individuals with a high GGT level are persistent heavy drinkers (i.e., consuming eight or more drinks daily on a regular basis). A second test with comparable or even higher levels of sensitivity and specificity is carbohydrate-deficient transferrin (CDT), with levels of 20 units or higher useful in identifying individuals who regularly consume eight or more drinks daily. Given that both GGT and CDT levels return toward normal within days to weeks of stopping drinking, both state markers may be useful in monitoring abstinence, especially when the clinician observes increases, rather than decreases, in these values over time—a finding indicating that the individual is likely to have returned to heavy drinking. The combination of tests for CDT and GGT may have even higher levels of sensitivity and specificity than either test used alone. Additional useful tests include the mean corpuscular volume (MCV), which may be elevated to high-normal values in individuals who drink heavily—a change that is due to the direct toxic effects of alcohol on erythropoiesis. Although the MCV can be used to help

identify those who drink heavily, it is a poor method of monitoring abstinence because of the long half-life of red blood cells. Liver function tests (e.g., alanine aminotransferase and alkaline phosphatase) can reveal liver injury that is a consequence of heavy drinking. Other potential markers of heavy drinking that are more nonspecific for alcohol but can help the clinician think of the possible effects of alcohol include elevations in blood levels or lipids (e.g., triglycerides and high-density lipoprotein cholesterol) and high-normal levels of uric acid.

Additional diagnostic markers relate to signs and symptoms that reflect the consequences often associated with persistent heavy drinking. For example, dyspepsia, nausea, and bloating can accompany gastritis, and hepatomegaly, esophageal varices, and hemorrhoids may reflect alcohol-induced changes in the liver. Other physical signs of heavy drinking include tremor, unsteady gait, insomnia, and erectile dysfunction. Males with chronic alcohol use disorder may exhibit decreased testicular size and feminizing effects associated with reduced testosterone levels. Repeated heavy drinking in females is associated with menstrual irregularities and, during pregnancy, spontaneous abortion and fetal alcohol syndrome. Individuals with preexisting histories of epilepsy or severe head trauma are more likely to develop alcohol-related seizures. Alcohol withdrawal may be associated with nausea, vomiting, gastritis, hematemesis, dry mouth, puffy blotchy complexion, and mild peripheral edema.

Association With Suicidal Thoughts or Behavior

Most studies on suicidality and alcohol address alcohol consumption rather than alcohol use disorder. However, a psychological autopsy study in Australia found that aggression, psychiatric comorbidity, and recent interpersonal conflicts are suicide risk factors in individuals with alcohol use disorder. A review of studies from 1999 through 2014 conducted in several countries, including the United States, reported that both intoxication and chronic heavy alcohol use are associated with suicide, extensive population-level data link alcohol with suicide, and there is evidence suggesting that restrictive alcohol policies may help prevent suicide on a general population level. A meta-analysis of studies conducted in the United States and several other countries from 1996 through 2015 found that compared with nondrinking individuals, the acute use of alcohol was associated with a nearly

sevenfold increase in risk of suicide attempt. Moreover, in this meta-analysis, as well as in U.S.-based case-control crossover studies, heavier alcohol use within 24 hours was a much more potent risk factor for suicide attempt than lower alcohol use. In a cohort of patients in Mississippi, acute co-use of alcohol and sedatives had an even stronger association with suicide attempt compared with alcohol use alone. A systematic review and meta-analysis of studies from 1975 through 2014 in several countries, including the United States, found that alcohol use is associated with possession of firearms, that alcohol drinkers are four to six times more likely to die by suicide with a gun than nondrinkers, and that heavy drinkers are more likely to choose firearms over other suicide methods compared with nondrinkers.

Functional Consequences of Alcohol Use Disorder

The diagnostic features of alcohol use disorder highlight major areas of life functioning likely to be impaired. These include driving and operating machinery, school and work, interpersonal relationships and communication, and health. Alcohol-related disorders contribute to absenteeism from work, job-related accidents, and low employee productivity. Rates are elevated in homeless individuals, perhaps reflecting a downward spiral in social and occupational functioning, although most individuals

with alcohol use disorder continue to live with their families and function within their jobs.

Alcohol use disorder is associated with a significant increase in the risk of accidents, violence, and suicide. It is estimated that one in five intensive care unit admissions in some urban hospitals is related to alcohol and that 40% of individuals in the United States experience an alcohol-related adverse event at some time in their lives, with alcohol accounting for up to 55% of fatal driving events. Severe alcohol use disorder, especially in individuals with antisocial personality disorder, is associated with the commission of criminal acts, including homicide. Severe problematic alcohol use also contributes to disinhibition and feelings of sadness and irritability, which contribute to suicide attempts and suicide.

Unanticipated alcohol withdrawal in hospitalized individuals for whom a diagnosis of alcohol use disorder has been overlooked can add to the risks and costs of hospitalization and to time spent in the hospital.

Differential Diagnosis

Nonpathological use of alcohol. The key element of alcohol use disorder is the use of heavy doses of alcohol with resulting repeated and significant distress or impaired functioning. While most drinkers sometimes consume enough alcohol to feel intoxicated, only a minority (< 20%) ever develop alcohol use disorder. Therefore, drinking, even daily, in low doses and occasional intoxication do not by themselves make this diagnosis.

Alcohol intoxication, alcohol withdrawal, and alcohol-induced mental disorders. Alcohol use disorder is differentiated from alcohol intoxication, alcohol withdrawal, and alcohol induced mental disorders (e.g., alcohol-induced depressive disorder) in that alcohol use disorder describes a problematic pattern of alcohol use that involves impaired control over alcohol use, social impairment due to alcohol use, risky alcohol use (e.g., driving while intoxicated), and pharmacological symptoms (the development of tolerance or withdrawal), whereas alcohol intoxication, alcohol withdrawal, and alcohol-induced mental disorders describe psychiatric syndromes that develop in the context of heavy use. Alcohol intoxication, alcohol withdrawal, and alcohol-induced mental disorders occur frequently in individuals with alcohol use disorder. In such cases, a diagnosis of alcohol intoxication, alcohol withdrawal, or an alcohol-induced mental disorder should be given in addition to

a diagnosis of alcohol use disorder, the presence of which is indicated in the diagnostic code.

Sedative, hypnotic, or anxiolytic use disorder. The signs and symptoms of alcohol use disorder are similar to those seen in sedative, hypnotic, or anxiolytic use disorder. The two must be distinguished, however, because the course may be different, especially in relation to medical problems.

Conduct disorder in childhood and antisocial personality disorder. Alcohol use disorder, along with other substance use disorders, is seen in the majority of individuals with antisocial personality disorder and preexisting conduct disorder. Because these diagnoses are associated with an early onset of alcohol use disorder as well as a worse prognosis, it is important to establish both conditions.

Comorbidity

Bipolar disorders, schizophrenia, and antisocial personality disorder are associated with alcohol use disorder, and most anxiety and depressive disorders are associated

with alcohol use disorder as well. At least a part of the reported association between depression and moderate to severe alcohol use disorder may be attributable to temporary, alcohol-induced comorbid depressive symptoms resulting from the acute effects of intoxication or withdrawal, although this point has long been debated. Severe, repeated alcohol intoxication may also suppress immune mechanisms and predispose individuals to infections and increase the risk for cancers.

Alcohol Intoxication

Diagnostic Criteria

A. Recent ingestion of alcohol.
B. Clinically significant problematic behavioral or psychological changes (e.g., inappropriate sexual or aggressive behavior, mood lability, impaired judgment) that developed during, or shortly after, alcohol ingestion.
C. One (or more) of the following signs or symptoms developing during, or shortly after, alcohol use:
    1. Slurred speech.
    2. Incoordination.
    3. Unsteady gait.
    4. Nystagmus.
    5. Impairment in attention or memory.
    6. Stupor or coma.
D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Coding note: The ICD-10-CM code depends on whether there is a comorbid alcohol use disorder. If a mild alcohol use disorder is comorbid, the ICD-10-CM code is **F10.120,** and if a moderate or severe alcohol use disorder is comorbid, the ICD-10-CM code is **F10.220.** If there is no comorbid alcohol use disorder, then the ICD-10-CM code is **F10.920.**

Diagnostic Features

The essential feature of alcohol intoxication is the presence of clinically significant and sometimes life-threatening problematic behavioral or psychological changes (e.g., inappropriate sexual or aggressive behavior, mood lability, impaired judgment and decision-making, difficulties with complex tasks such as driving, and impaired social or occupational functioning) that develop during, or shortly after, alcohol ingestion (Criterion B). These changes are accompanied by evidence of impaired functioning and judgment and, if intoxication is intense, can result in a life-threatening coma. The symptoms must not be attributable to another medical condition (e.g., diabetic ketoacidosis), are not a reflection of conditions such as delirium, and are not related to intoxication with other depressant drugs (e.g., benzodiazepines) (Criterion D). The levels of incoordination can interfere with driving abilities and performance of usual activities to the point of causing vehicle crashes or other events resulting in injury. Evidence of alcohol use can be obtained by smelling alcohol on the individual's breath,

eliciting a history from the individual or another observer, and, when needed, having the individual provide breath, blood, or urine samples for toxicology analyses.

Associated Features

Signs and symptoms of intoxication are likely to be more intense when the blood alcohol level is rising than when it is falling. The duration of intoxication depends on how much alcohol was consumed over what period of time. In general, the body is able to metabolize approximately one drink per hour, so that the blood alcohol level generally decreases at a rate of 15–20 mg/dL per hour.

During even mild alcohol intoxication, different symptoms are likely to be observed at different time points. Evidence of mild intoxication with alcohol can be seen in most individuals after approximately two drinks (each standard drink is approximately 10–12 grams of ethanol and raises the blood alcohol concentration approximately 20 mg/dL). Early in the drinking period, when blood alcohol levels are rising, symptoms often reflect stimulation (e.g., talkativeness, a sensation of well-being, a bright, expansive mood). Later, especially when blood alcohol levels are falling, the individual is likely to become progressively more depressed, withdrawn, and cognitively impaired.

Alcohol intoxication is sometimes associated with amnesia for the events that occurred during the course of the intoxication ("blackouts"). This phenomenon is related to a relatively high blood alcohol level and, perhaps, to the rapidity with which this level is reached. Acute alcohol intoxication may cause metabolic alterations (e.g., hypoglycemia, electrolyte disturbances) and may have severe cardiovascular, respiratory, and/or gastrointestinal effects. At very high blood alcohol levels (e.g., 200–300 mg/dL), an individual who has not developed tolerance for alcohol is likely to fall asleep and enter a first stage of anesthesia. Higher blood alcohol levels (e.g., in excess of 300–400 mg/dL) can cause inhibition of respiration and pulse and even death in nontolerant individuals.

Alcohol intoxication is an important contributor to interpersonal violence and suicidal behavior. Among individuals intoxicated by alcohol, there appears to be an increased rate of accidental injury (including death due to behaviors associated with altered judgment, self-harm, and violence), suicidal behavior, and suicide.

Prevalence

The large majority of alcohol consumers are likely to have been intoxicated to some degree at some point in their lives. For example, in 2018, 43% of 12th-grade students in the United States reported having "been drunk" at least once in their lifetime, and 17.5% of them reported that they had gotten drunk at least once in the prior 30 days. Using a definition of intoxication of four or more standard drinks on any day for women and five or more

standard drinks on any day for men, the 12-month prevalence of high-risk drinking in U.S. adults is 17.4% for Native Americans, 15.1% for African Americans, 13.5% for Latinx, 12.3% for non-Latinx Whites, and 7.2% for Asians and Pacific Islanders.

Development and Course

Intoxication usually occurs as an episode developing over minutes to hours and typically lasting several hours. In the United States, the average age at first intoxication is approximately 15 years, with the highest prevalence at approximately 18–25 years. Frequency and intensity usually decrease with further advancing age. The earlier the

onset of regular intoxication, the greater the likelihood the individual will go on to develop alcohol use disorder.

Risk and Prognostic Factors

Temperamental.

Episodes of alcohol intoxication increase with personality characteristics of sensation seeking and impulsivity.

Environmental.

Episodes of alcohol intoxication increase with having heavy-drinking peers, holding beliefs that heavy drinking is an important component of having fun, and using alcohol to cope with stress.

Culture-Related Diagnostic Issues

The major issues parallel the cultural differences regarding the use of alcohol overall. For example, some college fraternities and sororities encourage alcohol intoxication. This condition is also frequent on certain dates of cultural significance (e.g., New Year's Eve) and, for some subgroups, during specific events (e.g., wakes following funerals). Other subgroups encourage drinking at religious celebrations (e.g., Jewish and Catholic holidays), while still others strongly discourage all drinking or intoxication (e.g., some religious groups, such as Mormons, fundamentalist Christians, and Muslims).

Sex- and Gender-Related Diagnostic Issues

Historically, in many Western societies, acceptance of drinking and drunkenness is more tolerated for men, but such gender differences may be much less prominent in recent years, especially during adolescence and young adulthood. In general, women are less tolerant of the same amount of alcohol than men.

Diagnostic Markers

Intoxication is usually established by observing an individual's behavior and smelling alcohol on the breath. The degree of intoxication increases with an individual's blood or breath alcohol level and with the ingestion of other substances, especially those with sedating effects.

Association With Suicidal Thoughts or Behavior

A collaborative, international study in emergency departments in 17 countries found that acute alcohol use, independent of chronic use, increases the risk of suicide attempt, with each drink raising the risk by 30%. For more information, see "Association With Suicidal Thoughts or Behavior" in the Alcohol Use Disorder section.

Functional Consequences of Alcohol Intoxication

Alcohol intoxication contributed to the more than 95,000 deaths and 2.8 million years of potential life lost each year in the United States from 2011 through 2015, shortening the

lives of those who died by an average of 30 years. In addition, intoxication with this drug contributes to huge costs associated with drunk driving and lost time from school

or work, as well as interpersonal arguments and physical fights.

Differential Diagnosis

Other medical conditions. Several medical (e.g., diabetic acidosis) and neurological conditions (e.g., cerebellar ataxia, multiple sclerosis) can temporarily resemble alcohol intoxication.

Alcohol-induced mental disorders. Alcohol intoxication is distinguished from alcohol-induced mental disorders (e.g., alcohol-induced depressive disorder, with onset during intoxication) because the symptoms (e.g., depressed mood) in these latter disorders are in excess of those usually associated with alcohol intoxication, predominate in the clinical presentation, and are severe enough to warrant clinical attention.

Sedative, hypnotic, or anxiolytic intoxication. Intoxication with sedative, hypnotic, or anxiolytic drugs or with other sedating substances (e.g., antihistamines, anticholinergic drugs) can be mistaken for alcohol intoxication. The differential requires observing alcohol on the breath, measuring blood or breath alcohol levels, ordering a medical workup, and gathering a good history. The signs and symptoms of sedative-hypnotic intoxication are very similar to those observed with alcohol and include similar problematic behavioral or psychological changes. These changes are accompanied by evidence of impaired functioning and judgment—which, if intense, can result in a life-threatening coma—and levels of incoordination that can interfere with driving abilities and with performing usual activities. However, there is no smell as there is with alcohol, but there is likely to be evidence of misuse of the depressant drug in the blood or urine toxicology analyses.

Comorbidity

Alcohol intoxication may occur comorbidly with other substance intoxication, especially in individuals with conduct disorder or antisocial personality disorder. Given the typical overlap of alcohol intoxication with alcohol use disorder, see "Comorbidity" under Alcohol Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Alcohol Withdrawal

Diagnostic Criteria

A. Cessation of (or reduction in) alcohol use that has been heavy and prolonged.

B. Two (or more) of the following, developing within several hours to a few days after the cessation of (or reduction in) alcohol use described in Criterion A:

   1. Autonomic hyperactivity (e.g., sweating or pulse rate greater than 100 bpm).
   2. Increased hand tremor.
   3. Insomnia.
   4. Nausea or vomiting.
   5. Transient visual, tactile, or auditory hallucinations or illusions.
   6. Psychomotor agitation.
   7. Anxiety.
   8. Generalized tonic-clonic seizures.

C. The signs or symptoms in Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication or withdrawal from another substance.

Specify if:

With perceptual disturbances: This specifier applies in the rare instance when hallucinations (usually visual or tactile) occur with intact reality testing, or auditory, visual, or tactile illusions occur in the absence of a delirium.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid alcohol use disorder and whether or not there are perceptual disturbances.

For alcohol withdrawal, without perceptual disturbances: If a mild alcohol use disorder is comorbid, the ICD-10-CM code is **F10.130**, and if a moderate or severe alcohol use disorder is comorbid, the ICD-10-CM code is **F10.230**. If there is no comorbid alcohol use disorder, then the ICD-10-CM code is **F10.930**.

For alcohol withdrawal, with perceptual disturbances: If a mild alcohol use disorder is comorbid, the ICD-10-CM code is **F10.132**, and if a moderate or severe alcohol use disorder is comorbid, the ICD-10-CM code is **F10.232**. If there is no comorbid alcohol use disorder, then the ICD-10-CM code is **F10.932**.

Specifiers

When hallucinations occur in the absence of delirium (i.e., in a clear sensorium), a diagnosis of substance/medication-induced psychotic disorder should be considered.

Diagnostic Features

The essential feature of alcohol withdrawal is the presence of a characteristic withdrawal syndrome that develops within several hours to a few days after the cessation of (or reduction in) heavy and prolonged alcohol use (Criteria A and B). The withdrawal syndrome includes two or more of the symptoms reflecting autonomic hyperactivity and anxiety listed in Criterion B, along with gastrointestinal symptoms.

Withdrawal symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C). The symptoms must not be attributable to another medical condition and are not better explained by another mental disorder (e.g., generalized anxiety disorder), including intoxication or withdrawal from another substance (e.g., sedative, hypnotic, or anxiolytic withdrawal) (Criterion D).

Symptoms can be relieved by administering alcohol or benzodiazepines (e.g., diazepam). The withdrawal symptoms typically begin when blood concentrations of alcohol decline sharply (i.e., within 4–12 hours) after alcohol use has been stopped or reduced. Reflecting the relatively fast metabolism of alcohol, symptoms of alcohol withdrawal usually peak in intensity during the second day of abstinence and are likely to improve markedly by the fourth or fifth day. Following acute withdrawal, however,

symptoms of anxiety, insomnia, and autonomic dysfunction may persist for up to 3–6 months at lower levels of intensity.

Fewer than 10% of individuals who develop alcohol withdrawal will ever develop dramatic symptoms (e.g., severe autonomic hyperactivity, tremors, alcohol withdrawal delirium). Tonic-clonic seizures occur in fewer than 3% of individuals.

Associated Features

Although confusion and changes in consciousness are not core criteria for alcohol withdrawal, alcohol withdrawal delirium (see "Delirium" in the chapter "Neurocognitive Disorders") may occur in the context of withdrawal. As is true for any agitated, confused state, regardless of the cause, in addition to a disturbance of consciousness and cognition, withdrawal delirium can include visual, tactile, or (rarely) auditory hallucinations

(delirium tremens). When alcohol withdrawal delirium develops, it is likely that a clinically relevant medical condition may be present (e.g., liver failure, pneumonia, gastrointestinal bleeding, sequelae of head trauma, hypoglycemia, an electrolyte imbalance, postoperative status).

Prevalence

It is estimated that approximately 50% of middle-class, highly functional individuals with alcohol use disorder in the United States have ever experienced a full alcohol withdrawal syndrome. Among individuals with alcohol use disorder who are hospitalized or homeless, the rate of alcohol withdrawal may be greater than 80%. Less than 10% of individuals in withdrawal ever demonstrate alcohol withdrawal delirium or withdrawal seizures. The prevalence of alcohol withdrawal symptoms does not seem to vary across U.S. ethnoracial groups.

Development and Course

Acute alcohol withdrawal occurs as an episode usually lasting 4–5 days and only after extended periods of heavy drinking. Withdrawal is relatively rare in individuals younger than 30 years, and the risk and severity increase with increasing age.

Risk and Prognostic Factors

Alcohol withdrawal is more likely to occur with heavier alcohol intake, and that might be most often observed in individuals with conduct disorder and antisocial personality disorder. Withdrawal states are also more severe in individuals who are also dependent on other depressant drugs (sedative-hypnotics) and individuals who have had more alcohol withdrawal experiences in the past. Predictors of severe alcohol withdrawal include alcohol withdrawal delirium, prior histories of severe withdrawal syndromes, low blood potassium levels, decreased platelet counts, and systolic hypertension.

Environmental. The probability of developing alcohol withdrawal increases with the quantity and frequency of alcohol consumption. Most individuals with this condition are drinking daily, consuming large amounts (approximately more than eight drinks per day) for multiple days. However, there are large inter-individual differences, with enhanced risks for individuals with concurrent medical conditions, those with family histories of alcohol withdrawal (i.e., a genetic component), those with prior withdrawals, and individuals who consume sedative, hypnotic, or anxiolytic drugs.

Diagnostic Markers

Autonomic hyperactivity in the context of moderately high but falling blood alcohol levels and a history of prolonged, heavy drinking indicate a likelihood of alcohol withdrawal.

Functional Consequences of Alcohol Withdrawal

Symptoms of withdrawal may serve to perpetuate drinking behaviors and contribute to relapse, resulting in persistently impaired social and occupational functioning. Symptoms requiring medically supervised detoxification result in hospital utilization and loss of work productivity. Overall, the presence of withdrawal is associated with greater functional impairment and poor prognosis among individuals with alcohol use disorder.

Differential Diagnosis

Other medical conditions. The symptoms of alcohol withdrawal can also be mimicked by some medical conditions (e.g., hypoglycemia and diabetic ketoacidosis). Essential tremor,

a disorder that frequently runs in families, may erroneously suggest the tremulousness associated with alcohol withdrawal.

Alcohol-induced mental disorders. Alcohol withdrawal is distinguished from alcohol-induced mental disorders (e.g., alcohol-induced anxiety disorder, with onset during withdrawal) because the symptoms (e.g., anxiety) in these latter disorders are in excess of those usually associated with alcohol withdrawal, predominate in the clinical presentation, and are severe enough to warrant clinical attention.

Sedative, hypnotic, or anxiolytic withdrawal. Sedative, hypnotic, or anxiolytic withdrawal produces a syndrome very similar to that of alcohol withdrawal.

Comorbidity

Given the typical overlap of alcohol withdrawal with alcohol use disorder, see “Comorbidity” under Alcohol Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Alcohol-Induced Mental Disorders

The following alcohol-induced mental disorders are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): alcohol-induced psychotic disorder (“Schizophrenia Spectrum and Other Psychotic Disorders”); alcohol-induced bipolar and related disorder (“Bipolar and Related Disorders”); alcohol-induced depressive disorder (“Depressive Disorders”); alcohol-induced anxiety disorder (“Anxiety Disorders”); alcohol-induced sleep disorder (“Sleep-Wake Disorders”); alcohol-induced sexual dysfunction (“Sexual Dysfunctions”); and alcohol-induced major or mild neurocognitive disorder (“Neurocognitive Disorders”). For alcohol intoxication delirium and alcohol withdrawal delirium, see the criteria and discussion of delirium in the chapter “Neurocognitive Disorders.” These alcohol-induced mental disorders are diagnosed instead of alcohol intoxication or alcohol withdrawal only when the symptoms are sufficiently severe to warrant independent clinical attention.

Diagnostic and Associated Features

The symptom profiles for an alcohol-induced condition resemble the corresponding independent mental disorders as described elsewhere in this manual. Moreover, while alcohol-induced conditions can have the same severe consequences as independent mental disorders (e.g., suicide attempts), they are likely to improve without formal treatment in a matter of days to weeks after cessation of severe intoxication and/or withdrawal.

Each alcohol-induced mental disorder is listed in the relevant diagnostic section and therefore only a brief description is offered here. These alcohol-induced mental disorders must have developed in the context of severe alcohol intoxication and/or alcohol withdrawal.

Given that the presentation of an alcohol-induced mental disorder symptomatically resembles the presentations of independent mental disorders from the same diagnostic class, they must be differentiated based on the temporal relationship between the alcohol use and the psychiatric symptoms. Individuals with alcohol-induced mental disorders are likely to also demonstrate the associated features seen with an alcohol use disorder, as listed in that subsection.

There must be evidence that the disorder being observed is not likely to be better explained by an independent mental disorder. The latter is likely to occur if the mental

disorder was present before the severe intoxication or withdrawal, or continued for more than 1 month after the cessation of severe intoxication or withdrawal. When symptoms are observed only during a delirium, they should be considered part of the delirium and not diagnosed separately, as many symptoms (including disturbances in mood, anxiety, and reality testing) are commonly seen during agitated, confused states. The alcohol-induced mental disorder must be clinically relevant, causing significant distress or significant functional impairment. Finally, there are indications that the intake of substances of abuse in the context of a preexisting mental disorder are likely to result in an intensification of the preexisting independent syndrome.

Rates of alcohol-induced mental disorders vary somewhat by diagnostic category. For example, the lifetime risk for major depressive episodes in individuals with alcohol use disorder is approximately 40%, but only about one-third to one-half of these represent independent major depressive syndromes observed outside the context of intoxication. Similar rates of alcohol-induced sleep and anxiety disorders are likely, but alcohol-induced psychotic episodes are estimated to be seen in less than 5% of individuals with alcohol use disorder.

Development and Course

Once present, the symptoms of an alcohol-induced mental disorder are likely to remain clinically relevant as long as the individual continues to experience severe intoxication or withdrawal. While the symptoms may be identical to those of independent mental disorders (e.g., psychoses, major depressive disorder), and while they can have the same severe consequences (e.g., suicide attempts), all alcohol-induced mental disorders other than alcohol-induced neurocognitive disorder, amnestic confabulatory type (alcohol-induced persisting amnestic disorder), regardless of the severity of the symptoms, are likely to improve relatively quickly and unlikely to remain clinically relevant for more than 1 month after cessation of severe intoxication and/or withdrawal.

The alcohol-induced mental disorders are an important part of the differential diagnoses for the independent mental conditions. Independent schizophrenia, major

depressive disorder, bipolar disorder, and anxiety disorders, such as panic disorder, are likely to be associated with much longer-lasting periods of symptoms and often require longer-term medications to optimize the probability of improvement or recovery. The alcohol-induced mental disorders, on the other hand, are likely to be much shorter in duration and disappear within several days to 1 month after cessation of severe intoxication and/or withdrawal, even without psychotropic medications.

The importance of recognizing an alcohol-induced mental disorder is similar to the relevance of identifying the possible role of some endocrine conditions and medication reactions before diagnosing an independent mental disorder. In light of the high prevalence of alcohol use disorders worldwide, it is important that these alcohol-induced diagnoses be considered before independent mental disorders are diagnosed.

Unspecified Alcohol-Related Disorder

F10.99

This category applies to presentations in which symptoms characteristic of an alcohol-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific alcohol-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Caffeine-Related Disorders

Caffeine Intoxication

Caffeine Withdrawal

Caffeine-Induced Mental Disorders

Unspecified Caffeine-Related Disorder

Caffeine Intoxication

Diagnostic Criteria **F15.920**

A. Recent consumption of caffeine (typically a high dose well in excess of 250 mg).
B. Five (or more) of the following signs or symptoms developing during, or shortly after, caffeine use:
    1. Restlessness.
    2. Nervousness.
    3. Excitement.
    4. Insomnia.
    5. Flushed face.
    6. Diuresis.

7. Gastrointestinal disturbance.
8. Muscle twitching.
9. Rambling flow of thought and speech.
10. Tachycardia or cardiac arrhythmia.
11. Periods of inexhaustibility.
12. Psychomotor agitation.

C. The signs or symptoms in Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Diagnostic Features

Caffeine can be consumed from a number of different sources, including coffee, tea, caffeinated soda, "energy" drinks, over-the-counter analgesics and cold remedies, weight-loss aids, and chocolate. Caffeine is also increasingly being used as an additive to vitamins and to food products. More than 85% of children and adults in the United States consume caffeine. Some caffeine users display symptoms consistent with problematic use, including tolerance and withdrawal (see "Caffeine Withdrawal" later in this chapter); the data are not available at this time to determine the clinical significance of a caffeine use disorder and its prevalence. In contrast, there is evidence that caffeine withdrawal and caffeine intoxication are clinically significant and sufficiently prevalent.

The essential feature of caffeine intoxication is recent consumption of caffeine and five or more signs or symptoms that develop during or shortly after caffeine use (Criteria A and B). Symptoms include restlessness, nervousness, excitement, insomnia, flushed face, diuresis, and gastrointestinal complaints, which can occur with low doses (e.g., 200 mg) in

vulnerable individuals such as children, the elderly, or individuals who have not been exposed to caffeine previously. Symptoms that generally appear at levels of more than 1 g/day include muscle twitching, rambling flow of thought and speech, tachycardia or cardiac arrhythmia, periods of inexhaustibility, and psychomotor agitation. Caffeine intoxication may not occur despite high caffeine intake because of the development of tolerance. The signs or symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C). The signs or symptoms must not be attributable to another medical condition and are not better explained by another mental disorder (e.g., an anxiety disorder) or intoxication with another substance (Criterion D).

Associated Features

Mild sensory disturbances (e.g., ringing in the ears and flashes of light) may occur with high doses of caffeine. Although large doses of caffeine can increase heart rate, typical dietary doses can slow heart rate. Whether excess caffeine intake can cause headaches is unclear. On physical examination, agitation, restlessness, sweating, tachycardia, flushed face, and increased bowel motility may be seen. Caffeine blood levels may provide important information for diagnosis, particularly when the individual is a poor

historian, although these levels are not diagnostic by themselves in view of the individual variation in response to caffeine.

Prevalence

The prevalence of caffeine intoxication in the general population is unclear. In the United States, approximately 7% of individuals in the population may experience five or more symptoms along with functional impairment consistent with a diagnosis of caffeine intoxication.

Consumption of caffeinated energy drinks, often together with alcohol, leading to caffeine intoxication, has increased among adolescents and young adults in high-income countries, resulting in the doubling of U.S. emergency department visits related to caffeinated energy drinks between 2007 and 2011.

Development and Course

Consistent with a half-life of caffeine of approximately 4–6 hours, caffeine intoxication symptoms usually remit within the first day or so and do not have any known long-lasting consequences. However, individuals who consume very high doses of caffeine (i.e., 5–10 g) may require immediate medical attention, as such doses can be lethal.

With advancing age, individuals are likely to demonstrate increasingly intense reactions to caffeine, with greater complaints of interference with sleep or feelings of hyperarousal. Caffeine intoxication among young individuals after consumption of highly caffeinated products, including energy drinks, has been observed. Children and adolescents may be at increased risk for caffeine intoxication because of low body weight, lack of tolerance, and lack of knowledge about the pharmacological effects of caffeine.

Risk and Prognostic Factors

Environmental. Caffeine intoxication is often seen among individuals who use caffeine less frequently or in those who have recently increased their caffeine intake by a substantial amount. Furthermore, oral contraceptives significantly decrease the elimination of caffeine and consequently may increase the risk of intoxication.

Genetic and physiological. Genetic factors may affect risk of caffeine intoxication.

Functional Consequences of Caffeine Intoxication

Impairment from caffeine intoxication may have serious consequences, including dysfunction at work or school, social indiscretions, or failure to fulfill role obligations. Moreover, extremely high doses of caffeine can be fatal. In some cases, caffeine intoxication may precipitate a caffeine-induced disorder.

Differential Diagnosis

Independent mental disorders. Caffeine intoxication may be characterized by symptoms (e.g., panic attacks) that resemble independent mental disorders. To meet criteria for caffeine intoxication, the symptoms must not be associated with another medical condition or another mental disorder, such as an anxiety disorder, that could better explain them. Manic episodes; panic disorder; generalized anxiety disorder; amphetamine intoxication; sedative, hypnotic, or anxiolytic withdrawal or tobacco withdrawal; sleep disorders; and medication-induced side effects (e.g., akathisia) can cause a clinical picture that is similar to that of caffeine intoxication.

Caffeine-induced mental disorders.

The temporal relationship of the symptoms to increased caffeine use or to abstinence from caffeine helps to establish the diagnosis. Caffeine intoxication is differentiated from caffeine-induced anxiety disorder, with onset during intoxication (see "Substance/Medication-Induced Anxiety Disorder" in the chapter "Anxiety Disorders"), and caffeine-induced sleep disorder, with onset during intoxication (see "Substance/Medication-Induced Sleep Disorder" in the chapter "Sleep-Wake Disorders"), because the symptoms (e.g., anxiety and insomnia, respectively) in these latter disorders are in excess of those usually associated with caffeine intoxication, predominate in the clinical presentation, and are severe enough to warrant independent clinical attention.

Comorbidity

Typical dietary doses of caffeine have not been consistently associated with medical problems. However, heavy use (e.g., > 400 mg) can cause or exacerbate anxiety and somatic symptoms and gastrointestinal distress. With acute, extremely high doses of caffeine, grand mal seizures and respiratory failure may result in death. Excessive caffeine use is associated with depressive disorders, bipolar disorders, eating disorders, psychotic disorders, sleep disorders, and substance-related disorders, whereas individuals with anxiety disorders are more likely to avoid caffeine.

Caffeine Withdrawal

Diagnostic Criteria **F15.93**

A. Prolonged daily use of caffeine.
B. Abrupt cessation of or reduction in caffeine use, followed within 24 hours by three (or more) of the following signs or symptoms:
   1. Headache.
   2. Marked fatigue or drowsiness.
   3. Dysphoric mood, depressed mood, or irritability.
   4. Difficulty concentrating.
   5. Flu-like symptoms (nausea, vomiting, or muscle pain/stiffness).

C. The signs or symptoms in Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
D. The signs or symptoms are not associated with the physiological effects of another medical condition (e.g., migraine, viral illness) and are not better explained by another mental disorder, including intoxication or withdrawal from another substance.

Diagnostic Features

The essential feature of caffeine withdrawal is the presence of a characteristic withdrawal syndrome that develops after the abrupt cessation of (or substantial reduction in) prolonged daily caffeine ingestion (Criterion B). Because individuals may be unaware of the wide array of sources of caffeine beyond coffee, colas, and energy drinks (e.g., over-the-counter analgesics and cold remedies, weight loss aids,

chocolate), they may not connect ingestion of these substances with symptoms of caffeine withdrawal. The caffeine withdrawal syndrome is indicated by three or more of the following (Criterion B): headache; marked fatigue or drowsiness; dysphoric mood, depressed mood, or irritability; difficulty concentrating; and flu-like symptoms (nausea, vomiting, or muscle pain/stiffness). The withdrawal syndrome causes clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C). The symptoms must not be associated with the physiological effects of another medical condition and are not better explained by another mental disorder (Criterion D).

Headache is the hallmark feature of caffeine withdrawal and may be diffuse, gradual in development, throbbing, severe, and sensitive to movement. However, other symptoms of caffeine withdrawal can occur in the absence of headache. Caffeine is the most widely used behaviorally active drug in the world and is present in many different types of beverages (e.g., coffee, tea, mate, soft drinks, energy drinks), foods, energy aids, medications, and dietary supplements. Because caffeine ingestion is often integrated into social customs and daily rituals (e.g., coffee break, tea time), some caffeine consumers may be unaware of their physical dependence on caffeine. Thus, caffeine withdrawal symptoms could be unexpected and misattributed to other causes (e.g., the flu, migraine). Furthermore, caffeine withdrawal symptoms may occur when individuals are required to abstain from foods and beverages prior to medical procedures or when a usual caffeine dose is missed because of a change in routine (e.g., during travel, weekends).

The probability and severity of caffeine withdrawal generally increase as a function of usual daily caffeine dose. However, there is large variability among individuals and within individuals across different episodes in the incidence, severity, and time course of withdrawal symptoms. Caffeine withdrawal symptoms may occur after abrupt cessation of relatively low chronic daily doses of caffeine (i.e., 100 mg).

Associated Features

Caffeine abstinence has been shown to be associated with impaired behavioral and cognitive performance (e.g., sustained attention), as well as with increased total sleep time, sleep efficiency, and slow-wave sleep. Electroencephalographic studies have shown that caffeine withdrawal symptoms are significantly associated with increases in theta power and decreases in beta-2 power. Decreased motivation to work and decreased sociability have also been reported during caffeine withdrawal. Increased analgesic use during caffeine withdrawal has been documented.

Prevalence

More than 85% of adults and children in the United States regularly consume caffeine, with adult caffeine consumers ingesting about 280 mg/day on average. The incidence and prevalence of the caffeine withdrawal syndrome in the general population are unclear. In the United States, headache may occur in approximately 50% of cases of caffeine abstinence.

In attempts to permanently stop caffeine use, more than 70% of individuals in a U.S. metropolitan county reported at least one caffeine withdrawal symptom (47% experienced headache), and 24% experienced headache plus one or more other symptoms as well as functional impairment due to withdrawal. Among individuals who abstained from caffeine for at least 24 hours but were not trying to permanently stop caffeine use, 11% experienced headache plus one or more other symptoms as well as functional impairment. Caffeine consumers can decrease the incidence of caffeine

withdrawal by using caffeine daily or only infrequently (e.g., no more than 2 consecutive days). Gradual reduction in caffeine over a period of days or weeks may decrease the incidence and severity of caffeine withdrawal.

Development and Course

Symptoms usually begin 12–24 hours after the last caffeine dose and peak after 1–2 days of abstinence. Caffeine withdrawal symptoms last for 2–9 days, with the possibility of withdrawal headaches occurring for up to 21 days. Symptoms usually remit rapidly (within 30–60 minutes) after re-ingestion of caffeine. Doses of caffeine significantly less than the individual's usual daily dose may be sufficient to prevent or attenuate caffeine withdrawal symptoms (e.g., consumption of 25 mg by an individual who typically consumes 300 mg).

Caffeine is unique in that it is a behaviorally active drug that is consumed by individuals of nearly all ages, with rates of caffeine consumption and overall level of caffeine consumption increasing with age. Although caffeine withdrawal among children and adolescents has been documented, relatively little is known about risk factors for caffeine withdrawal among this age group. The use of highly caffeinated energy drinks is increasing in young people, which could increase the risk for caffeine withdrawal.

Risk and Prognostic Factors

Temperamental. Heavy caffeine use has been observed among individuals with mental disorders, including eating disorders and alcohol and other substance use disorders, as well as among individuals who smoke cigarettes and those who are incarcerated. Thus, these individuals could be at higher risk for caffeine withdrawal upon acute caffeine abstinence.

Environmental. The unavailability of caffeine is an environmental risk factor for incipient withdrawal symptoms. While caffeine is legal and usually widely available, there are conditions in which caffeine use may be restricted, such as during medical procedures, pregnancy, hospitalizations, religious observances, wartime, travel, and research participation. These external environmental circumstances may precipitate a withdrawal syndrome in vulnerable individuals.

Genetic and physiological. Genetic factors appear to increase vulnerability to caffeine withdrawal, but no specific genes have been identified.

Culture-Related Diagnostic Issues

Habitual caffeine consumers who fast for religious reasons may be at increased risk for caffeine withdrawal.

Sex- and Gender-Related Diagnostic Issues

Metabolism of caffeine is slower in females who use oral contraceptives and in the luteal phase of the menstrual cycle, and caffeine metabolism becomes progressively slower in the second and third trimesters of pregnancy compared with the first trimester and the nonpregnant state. These features reduce the rate of clearance and may diminish withdrawal, although they can also lengthen the duration of caffeine-associated adverse symptoms. It is unlikely that doses < 300 mg/day are associated with adverse reproductive outcomes in pregnancy.

Functional Consequences of Caffeine Withdrawal

Caffeine withdrawal symptoms can vary from mild to extreme, at times causing functional impairment in normal daily activities. Rates of functional impairment in studies conducted largely in the United States range from 10% to 55% (median 13%), with rates as high as 73% found among individuals who also show other problematic features of caffeine use. Examples of functional impairment include being unable to work, exercise, or care for children; staying in bed all day; missing religious services; ending a vacation early; and canceling a social gathering. Caffeine withdrawal headaches may be described by individuals as "the worst headaches" ever experienced. Decrements in cognitive and motor performance have also been observed.

Differential Diagnosis

Other medical conditions and medication side effects. Caffeine withdrawal can mimic migraine and other headache disorders, viral illnesses, sinus conditions, tension, other drug withdrawal states (e.g., from amphetamines, cocaine), and medication side effects. The final determination of caffeine withdrawal should rest on a determination of the pattern and amount consumed, the time interval between caffeine abstinence and onset of symptoms, and the particular clinical features presented by the individual. A challenge dose of caffeine followed by symptom remission may be used to confirm the diagnosis.

Caffeine-induced sleep disorder. Caffeine withdrawal is distinguished from caffeine-induced sleep disorder (e.g., caffeine-induced sleep disorder, insomnia type, with onset during withdrawal) because the sleep symptoms are in excess of those usually associated caffeine withdrawal, predominate in the clinical presentation, and are severe enough to warrant clinical attention.

Comorbidity

Caffeine withdrawal may be associated with major depressive disorder, generalized anxiety disorder, panic disorder, antisocial personality disorder, moderate to severe alcohol use disorder, and cannabis and cocaine use.

Caffeine-Induced Mental Disorders

The following caffeine-induced mental disorders are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): caffeine-induced anxiety disorder ("Anxiety Disorders") and caffeine-induced sleep disorder ("Sleep-Wake Disorders"). These caffeine-induced mental disorders are diagnosed instead of caffeine intoxication or caffeine withdrawal only when the symptoms are sufficiently severe to warrant independent clinical attention.

Unspecified Caffeine-Related Disorder

F15.99

This category applies to presentations in which symptoms characteristic of a caffeine-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific caffeine-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Cannabis-Related Disorders

Cannabis Use Disorder

Cannabis Intoxication

Cannabis Withdrawal

Cannabis-Induced Mental Disorders

Unspecified Cannabis-Related Disorder

Cannabis Use Disorder

Diagnostic Criteria

A. A problematic pattern of cannabis use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. Cannabis is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control cannabis use.
3. A great deal of time is spent in activities necessary to obtain cannabis, use cannabis, or recover from its effects.
4. Craving, or a strong desire or urge to use cannabis.
5. Recurrent cannabis use resulting in a failure to fulfill major role obligations at work, school, or home.
6. Continued cannabis use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of cannabis.
7. Important social, occupational, or recreational activities are given up or reduced because of cannabis use.
8. Recurrent cannabis use in situations in which it is physically hazardous.
9. Cannabis use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by cannabis.
10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of cannabis to achieve intoxication or desired effect.
    b. Markedly diminished effect with continued use of the same amount of cannabis.
11. Withdrawal, as manifested by either of the following:
    a. The characteristic withdrawal syndrome for cannabis (refer to Criteria A and B of the criteria set for cannabis withdrawal).
    b. Cannabis (or a closely related substance) is taken to relieve or avoid withdrawal symptoms.

Specify if:

In early remission: After full criteria for cannabis use disorder were previously met, none of the criteria for cannabis use disorder have been met for at least 3 months but

for less than 12 months (with the exception that Criterion A4, "Craving, or a strong desire or urge to use cannabis," may be met).

In sustained remission: After full criteria for cannabis use disorder were previously met, none of the criteria for cannabis use disorder have been met at any time during a period of 12 months or longer (with the exception that Criterion A4, "Craving, or a strong desire or urge to use cannabis," may be present).

Specify if:

In a controlled environment: This additional specifier is used if the individual is in an environment where access to cannabis is restricted.

Code based on current severity/remission: If a cannabis intoxication, cannabis withdrawal, or another cannabis-induced mental disorder is also present, do not use the codes below for cannabis use disorder. Instead, the comorbid cannabis use disorder is indicated in the 4th character of the cannabis-induced disorder code (see the coding note for cannabis intoxication, cannabis withdrawal, or a specific cannabis-induced mental disorder). For example, if there is comorbid cannabis-induced anxiety disorder and cannabis use disorder, only the cannabis-induced anxiety disorder code is given, with the 4th character indicating whether the comorbid cannabis use disorder is mild, moderate, or severe: F12.180 for mild cannabis use disorder with cannabis-induced anxiety disorder or F12.280 for a moderate or severe cannabis use disorder with cannabis-induced anxiety disorder.

Specify current severity/remission:

F12.10 Mild: Presence of 2–3 symptoms.

F12.11 Mild, In early remission

F12.11 Mild, In sustained remission

F12.20 Moderate: Presence of 4–5 symptoms.

F12.21 Moderate, In early remission

F12.21 Moderate, In sustained remission

F12.20 Severe: Presence of 6 or more symptoms.

F12.21 Severe, In early remission

F12.21 Severe, In sustained remission

Specifiers

"In a controlled environment" applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

Changing severity across time in an individual may also be reflected by changes in the frequency (e.g., days of use per month or times used per day) and/or dose (e.g.,

amount used per episode) of cannabis, as assessed by individual self-report, report of knowledgeable others, clinician's observations, and biological testing.

Diagnostic Features

Cannabis use disorder includes problems associated with use of substances derived from the cannabis plant and chemically similar synthetic compounds. In these substances, the primary component with psychoactive effects (and hence, addiction potential) is the cannabinoid delta-9-tetrahydrocannabinol (delta-9-THC or THC). Cannabinoids have diverse effects in the brain, prominent among which are actions on CB_1 and CB_2 cannabinoid receptors found throughout the central nervous system.

Cannabis is used in many forms. It is most commonly smoked in a cigarette-like form (often called "joints" or "reefers"), and also in pipes, water pipes (bongs or hookahs), or hollowed-out cigars ("blunts"). More recently developed methods include "vaping" (vaporizing) by heating without combustion plant cannabis material to release psychoactive components for inhalation, and "dabbing," in which a concentrated cannabis product (butane hash oil, known as "dabs"), created through butane extraction of THC from cannabis plant material, is heated and inhaled. Vaping and dabbing are gaining popularity, particularly among youth. Cannabis can also be ingested orally in food (edibles) or beverages. Inhalation typically produces more rapid and intense onset of effects than oral administration. Hashish or hash oil, a concentrated extraction of the cannabis plant, is also used. Across products, cannabis potency (THC concentration) varies greatly, averaging 10%–15% in typical cannabis plant material, 30%–40% in hashish, and 50%–55% in hash oil. During the past two decades, the potency of seized illegal plant cannabis has steadily increased, and legal cannabis products may have even higher THC potency (e.g., 20% for plant material and 68% for cannabis extracts). Synthetic oral THC formulations (pill/capsules/sprays) are also available for various medical uses (e.g., chronic pain; nausea and vomiting caused by chemotherapy or ano-rexia; weight loss among those with AIDS). Other entirely illicit synthetic cannabinoid compounds (e.g., K2, Spice, JWH-018, JWH-073) are in the form of plant material sprayed with a cannabinoid formulation. Although such synthetic cannabinoids are designed to mimic cannabis effects, their chemical composition, potency, effects, and duration of action are unpredictable, and they may cause more severe adverse effects than cannabis plant products, including seizures, cardiac conditions, psychosis, and even death.

In the United States, cannabis remains an illegal substance under federal law, while the legal status of cannabis varies by state. Thus, cannabis use under state law can involve an illicit product, a product authorized for medical purposes, or a completely legal product. The most common medical purpose for cannabis use is chronic pain, and the conditions approved for medicinal cannabis use vary from state to state. When cannabis or a cannabinoid is taken as indicated for a medical condition, tolerance and withdrawal (physiological dependence) may occur but should not be the primary basis for diagnosing cannabis use disorder. The efficacy of cannabis for different medical conditions continues to be debated, and cannabis use as medically advised should be taken into account when a cannabis use disorder diagnosis is being considered.

Patterns of cannabis use can range from light, infrequent use to heavy, frequent use. Individuals with DSM-5 cannabis use disorder use cannabis frequently (on average, 4 or more days a week), and some individuals may use cannabis throughout the day over a period of months or years. Because of the increasingly common perception that cannabis use is harmless, individuals may not recognize that symptoms of cannabis use disorder (e.g., withdrawal symptoms) are cannabis related. Additionally, among

individuals with multiple substance use disorders, lack of clarity about whether symptoms are caused by cannabis or by other substances may lead to underreporting of cannabis use disorder symptoms.

Cannabis use disorder is defined by the same 11 criteria that define the other substance use disorders, as supported by considerable empirical evidence. These criteria, a cluster of behavioral and physical symptoms, lead to clinically significant impairment or distress and can include withdrawal, tolerance, craving, spending a great deal of time in activities related to the substance, and hazardous use (e.g., driving while under its influence). Some individuals who use cannabis multiple times per day do not perceive themselves as spending excessive time under the influence of cannabis or recovering from its effects, despite being intoxicated from cannabis or coming down from its effects most of the time, most days. An important marker of a severe cannabis use disorder is continued use despite negative effects on other important activities or relationships (e.g., school, work, sports, partner or parent relationship).

Regular cannabis users become tolerant to many acute cannabis effects, and cessation of regular cannabis use generally leads to a cannabis withdrawal syndrome. Cannabis withdrawal can cause significant distress, leading to continued use to relieve the symptoms and difficulty quitting use or relapse.

Associated Features

Individuals who regularly use cannabis often report using it to cope with mood, insomnia, anger, pain, or other physiological or psychological problems, and individuals diagnosed with cannabis use disorder frequently have other concurrent mental disorders. Careful assessment can reveal that cannabis use contributes to exacerbation of these symptoms, as well as other reasons for frequent use (e.g., the coping motives listed above; to experience euphoria; as an enjoyable social activity). Chronic intake of cannabis can produce a lack of motivation that resembles persistent depressive disorder.

Because some individuals may underreport the amount or frequency of their cannabis use, provider awareness of common signs and symptoms of cannabis use and intoxication facilitates better assessment of cannabis use disorder. Some additional signs of acute and chronic use are red eyes (conjunctival injection), cannabis odor on clothing, yellowing of fingertips (from smoking joints), chronic cough, burning of incense (to hide the odor), and exaggerated craving and impulse for specific foods, sometimes at odd times of the day or night.

Prevalence

Cannabinoids, especially cannabis, are the most widely used illicit psychoactive substances in the United States. The following prevalence data are drawn from U.S.-based studies, unless otherwise noted. Among youth (ages 12–17 years), the past-year prevalence of DSM-IV cannabis use disorder is 2.7%–3.1%. Among adults age 18 years and older, the prevalence is 1.5%–2.9%. Among cannabis users, the prevalence of DSM-IV cannabis use disorder is 20.4% among youth and 30.6% among adults. For DSM-5 cannabis use disorder, 12-month prevalence is approximately 2.5% among adults (1.4%, 0.6%, and 0.6% at mild, moderate, and severe levels, respectively). During the past decade, the prevalence of cannabis use disorder has decreased among adolescents. In contrast, among adults, some studies suggest that the prevalence of cannabis use disorder has either remained stable or increased—for example, among adults in the general population, patients in inpatient settings, and patients in the Veterans Health Administration. Globally, the age-standardized rate of cannabis use

disorders was 289.7 per 100,000 people in 2016, a 25.6% increase over 1990. Prevalence varies widely across geographic regions, being lowest in Western Sub-Saharan Africa and highest in North America.

According to age, the prevalence of cannabis use disorder in the United States is highest among individuals ages 18–29 years (6.9%) and lowest among individuals age 45 years and older (0.8%). Rates of cannabis use disorder are greater in men than in women (3.5% vs. 1.7%) and in boys than in girls ages 12–17 years (3.4% vs. 2.8%), although gender differences have been narrowing in recent birth cohorts across several countries. Regarding ethnoracial differences, for adolescents ages 12–17 years, rates are highest among Hispanics (3.8%), followed by Whites (3.1%), African Americans (2.9%), and other ethnoracial groups (2.3%). Among adults, the prevalence of cannabis use disorder is 5.3% in American Indians and Alaska Natives, 4.5% in African Americans, 2.6% in Hispanics, 2.2% in Whites, and 1.3% in Asians and Pacific Islanders. In the United States and other high-income countries, the number of individuals seeking treatment for cannabis-related problems has increased since the 1990s. However, among adults with cannabis use disorder, only 7%–8% received any type of cannabis-specific treatment in the past year, indicating that cannabis use disorder is a seriously undertreated condition.

Development and Course

The onset of cannabis use disorder can occur at any age but is most common during adolescence or young adulthood. The increasing acceptability and availability of medical and recreational marijuana may impact the development and course of cannabis use disorder, with increased onset among older adults.

Generally, cannabis use disorder develops over an extended period of time, although the progression may be more rapid in adolescents, particularly in those with conduct problems. Most individuals who develop a cannabis use disorder establish a pattern of cannabis use that gradually increases in frequency and amount. Beginning around 2010, cannabis has increasingly displaced alcohol and tobacco in the United States as the first psychoactive substance used during adolescence. This may be attributable to the decrease in perceived harmfulness of cannabis use among adolescents and adults and the fact that many now perceive cannabis use as less harmful than alcohol or tobacco use.

Cannabis use disorder among preteens, adolescents, and young adults is associated with preferences for novelty-seeking and risk-taking, norm-violating or other illegal behaviors, and conduct disorder. Milder cases of cannabis use disorder in youth primarily reflect continued use despite problems related to disapproval of use by peers, school administration, or family, and can place youths at risk for physical or behavioral consequences. In more severe cases, progression to using alone or using throughout the day interferes with daily functioning and takes the place of previously established, prosocial activities.

Cannabis use disorder among adults typically involves well-established patterns of daily cannabis use that continue despite clear psychosocial or medical problems. Many adults experience repeated desire to stop or have failed at repeated cessation attempts. Milder adult cases may resemble mild adolescent cases in that cannabis use is not as frequent or heavy but continues despite potential significant consequences of sustained use. The rate of use among U.S. middle-age and older adults is increasing, which may be atributable to increased availability and acceptability, along with a possible “baby boomer” cohort effect resulting from high prevalence of use among those who were young adults in the late 1960s and the 1970s.

Early onset of cannabis use (e.g., prior to age 15 years) is a robust predictor of the development of cannabis use disorder and other types of substance use disorders and mental disorders during young adulthood. Such early onset is often concurrent with other externalizing problems (e.g., symptoms of conduct disorder). However, early onset is also a predictor of internalizing problems and as such may reflect a general risk factor for the development of mental disorders.

Risk and Prognostic Factors

Temperamental. A history of conduct disorder in childhood or adolescence and antisocial personality disorder are risk factors for the development of many substance use disorders, including cannabis use disorder. Other risk factors include externalizing or internalizing disorders during childhood or adolescence. Youth with high behavioral disinhibition scores show early-onset substance use disorders, including cannabis use disorder and multiple substance involvement, and early conduct problems.

Environmental. Risk factors include unstable or abusive family situations, use of cannabis among immediate family members, a childhood history of emotional or physical abuse or the violent death of a close family member or friend, a family history of substance use disorders, and low socioeconomic status. As with all substances of abuse, the ease of availability of the substance is a risk factor; cannabis is relatively easy to obtain in most cultures, which increases the risk of developing a cannabis use disorder. Increasingly permissive U.S. state medical and recreational marijuana laws have reduced barriers to obtaining cannabis in about two-thirds of U.S. states. Living in a U.S. state that has legalized recreational

marijuana use increases the risk for adult cannabis use disorder. The risk of the disorder among past-year cannabis users is higher among Black, Native American, Hispanic, and Asian American adults and adolescents, relative to non-Hispanic Whites.

Genetic and physiological. Genetic influences contribute to the development of cannabis use disorders. Heritable factors contribute between 30% and 80% of the total variance in risk of cannabis use disorders, although studies have not yet definitively identified the specific genetic variants involved. Genetic and environmental influences shared between cannabis and other types of substance use disorders suggest a general common basis for substance use disorders that includes cannabis use disorder.

Culture-Related Diagnostic Issues

The acceptability of cannabis for medical and recreational use has varied widely over time and across cultural contexts. Currently, cannabis is one of the world's most commonly used psychoactive substances. In some cultural settings, cannabis use is influenced by ethnicity, religion, and sociocultural practices, such as political movements.

Sex- and Gender-Related Diagnostic Issues

Compared with men, women report more severe cannabis withdrawal symptoms, especially mood symptoms such as irritability, restlessness, and anger, and gastrointestinal symptoms such as stomachache and nausea, which may contribute to potential telescoping (faster transition from first cannabis use to cannabis use disorder).

Past-month cannabis use was reported by 7.0% of pregnant women in a nationally representative U.S. survey in 2016–2017. The rate of cannabis use is lower in pregnant compared with nonpregnant women, but resumption of use following delivery occurs in the majority who attain abstinence in pregnancy.

Diagnostic Markers

Detection of 11-nor-9-carboxy-delta-9-tetrahydrocannabinol (THCCOOH) in urine is often used as a biological marker of cannabis use. In frequent users, urine tests for THCCOOH often remain positive for weeks after last use, limiting the uses for these tests (e.g., remission status), and expertise in urine testing methods is needed to reliably interpret results. However, a positive result can be useful in working with individuals who deny all use despite concerns of family or friends. Tests for the presence of cannabinoids in blood that give more fine-grained results are under active development, and the development of detection using oral fluids may eventually offer the possibility of roadside tests to use in driving safety efforts.

Association With Suicidal Thoughts or Behavior

In a study of Iraq/Afghanistan-era veterans, after adjustment for multiple sociodemographic factors, psychiatric and other substance comorbidities, and past trauma, including combat, cannabis use disorder was still associated with increased risk of both suicidal and nonsuicidal self-injury. In a study of all U.S. Veterans Health Administration patients in 2005, any current substance use disorder was associated with increased suicide risk in both sexes but especially among women. In particular, men with cannabis use disorder had a suicide rate of 79 per 100,000 person-years, and women with cannabis use disorder had a suicide rate of 47 per 100,000 person-years. A review and meta-analysis of the international literature from 1990 through 2015 found evidence that chronic cannabis use, but not acute cannabis use, is associated with suicidal thoughts and behavior.

Functional Consequences of Cannabis Use Disorder

Functional consequences of cannabis use disorder are part of the diagnostic criteria. Many areas of psychosocial, cognitive, and health functioning may be compromised in relation to

cannabis use disorder. Although it can be difficult to distinguish the short-term impairments due to cannabis intoxication from the longer-term functional consequences of cannabis use disorder, cognitive function (particularly higher executive function) even while unintoxicated may become compromised in cannabis users in a cumulative dose-dependent relationship, which may contribute to difficulty at school or work. Accidents due to potentially dangerous activities while under the influence (e.g., driving, sports, at work) are also of concern. In particular, placebo-controlled studies and large-scale epidemiological studies show that cannabis use impairs driver reaction time, spatial perceptions, and decision-making. Cannabis use has also been linked to a reduction in goal-directed activity and decreased self-efficacy, labeled an *amotivational syndrome*, that manifests itself in poor school or work performance. Similarly, cannabis-associated problems with social relationships are commonly reported in those with cannabis use disorder. Cannabis use is associated with poorer life satisfaction and increased treatment and hospitalization for mental health problems.

Differential Diagnosis

Nonproblematic use of cannabis. Although the majority of individuals who use cannabis do not have problems related to its use, 20%–30% of cannabis users do experience symptoms and associated consequences consistent with a cannabis use disorder. Differentiating nonproblematic use of cannabis and cannabis use disorder can be

challenging because individuals may not attribute cannabis-related social, behavioral, or psychological problems to the substance, especially in the context of polysubstance use. Also, failure to acknowledge heavy cannabis use and its role in associated problems is common among individuals referred to treatment by others (i.e., school, family, employer, criminal justice system).

Cannabis intoxication, cannabis withdrawal, and cannabis-induced mental disorders. Cannabis use disorder is differentiated from cannabis intoxication, cannabis withdrawal, and cannabis-induced mental disorders (e.g., cannabis-induced anxiety disorder) in that cannabis use disorder describes a problematic pattern of cannabis use that involves impaired control over cannabis use, social impairment due to cannabis use, risky cannabis use (e.g., driving while intoxicated), and pharmacological symptoms (the development of tolerance or withdrawal), whereas cannabis intoxication, cannabis withdrawal, and cannabis-induced mental disorders describe psychiatric syndromes that develop in the context of heavy use. Cannabis intoxication, cannabis withdrawal, and cannabis-induced mental disorders occur frequently in individuals with cannabis use disorder. In such cases, a diagnosis of cannabis intoxication, cannabis withdrawal, or a cannabis-induced mental disorder should be given in addition to a diagnosis of cannabis use disorder, the presence of which is indicated in the diagnostic code.

Comorbidity

Cannabis use disorder is highly comorbid with other substance use disorders (e.g., alcohol, cocaine, opioids). For example, compared with adults without cannabis use disorder, having a cannabis use disorder multiplies the risk for any other substance disorder by a factor of about nine. Cannabis has been commonly considered as a "gateway" drug because individuals who use cannabis have a substantially greater lifetime probability than nonusers of subsequently using other, more risky substances (e.g., opioids or cocaine). Among adults seeking treatment for a cannabis use disorder, many (63%) report problematic use of secondary or tertiary substances, including alcohol, cocaine, methamphetamine/amphetamine, and heroin or other opiates, and cannabis use disorder is often a secondary or tertiary problem among those with a primary diagnosis of other substance use disorders. Among adolescents in treatment, cannabis is frequently the primary substance of abuse (76%).

Among adults with DSM-5 cannabis use disorder, 64% had a past-year tobacco use disorder, and the odds of a comorbid tobacco disorder increased sharply as the severity of cannabis use disorder increased.

Co-occurring mental disorders are also common among those with cannabis use disorder and include major depressive disorder, bipolar I and II disorders, anxiety disorders, posttraumatic stress disorder, and personality disorders. In a Minnesota twin study, about half of adolescents with cannabis use disorder had internalizing disorders (e.g., anxiety, depression, posttraumatic stress disorder), and 64% had externalizing disorders (e.g., conduct disorder, attention-deficit/hyperactivity disorder).

Considerable concern has been raised about cannabis use as a risk factor in schizophrenia and other psychotic disorders. Cannabis use in critical periods is consistently associated with a threefold increase in the risk for psychosis. Differences in frequency of daily cannabis use and use of high-potency varieties of cannabis may have contributed to the striking variation in the incidence of psychotic disorder across 11 European sites. The population attributable fraction from regular cannabis in explaining hospital admissions for psychosis was estimated to be 17.7% (95% CI: 1.2%–45.5%) in Chile. On the other hand, some data suggest that childhood abuse may be the determining factor that increases the risk for cannabis abuse and for psychosis.

Overall, cannabis use may contribute to the onset of an acute psychotic episode, can exacerbate some symptoms, and can adversely affect treatment of a major psychotic illness.

Regarding medical conditions, cannabinoid hyperemesis syndrome is a syndrome of nausea and cyclic vomiting associated with regular cannabis use that is increasingly seen in emergency departments as the prevalence of cannabis use increases. In addition, respiratory disorders (e.g., asthma, chronic obstructive pulmonary disease, pneumonia) are associated with regular cannabis use (by smoking, vaping, or e-cigarettes) regardless of tobacco co-use, as are some adverse cardiovascular outcomes.

Cannabis Intoxication

Diagnostic Criteria

A. Recent use of cannabis.

B. Clinically significant problematic behavioral or psychological changes (e.g., impaired motor coordination, euphoria, anxiety, sensation of slowed time, impaired judgment, social withdrawal) that developed during, or shortly after, cannabis use.

C. Two (or more) of the following signs or symptoms developing within 2 hours of cannabis use:

1. Conjunctival injection.
2. Increased appetite.
3. Dry mouth.
4. Tachycardia.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Specify if:

With perceptual disturbances: Hallucinations with intact reality testing or auditory, visual, or tactile illusions occur in the absence of a delirium.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid cannabis use disorder and whether or not there are perceptual disturbances.

For cannabis intoxication, without perceptual disturbances: If a mild cannabis use disorder is comorbid, the ICD-10-CM code is **F12.120,** and if a moderate or severe cannabis use disorder is comorbid, the ICD-10-CM code is **F12.220.** If there is no comorbid cannabis use disorder, then the ICD-10-CM code is **F12.920.**

For cannabis intoxication, with perceptual disturbances: If a mild cannabis use disorder is comorbid, the ICD-10-CM code is **F12.122,** and if a moderate or severe cannabis use disorder is comorbid, the ICD-10-CM code is **F12.222.** If there is no comorbid cannabis use disorder, then the ICD-10-CM code is **F12.922.**

Specifiers

When hallucinations occur in the absence of intact reality testing, a diagnosis of substance/medication-induced psychotic disorder should be considered.

Diagnostic Features

The essential feature of cannabis intoxication is the presence of clinically significant problematic behavioral or psychological changes that develop during, or shortly after, cannabis use (Criterion B). Intoxication typically begins with a “high” feeling followed by symptoms that include euphoria with inappropriate laughter and grandiosity, sedation, lethargy, impairment in short-term memory, difficulty carrying out complex mental processes, impaired judgment, distorted sensory perceptions, impaired motor performance, and the sensation that time is passing slowly. Occasionally, anxiety (which can be severe), dysphoria, or social withdrawal occurs. These psychoactive effects are accompanied by two or more of the following signs, developing within 2 hours of cannabis use: conjunctival injection, increased appetite, dry mouth, and tachycardia (Criterion C).

Intoxication develops within minutes if plant cannabis is smoked, and may take a few hours to develop when the cannabis is ingested orally. The effects usually last 3–4 hours, with duration longer when the substance is ingested orally. The magnitude of the behavioral and physiological changes depends on the dose, the method of administration, and the characteristics of the individual using the substance, such as rate of absorption, tolerance, and sensitivity to the effects of the substance. Because most cannabinoids, including delta-9-tetrahydrocannabinol (delta-9-THC), are fat soluble, the effects of cannabis or hashish may occasionally persist or reoccur for 12–24 hours because of the slow release of psychoactive substances from fatty tissue or to enterohepatic circulation.

Synthetic cannabinoids (e.g., Spice), whose use has become more common in recent years, also produce rapid effects, including euphoria, talkativeness, feelings of joy and laughter, and relaxation. In terms of psychoactive effects, low doses of synthetic cannabinoids and other cannabis products are similar. At higher doses of synthetic cannabinoids, delusional and hallucinatory symptoms are more likely to occur.

Prevalence

The prevalence of episodes of cannabis intoxication in the general population is unknown. However, it is probable that most individuals using cannabis would at some time experience symptoms that meet criteria for cannabis intoxication. Given this, the prevalence of individuals using cannabis and the prevalence of individuals experiencing cannabis intoxication are likely similar.

Functional Consequences of Cannabis Intoxication

Impairment from cannabis intoxication may have serious consequences, including dysfunction at work or school, social indiscretions, failure to fulfill role obligations, traffic accidents, and having unprotected sex. In rare cases, cannabis intoxication may precipitate a psychosis that may vary in duration.

Differential Diagnosis

Note that if the clinical presentation includes hallucinations in the absence of intact reality testing, a diagnosis of substance/medication-induced psychotic disorder should be considered.

Other substance intoxication.

Cannabis intoxication may resemble intoxication with other types of substances. However, in contrast to cannabis intoxication, alcohol intoxication and sedative, hypnotic, or anxiolytic intoxication frequently decrease appetite, increase aggressive behavior, and produce nystagmus or ataxia. Hallucinogens in low doses may cause a clinical picture that resembles cannabis intoxication. Phencyclidine, like cannabis, can be smoked and also causes perceptual changes, but phencyclidine intoxication is much more likely to cause ataxia and aggressive behavior.

Cannabis-induced mental disorders. Cannabis intoxication is distinguished from cannabis-induced mental disorders (e.g., cannabis-induced anxiety disorder, with onset during intoxication) because the symptoms (e.g., anxiety) in these latter disorders are in excess of those usually associated with cannabis intoxication, predominate in the clinical presentation, and are severe enough to warrant independent clinical attention.

Comorbidity

Given the typical overlap of cannabis intoxication with cannabis use disorder, see "Comorbidity" under Cannabis Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Cannabis Withdrawal

Diagnostic Criteria

A. Cessation of cannabis use that has been heavy and prolonged (i.e., usually daily or almost daily use over a period of at least a few months).

B. Three (or more) of the following signs and symptoms develop within approximately 1 week after Criterion A:

   1. Irritability, anger, or aggression.
   2. Nervousness or anxiety.
   3. Sleep difficulty (e.g., insomnia, disturbing dreams).
   4. Decreased appetite or weight loss.
   5. Restlessness.
   6. Depressed mood.
   7. At least one of the following physical symptoms causing significant discomfort: abdominal pain, shakiness/tremors, sweating, fever, chills, or headache.

C. The signs or symptoms in Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication or withdrawal from another substance.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid cannabis use disorder. If a mild cannabis use disorder is comorbid, the ICD-10-CM code is **F12.13**, and if a moderate or severe cannabis use disorder is comorbid, the ICD-10-CM code is **F12.23**. For cannabis withdrawal occurring in the absence of a cannabis use disorder (e.g., in a patient taking cannabis solely under appropriate medical supervision), the ICD-10-CM code is **F12.93**.

Diagnostic Features

The essential feature of cannabis withdrawal is the presence of a characteristic withdrawal syndrome that develops after the cessation of regular cannabis use. Regular users become tolerant to many acute cannabis effects, and cessation of regular use can lead to a cannabis withdrawal syndrome. Common cannabis withdrawal symptoms include irritability, depressed mood, anxiety, restlessness, sleep difficulty, and decreased appetite or weight loss. Cannabis withdrawal can cause significant distress, leading to continued use to relieve the symptoms, difficulty in quitting, and relapse. Unlike withdrawal from other substances (i.e., opioids, alcohol, sedatives), behavioral and emotional symptoms (e.g., nervousness, irritability, sleep difficulty) are often more common than physical symptoms (e.g., shakiness, sweating).

Associated Features

Cannabis withdrawal may be accompanied by observed fatigue, yawning, difficulty concentrating, and rebound periods of increased appetite and hypersomnia that follow initial periods of loss of appetite and insomnia.

Prevalence

Among adult and adolescent cannabis users, prevalence estimates of cannabis withdrawal symptoms vary widely, from 35% to 95%, based on research in the United States and other countries. Some of the variation in rates is likely attributable to assessment methods, and some to differences between samples. Among adult regular cannabis users in the general population, 12% reported signs and symptoms that met criteria for the full syndrome of DSM-5 cannabis withdrawal, with substantial differences in prevalence among non-Latinx Whites (10%), African Americans (15.3%), and Asian Americans, Native Hawaiians, and Pacific Islanders (31%). Among adults and adolescents who are enrolled in treatment or are heavy cannabis users, 50%–95% report cannabis withdrawal. These findings indicate that cannabis withdrawal occurs among a substantial subset of regular cannabis users who try to quit.

Development and Course

Withdrawal onset typically occurs within 24–48 hours after cessation of use. It peaks within 2–5 days and resolves within 1–2 weeks, although sleep disturbance can persist longer. The amount, duration, and frequency of cannabis smoking required to produce cannabis withdrawal are unknown, but more chronic and frequent cannabis use is associated with greater quantity and severity of withdrawal symptoms. Cannabis withdrawal can occur in adults and adolescents. Women may experience more severe cannabis withdrawal symptoms than men.

Risk and Prognostic Factors

Among cannabis users, the propensity to experience cannabis withdrawal is moderately heritable, indicating genetic influences. The prevalence and severity of cannabis withdrawal are greater among heavier cannabis users, particularly those seeking treatment for cannabis use disorder. Withdrawal severity may also be related to the presence and severity of comorbid symptoms of mental disorders.

Functional Consequences of Cannabis Withdrawal

Cannabis users report using cannabis to relieve withdrawal symptoms, making cannabis withdrawal a contributor to the persistence of cannabis use disorder. This

makes cannabis withdrawal a current target for medication development. Worse outcomes may be

associated with greater withdrawal. Sleep difficulty has been reported as the withdrawal symptom most often associated with relapse to cannabis use. Cannabis users report having relapsed to cannabis use or initiating use of other drugs (e.g., tranquilizers) to provide relief from cannabis withdrawal symptoms.

Differential Diagnosis

Because many of the symptoms of cannabis withdrawal are also symptoms of other substance withdrawal syndromes or of depressive or bipolar disorders, careful evaluation should focus on ensuring that the symptoms are not better explained by cessation of another substance (e.g., tobacco or alcohol withdrawal), another mental disorder (generalized anxiety disorder, major depressive disorder), or another medical condition. Given the increasingly common belief that cannabis use is harmless, regular cannabis users experiencing cannabis withdrawal may not realize that their withdrawal symptoms are due to the effects of cannabis wearing off, and continue to use cannabis as a form of self-medication.

Comorbidity

Among adult frequent cannabis users, cannabis withdrawal is associated with comorbid depression, anxiety, and antisocial personality disorder. Given the typical overlap of cannabis withdrawal with cannabis use disorder, see “Comorbidity” under Cannabis Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Cannabis-Induced Mental Disorders

The following cannabis-induced mental disorders are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): cannabis-induced psychotic disorder (“Schizophrenia Spectrum and Other Psychotic Disorders”); cannabis-induced anxiety disorder (“Anxiety Disorders”); and cannabis-induced sleep disorder (“Sleep-Wake Disorders”). For cannabis intoxication delirium and delirium induced by pharmaceutical cannabis receptor agonists taken as prescribed, see the criteria and discussion of delirium in the chapter “Neurocognitive Disorders.” These cannabis-induced mental disorders are diagnosed instead of cannabis intoxication or cannabis withdrawal when the symptoms are sufficiently severe to warrant independent clinical attention.

Unspecified Cannabis-Related Disorder

F12.99

This category applies to presentations in which symptoms characteristic of a cannabis-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific cannabis-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Hallucinogen-Related Disorders

Phencyclidine Use Disorder

Other Hallucinogen Use Disorder

Phencyclidine Intoxication

Other Hallucinogen Intoxication

Hallucinogen Persisting Perception Disorder

Phencyclidine-Induced Mental Disorders

Hallucinogen-Induced Mental Disorders

Unspecified Phencyclidine-Related Disorder

Unspecified Hallucinogen-Related Disorder

Phencyclidine Use Disorder

Diagnostic Criteria

A. A pattern of phencyclidine (or a pharmacologically similar substance) use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. Phencyclidine is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control phencyclidine use.
3. A great deal of time is spent in activities necessary to obtain phencyclidine, use the phencyclidine, or recover from its effects.
4. Craving, or a strong desire or urge to use phencyclidine.
5. Recurrent phencyclidine use resulting in a failure to fulfill major role obligations at work, school, or home (e.g., repeated absences from work or poor work performance related to phencyclidine use; phencyclidine-related absences, suspensions, or expulsions from school; neglect of children or household).
6. Continued phencyclidine use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the phencyclidine (e.g., arguments with a spouse about consequences of intoxication; physical fights).
7. Important social, occupational, or recreational activities are given up or reduced because of phencyclidine use.
8. Recurrent phencyclidine use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by a phencyclidine).
9. Phencyclidine use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is

likely to have been caused or exacerbated by the phencyclidine.

10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of the phencyclidine to achieve intoxication or desired effect.

    b. A markedly diminished effect with continued use of the same amount of the phencyclidine.

Note: Withdrawal symptoms and signs are not established for phencyclidines, and so this criterion does not apply. (Withdrawal from phencyclidines has been reported in animals but not documented in human users.)

Specify if:

In early remission: After full criteria for phencyclidine use disorder were previously met, none of the criteria for phencyclidine use disorder have been met for at least 3 months but for less than 12 months (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the phencyclidine,” may be met).

In sustained remission: After full criteria for phencyclidine use disorder were previously met, none of the criteria for phencyclidine use disorder have been met at any time during a period of 12 months or longer (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the phencyclidine,” may be met).

Specify if:

In a controlled environment: This additional specifier is used if the individual is in an environment where access to phencyclidines is restricted.

Code based on current severity/remission: If a phencyclidine intoxication or another phencyclidine-induced mental disorder is also present, do not use the codes below for phencyclidine use disorder. Instead, the comorbid phencyclidine use disorder is indicated in the 4th character of the phencyclidine-induced disorder code (see the coding note for phencyclidine intoxication or a specific phencyclidine-induced mental disorder). For example, if there is comorbid phencyclidine-induced psychotic disorder, only the phencyclidine-induced psychotic disorder code is given, with the 4th character indicating whether the comorbid phencyclidine use disorder is mild, moderate, or severe: F16.159 for mild phencyclidine use disorder with phencyclidine-induced psychotic disorder or F16.259 for a moderate or severe phencyclidine use disorder with phencyclidine-induced psychotic disorder.

Specify current severity/remission:

F16.10 Mild: Presence of 2–3 symptoms.

F16.11 Mild, In early remission

F16.11 Mild, In sustained remission

F16.20 Moderate: Presence of 4–5 symptoms.

F16.21 Moderate, In early remission

F16.21 Moderate, In sustained remission

F16.20 Severe: Presence of 6 or more symptoms.

F16.21 Severe, In early remission

F16.21 Severe, In sustained remission

Specifiers

"In a controlled environment" applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

Diagnostic Features

The phencyclidines (or phencyclidine-like substances) include phencyclidine (e.g., PCP, "angel dust") and less potent but similarly acting compounds such as ketamine,

cyclohexamine, and dizocilpine. These substances were first developed as dissociative anesthetics in the 1950s and became street drugs in the 1960s. They produce feelings of separation from mind and body (hence "dissociative") in low doses, and at high doses, stupor and coma can result. These substances are most commonly smoked or taken orally, but they may also be snorted or injected. Although the primary psychoactive effects of phencyclidine last for a few hours, the total elimination rate of this drug from the body typically extends 8 days or longer. The hallucinogenic effects in vulnerable individuals may last for weeks and may precipitate a persistent psychotic episode resembling schizophrenia. Ketamine has been observed to have utility in the treatment of major depressive disorder. Withdrawal symptoms have not been clearly established in humans, and therefore the withdrawal criterion is not included in the diagnosis of phencyclidine use disorder.

Associated Features

Phencyclidine may be detected in urine for up to 8 days or even longer at very high doses. In addition to laboratory tests to detect its presence, characteristic symptoms resulting from intoxication with phencyclidine or related substances may aid in its diagnosis. Phencyclidine is likely to produce dissociative symptoms, analgesia, nystagmus, risk of hypertension/hypotension and shock, euphoria, visual/auditory hallucinations, derealization, and unusual thought content. Violent behavior can also occur with phencyclidine use, as intoxicated individuals may believe that they are being attacked.

Prevalence

Data on the prevalence of phencyclidine use disorder are not available, but rates appear to be low (based on rates of the overall category of hallucinogen use disorder, which includes phencyclidine, of about 0.1% among individuals age 12 and older in the United States). Furthermore, among U.S. substance use treatment facility admissions, only 0.3% of the admitted individuals endorsed phencyclidine as their primary drug.

Risk and Prognostic Factors

In a general population study in Australia, ketamine users were more likely to be men and to have consumed more than 11 standard drinks per day.

Sex- and Gender-Related Diagnostic Issues

The gender ratio for phencyclidine use disorder is not known, but among U.S. substance use treatment facility admissions endorsing phencyclidine as the primary drug, 62% were men.

Diagnostic Markers

Laboratory testing may be useful, as phencyclidine is present in the urine in intoxicated individuals up to 8 days after ingestion. The individual's history along with certain physical signs (e.g., nystagmus, analgesia, prominent hypertension) may aid in distinguishing the phencyclidine clinical picture from that of other hallucinogens.

Functional Consequences of Phencyclidine Use Disorder

In individuals with phencyclidine use disorder, there may be physical evidence of injuries from accidents, fights, and falls. Chronic use of phencyclidine can lead to acute and persistent cognitive impairment; urinary tract and intestinal symptoms; abdominal pain, chest pain, palpitations, and tachycardia; respiratory depression; sleep disorders; and depression.

Differential Diagnosis

Other substance use disorders. Distinguishing the effects of phencyclidine from those of other substances may be important, because phencyclidine can be an additive to other substances (e.g., cannabis, cocaine).

Phencyclidine intoxication and phencyclidine-induced mental disorders. Phencyclidine use disorder is differentiated from phencyclidine intoxication and phencyclidine-induced mental disorders (e.g., phencyclidine-induced psychotic disorder) in that phencyclidine use disorder describes a problematic pattern of phencyclidine use that involves impaired control over phencyclidine use, social impairment attributable to phencyclidine use, risky phencyclidine use (e.g., driving while intoxicated), and pharmacological symptoms (the development of tolerance), whereas phencyclidine intoxication and phencyclidine-induced mental disorders describe psychiatric syndromes that occur in the context of heavy use. Phencyclidine intoxication and phencyclidine-induced mental disorders occur frequently in individuals with phencyclidine use disorder. In such cases, a diagnosis of phencyclidine intoxication or a phencyclidine-induced mental disorder should be given in addition to a diagnosis of phencyclidine use disorder, the presence of which is indicated in the diagnostic code.

Independent mental disorders. Some of the effects of phencyclidine use may resemble symptoms of independent mental disorders, such as psychosis (schizophrenia); low mood (major depressive disorder); and violent, aggressive behaviors (conduct disorder, antisocial personality disorder). Discerning whether these behaviors occurred before the intake of the drug is important in the differentiation of acute drug effects from a preexisting mental disorder.

Comorbidity

Conduct disorder in adolescents and antisocial personality disorder may be associated with phencyclidine use. Other substance use disorders, especially alcohol, cocaine, and amphetamine use disorders, are common among those with phencyclidine use disorder.

Other Hallucinogen Use Disorder

Diagnostic Criteria

A. A problematic pattern of hallucinogen (other than phencyclidine) use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. The hallucinogen is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control hallucinogen use.
3. A great deal of time is spent in activities necessary to obtain the hallucinogen, use the hallucinogen, or recover from its effects.
4. Craving, or a strong desire or urge to use the hallucinogen.
5. Recurrent hallucinogen use resulting in a failure to fulfill major role obligations at work, school, or home (e.g., repeated absences from work or poor work performance related to hallucinogen use; hallucinogen-related absences, suspensions, or expulsions from school; neglect of children or household).
6. Continued hallucinogen use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the hallucinogen (e.g., arguments with a spouse about consequences of intoxication; physical fights).

7. Important social, occupational, or recreational activities are given up or reduced because of hallucinogen use.
8. Recurrent hallucinogen use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by the hallucinogen).
9. Hallucinogen use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the hallucinogen.
10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of the hallucinogen to achieve intoxication or desired effect.
    b. A markedly diminished effect with continued use of the same amount of the hallucinogen.

Note: Withdrawal symptoms and signs are not established for hallucinogens, and so this criterion does not apply.

Specify **the particular hallucinogen.**

Specify if:

In early remission: After full criteria for other hallucinogen use disorder were previously met, none of the criteria for other hallucinogen use disorder have been met for at least 3 months but for less than 12 months (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the hallucinogen,” may be met).

In sustained remission: After full criteria for other hallucinogen use disorder were previously met, none of the criteria for other hallucinogen use disorder have been met at any time during a period of 12 months or

longer (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the hallucinogen,” may be met).

Specify if:

In a controlled environment: This additional specifier is used if the individual is in an environment where access to hallucinogens is restricted.

Code based on current severity/remission: If a hallucinogen intoxication or another hallucinogen-induced mental disorder is also present, do not use the codes below for hallucinogen use disorder. Instead, the comorbid hallucinogen use disorder is indicated in the 4th character of the hallucinogen-induced disorder code (see the coding note for hallucinogen intoxication or specific hallucinogen-induced mental disorder). For example, if there is comorbid hallucinogen-induced psychotic disorder and hallucinogen use disorder, only the hallucinogen-induced psychotic disorder code is given, with the 4th character indicating whether the comorbid hallucinogen use disorder is mild, moderate, or severe: F16.159 for mild hallucinogen use disorder with hallucinogen-induced psychotic disorder or F16.259 for a moderate or severe hallucinogen use disorder with hallucinogen-induced psychotic disorder.

Specify current severity/remission:

F16.10 Mild: Presence of 2–3 symptoms.

F16.11 Mild, In early remission

F16.11 Mild, In sustained remission

F16.20 Moderate: Presence of 4–5 symptoms.

F16.21 Moderate, In early remission

F16.21 Moderate, In sustained remission

F16.20 Severe: Presence of 6 or more symptoms.

F16.21 Severe, In early remission

F16.21 Severe, In sustained remission

Specifiers

“In a controlled environment” applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

Diagnostic Features

Hallucinogens comprise a diverse group of substances that despite having different chemical structures and possibly involving different molecular mechanisms, produce similar alterations of perception, mood, and cognition in users. Hallucinogens included are phenylalkylamines (e.g., mescaline, DOM [2,5-dimethoxy-4-methylamphetamine], and MDMA [3,4-methylenedioxymethamphetamine; also called “ecstasy” or “molly”]); the indoleamines, including psilocybin (and its metabolite psilocin, the compound primarily responsible for the psychedelic effects of hallucinogenic mushrooms) and dimethyltryptamine (DMT); and the ergolines, such as LSD (lysergic acid diethylamide) and morning glory seeds. In addition, miscellaneous other

ethnobotanical compounds are classified as hallucinogens, of which *Salvia divinorum* and jimsonweed are two examples. Excluded from the hallucinogen group are cannabis and its active compound, delta-9-tetrahydrocannabinol (THC) (see the section "Cannabis-Related Disorders"). These substances can have hallucinogenic effects but are diagnosed separately because of significant differences in their psychological and behavioral effects.

Hallucinogens are usually taken orally, although some forms are smoked (e.g., DMT, salvia) or (rarely) taken intranasally or by injection (e.g., ecstasy). Duration of effects varies across types of hallucinogens. Some of these substances (i.e., LSD, MDMA) have a long half-life and extended duration such that users may spend hours to days using and/or recovering from the effects of these drugs. However, other hallucinogenic drugs (e.g., DMT, salvia) are short acting. Tolerance to hallucinogens develops with repeated use and has been reported to have both autonomic and psychological effects.

MDMA/ecstasy as a hallucinogen may have distinctive effects attributable to both its hallucinogenic and its stimulant properties. Ecstasy users have a higher risk of developing a hallucinogen use disorder than those using other hallucinogens. Among both adolescent and adult ecstasy users and users of other hallucinogens, the most frequently reported hallucinogen use disorder criteria are tolerance, hazardous use, use despite emotional or health problems, giving up activities in favor of use, and spending a lot of time obtaining, using, or recovering from the effects of use. As found for other substances, diagnostic criteria for other hallucinogen use disorder are arrayed along a single continuum of severity.

Given that a clinically significant withdrawal syndrome has not been consistently documented in humans, the diagnosis of hallucinogen withdrawal syndrome is not included in this manual and therefore is not part of the hallucinogen use disorder diagnostic criteria. However, there may be evidence of withdrawal from MDMA, with endorsement of any two or more withdrawal symptoms (e.g., malaise, appetite disturbance, mood changes [anxious, depressed, irritable], poor concentration, sleep disruption) or withdrawal avoidance observed in more than half of individuals in diverse samples of ecstasy users in the United States and internationally.

Associated Features

The characteristic symptom features of use of some hallucinogens can aid in diagnosis if urine or blood toxicology results are not available. For example, individuals who use LSD tend to experience visual hallucinations that can be frightening.

Prevalence

Other hallucinogen use disorder is rare. In the U.S. general population, about 0.1% of individuals age 12 or older endorsed the symptoms of past 12-month hallucinogen use disorder in 2018. The rate was 0.2% among those ages 12–17, 0.4% among those ages 18–25,

and < 0.1% among those age 26 and older. Prevalence is higher in U.S. clinical samples (e.g., 19% in adolescents in treatment), and among select groups of individuals who use hallucinogens frequently (e.g., recent heavy ecstasy use) in the United States and Australia, 73.5% of adults and 77% of adolescents have a problematic pattern of use that may meet other hallucinogen use disorder criteria.

Development and Course

Prevalence of other hallucinogen use disorder by age among adolescents is unknown. Among U.S. adults age 18 years and older, most (90%) of those with other hallucinogen use disorder are ages 18–29, suggesting that the disorder is not often persistent and is concentrated in young adults.

Risk and Prognostic Factors

Temperamental. The use of specific hallucinogens (i.e., ecstasy, salvia) has been linked with high sensation-seeking.

Environmental. On the basis of research in the United States, environmental risk factors of other hallucinogen use disorder include higher income, lower education, being never married, and residing in urban areas. Early onset of hallucinogen use has also been linked to transition to hallucinogen use disorder. Peer use of other drugs is also highly associated with ecstasy and salvia use.

Genetic and physiological. Among male twins, total variance due to additive genetics has been estimated to range from 26% to 79%, with inconsistent evidence for shared environmental influences.

Culture-Related Diagnostic Issues

Historically, hallucinogens have been used as part of established religious or spiritual practices, such as the use of peyote in the Native American Church and in Mexico. Ritual use by Indigenous populations of psilocybin obtained from certain types of mushrooms has occurred in South America, Mexico, and some areas in the United States, or of ayahuasca in the Santo Daime and União de Vegetal religious groups.

Sex- and Gender-Related Diagnostic Issues

Among U.S. adolescents, boys have greater 12-month prevalence rates of other hallucinogen use than girls, and these gender differences extend to specific hallucinogens, including LSD, MDMA, psilocybin, and salvia divinorum. Among U.S. adults, 60% of individuals with other hallucinogen use disorder are men. International research suggests that women administered MDMA may have greater subjective effects, such as altered state of consciousness, anxiety, and depression. No information from international studies is available regarding gender differences for other hallucinogen use disorder.

Diagnostic Markers

Laboratory testing can be useful in distinguishing among the different hallucinogens. However, because some agents (e.g., LSD) are so potent that as little as 75 micrograms can produce severe reactions, typical toxicological examination will not always reveal which substance has been used.

Functional Consequences of Other Hallucinogen Use Disorder

Although insufficient information exists to clearly note the functional consequences of other hallucinogen use disorder, complications of use of these substances have been

identified. Adverse effects of other hallucinogen use include those related to intoxication, such as hyperthermia, cardiac tachyarrhythmias, pneumothorax hypernatremia, motor incoordination, nystagmus, restlessness, hallucinations/delusions, mydriasis, increased alertness, and high blood pressure. Other more serious reactions related to consequences of repeated use of other hallucinogens

include renal failure, hepatic failure, seizures, cerebral infarction, rhabdomyolysis, cardiac complications, and hepatotoxicity.

There is evidence for persisting neurotoxic effects of MDMA/ecstasy use, including impairments in memory, psychological function, and neuroendocrine function; serotonin system dysfunction; and sleep disturbance; as well as adverse effects on brain microvasculature, white matter maturation, and damage to axons.

Differential Diagnosis

Other substance disorders. The effects of hallucinogen use must be distinguished from those of other substances (e.g., amphetamine use disorder, alcohol or sedative withdrawal), especially because contamination of the hallucinogens with other drugs is relatively common.

Hallucinogen intoxication and hallucinogen-induced mental disorders. Hallucinogen use disorder is differentiated from hallucinogen intoxication and hallucinogen-induced mental disorders (e.g., hallucinogen-induced psychotic disorder) in that hallucinogen use disorder describes a problematic pattern of hallucinogen use that involves impaired control over hallucinogen use, social impairment attributable to hallucinogen use, risky hallucinogen use (e.g., driving while intoxicated), and pharmacological symptoms (the development of tolerance), whereas hallucinogen intoxication and hallucinogen-induced mental disorders describe psychiatric syndromes that occur in the context of heavy use. Hallucinogen intoxication and hallucinogen-induced mental disorders occur frequently in individuals with hallucinogen use disorder. In such cases, a diagnosis of hallucinogen intoxication or a hallucinogen-induced mental disorder should be given in addition to a diagnosis of hallucinogen use disorder, the presence of which is indicated in the diagnostic code.

Independent mental disorders. Some of the effects of hallucinogen use may resemble symptoms of independent psychiatric disorders, such as schizophrenia and depressive and bipolar disorders. Discerning whether symptoms occurred before the intake of the drug is important in the differentiation of acute drug effects from a preexisting mental disorder. In particular, schizophrenia should be ruled out, as some affected individuals (e.g., individuals with schizophrenia who exhibit paranoia) may falsely attribute their symptoms to use of hallucinogens.

Comorbidity

Other hallucinogen use disorder is highly associated with cocaine use disorder, stimulant use disorder, other substance use disorder, tobacco (nicotine) use disorder, any personality disorder, posttraumatic stress disorder, and panic attacks.

Phencyclidine Intoxication

Diagnostic Criteria

A. Recent use of phencyclidine (or a pharmacologically similar substance).

B. Clinically significant problematic behavioral changes (e.g., belligerence, assaultiveness, impulsiveness, unpredictability, psychomotor agitation, impaired judgment) that developed during, or shortly after, phencyclidine use.

C. Within 1 hour, two (or more) of the following signs or symptoms:

Note: When the drug is smoked, "snorted," or used intravenously, the onset may be particularly rapid.

1. Vertical or horizontal nystagmus.
2. Hypertension or tachycardia.
3. Numbness or diminished responsiveness to pain.
4. Ataxia.
5. Dysarthria.
6. Muscle rigidity.
7. Seizures or coma.
8. Hyperacusis.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Coding note: The ICD-10-CM code depends on whether there is a comorbid phencyclidine use disorder. If a mild phencyclidine use disorder is comorbid, the ICD-10-CM code is **F16.120,** and if a moderate or severe phencyclidine use disorder is comorbid, the ICD-10-CM code is **F16.220.** If there is no comorbid phencyclidine use disorder, then the ICD-10-CM code is **F16.920.**

Note: In addition to the section "Functional Consequences of Phencyclidine Intoxication," see the corresponding section in Phencyclidine Use Disorder.

Diagnostic Features

Phencyclidine intoxication reflects the clinically significant behavioral changes that occur shortly after ingestion of this substance (or a pharmacologically similar substance). The most common clinical presentations of phencyclidine intoxication include disorientation; confusion without hallucinations; nystagmus; numbness or diminished responsiveness to pain; ataxia; dysarthria; muscle rigidity; hyperacusis; and coma of varying severity. Other clinically significant behavioral changes associated with phencyclidine intoxication include violent behavior, extreme agitation, persecutory delusions, euphoria, retrograde amnesia, and hypertension.

Prevalence

Use of phencyclidine or related substances (e.g., ketamine) may be taken as an estimate of the prevalence of intoxication. Phencyclidine use is rare, with < 0.1% of the U.S. population age 12 and older reporting past 12-month use in 2018. In surveys of U.S. students and young adults followed up from high school, past 12-month prevalence of ketamine use, which is assessed separately from other substances, was estimated at about 1.2% among 12th graders and 0.5% among young adults, ages 19–28 years.

Diagnostic Markers

Laboratory testing may be useful, as phencyclidine is detectable in urine for up to 8 days following use, although the levels are only weakly associated with an individual's clinical presentation and may therefore not be useful for case management. Creatine phosphokinase and aspartate aminotransferase levels may be elevated.

Functional Consequences of Phencyclidine Intoxication

Phencyclidine intoxication produces extensive cardiovascular and neurological (e.g., seizures, dystonias, dyskinesias, catalepsy, hypothermia or hyperthermia) toxicity.

Differential Diagnosis

In particular, in the absence of intact reality testing (i.e., without insight that the perceptual abnormalities are drug induced), an additional diagnosis of phencyclidine-induced psychotic disorder should be considered.

Other substance intoxication. Phencyclidine intoxication should be differentiated from intoxication due to other substances, including other hallucinogens; amphetamine, cocaine, or other stimulants; and anticholinergics, as well as withdrawal from benzodiazepines. Nystagmus and bizarre and violent behavior may distinguish intoxication due to phencyclidine from that due to other substances. Toxicological tests may be useful in making this distinction. However, the weak correlation between quantitative toxicology levels of phencyclidine and clinical presentation may diminish the utility of the laboratory findings for patient management.

Phencyclidine-induced mental disorders. Phencyclidine intoxication is distinguished from phencyclidine-induced mental disorders (e.g., phencyclidine-induced depressive disorder, with onset during intoxication) because the symptoms (e.g., depressed mood) in the latter disorders are in excess of those usually associated with phencyclidine intoxication, predominate in the clinical presentation, and are severe enough to warrant clinical attention.

Other medical conditions. Medical conditions to be considered include certain metabolic disorders like hypoglycemia and hyponatremia, central nervous system tumors, seizure disorders, sepsis, neuroleptic malignant syndrome, and vascular insults.

Comorbidity

Given the typical overlap of phencyclidine intoxication with phencyclidine use disorder, see “Comorbidity” under Phencyclidine Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Other Hallucinogen Intoxication

Diagnostic Criteria

A. Recent use of a hallucinogen (other than phencyclidine).
B. Clinically significant problematic behavioral or psychological changes (e.g., marked anxiety or depression, ideas of reference, fear of “losing one’s mind,” paranoid ideation, impaired judgment) that developed during, or shortly after, hallucinogen use.
C. Perceptual changes occurring in a state of full wakefulness and alertness (e.g., subjective intensification of perceptions, depersonalization, derealization, illusions, hallucinations, synesthesias) that developed during, or shortly after, hallucinogen use.
D. Two (or more) of the following signs developing during, or shortly after, hallucinogen use:
    1. Pupillary dilation.
    2. Tachycardia.

3. Sweating.
4. Palpitations.
5. Blurring of vision.
6. Tremors.
7. Incoordination.

E. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Coding note: The ICD-10-CM code depends on whether there is a comorbid hallucinogen use disorder. If a mild hallucinogen use disorder is comorbid, the ICD-10-CM code is **F16.120,** and if a moderate or severe hallucinogen use disorder is comorbid, the ICD-10-CM code is **F16.220.** If there is no comorbid hallucinogen use disorder, then the ICD-10-CM code is **F16.920.**

Note: For information on Associated Features and Culture-Related Diagnostic Issues, see the corresponding sections in Other Hallucinogen Use Disorder.

Diagnostic Features

Other hallucinogen intoxication reflects the clinically significant behavioral or psychological changes that occur shortly after ingestion of a hallucinogen. Depending on the specific hallucinogen, the intoxication may last only minutes (e.g., for salvia) or several hours or longer (e.g., for LSD [lysergic acid diethylamide] or MDMA [3,4-methylenedioxymethamphetamine]).

Prevalence

The prevalence of other hallucinogen intoxication is not fully known but may be approximated based on the prevalence of use of the substances. In 2018, 1.5% of individuals ages 12–17 years in the United States reported use of hallucinogens in the past year; among individuals ages 18–25, the rate was 6.9%, and among those age 26 or older, the rate was 1.3%. Rates were consistently higher for boys and men than for girls and women in every age group.

Association With Suicidal Thoughts or Behavior

Other hallucinogen intoxication may lead to increased suicidal thoughts or behavior, although suicide is rare among individuals who use hallucinogens. Of note, a study of more than 135,000 randomly selected U.S. adults, including more than 19,000 individuals who use psychedelics, did not find evidence, after adjustment for sociodemographics, other drug use, and childhood depression, that lifetime psychedelic use is an independent risk factor for mental health problems, suicidal thoughts, or suicide attempts. In addition, one large U.S. population survey found that a lifetime history of hallucinogen use was associated with lower odds of mental distress and suicidal thoughts or behavior, although a causal relationship between hallucinogenic drugs and lower distress cannot be inferred from this study. On the basis of these findings, the relationship of other hallucinogen use to suicidal thoughts and behaviors is uncertain.

Functional Consequences of Other Hallucinogen Intoxication

Other hallucinogen intoxication can have serious consequences. The perceptual disturbances and impaired judgment associated with other hallucinogen intoxication can result in injuries or fatalities from automobile crashes, physical fights, or unintentional self-injury (e.g., cuts or falls from impaired depth perception). When other hallucinogens are consumed in combination with other drugs (including alcohol), coma can occur, with the duration and profundity of coma greater than when other hallucinogens are taken alone. Continued use of hallucinogens, particularly MDMA, has also been linked with neurotoxic effects. Adverse effects of other hallucinogen use include hyperthermia, cardiac tachyarrhythmias, pneumothorax hypernatremia, motor incoordination, nystagmus, restlessness, hallucinations/delusions, mydriasis, increased alertness, and high blood pressure. More serious reactions include renal failure, hepatic failure, seizures, cerebral infarction, rhabdomyolysis, cardiac complications, and hepatotoxicity.

Differential Diagnosis

Other substance intoxication. Other hallucinogen intoxication should be differentiated from intoxication with amphetamine-type substances, cocaine, or other stimulants; anticholinergics, inhalants, and phencyclidine. Toxicological tests are useful in making this distinction, and determining the route of administration may also be useful.

Other conditions. Other disorders and conditions to be considered include schizophrenia, depression, withdrawal from other drugs (e.g., sedatives, alcohol), certain metabolic disorders (e.g., hypoglycemia), seizure disorders, tumors of the central nervous system, and vascular insults.

Hallucinogen persisting perception disorder. Other hallucinogen intoxication is distinguished from hallucinogen persisting perception disorder because the symptoms in the latter continue episodically or continuously for weeks (or longer) after the most recent intoxication.

Hallucinogen-induced mental disorders. Other hallucinogen intoxication is distinguished from hallucinogen-induced mental disorders (e.g., hallucinogen-induced anxiety disorder, with onset during intoxication) because the symptoms (e.g., anxiety) in these latter disorders are in excess of those usually associated with other hallucinogen intoxication, predominate in the clinical presentation, and are severe enough to warrant independent clinical attention.

Comorbidity

Given the typical overlap of other hallucinogen intoxication with other hallucinogen use disorder, see “Comorbidity” under Other Hallucinogen Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Hallucinogen Persisting Perception Disorder

Diagnostic Criteria — F16.983

A. Following cessation of use of a hallucinogen, the reexperiencing of one or more of the perceptual symptoms that were experienced while intoxicated with the hallucinogen (e.g., geometric hallucinations, false perceptions of movement in the peripheral visual fields, flashes of color,

intensified colors, trails of images of moving objects, positive afterimages, halos around objects, macropsia and micropsia).

B. The symptoms in Criterion A cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The symptoms are not attributable to another medical condition (e.g., anatomical lesions and infections of the brain, visual epilepsies) and are not better explained by another mental disorder (e.g., delirium, major neurocognitive disorder, schizophrenia) or hypnopompic hallucinations.

Diagnostic Features

The hallmark of hallucinogen persisting perception disorder is the reexperiencing, when the individual is sober, of the perceptual disturbances that were experienced while the individual was intoxicated with the hallucinogen (Criterion A). The symptoms may include any perceptual perturbations, but visual disturbances tend to be predominant. Typical of the abnormal visual perceptions are geometric hallucinations, false perceptions of

movement in the peripheral visual fields, flashes of color, intensified colors, trails of images of moving objects (i.e., images left suspended in the path of a moving object as seen in stroboscopic photography), perceptions of entire objects, visual snow, positive afterimages (i.e., a same-colored or complementary-colored "shadow" of an object remaining after removal of the object), halos around objects, or misperception of images as too large (macropsia) or too small (micropsia). Duration of the visual disturbances may be episodic or nearly continuous and must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion B). The disturbances may last for weeks, months, or years. Other explanations for the disturbances (e.g., brain lesions, preexisting psychosis, seizure disorders, migraine aura without headaches) must be ruled out (Criterion C).

Hallucinogen persisting perception disorder occurs primarily after LSD (lysergic acid diethylamide) use, but not exclusively. There does not appear to be a strong correlation between hallucinogen persisting perception disorder and number of occasions of hallucinogen use, with some instances of hallucinogen persisting perception disorder occurring in individuals with minimal exposure to hallucinogens. Some instances of hallucinogen persisting perception disorder may be triggered by use of other substances (e.g., cannabis or alcohol), adaptation to dark environments, exercise, and exposure to noise and photophobia.

Associated Features

Reality testing remains intact in individuals with hallucinogen persisting perception disorder (i.e., the individual is aware that the disturbance is linked to the effect of the drug). If this is not the case, another disorder might better explain the abnormal perceptions.

Prevalence

Prevalence estimates of hallucinogen persisting perception disorder are unknown. Initial prevalence estimates of the disorder among individuals who use hallucinogens is approximately 4.2%.

Development and Course

Little is known about the development of hallucinogen persisting perception disorder. Its course, as suggested by its name, is persistent, lasting for weeks, months, or even years in certain individuals.

Risk and Prognostic Factors

There is little evidence regarding risk factors for hallucinogen persisting perception disorder, although genetic factors have been suggested as a possible explanation underlying the susceptibility to LSD effects in this condition.

Functional Consequences of Hallucinogen Persisting Perception Disorder

Although hallucinogen persisting perception disorder remains a chronic condition in some cases, many individuals with the disorder are able to suppress the disturbances and continue to function normally.

Differential Diagnosis

Conditions to be ruled out include schizophrenia, other drug effects, neurodegenerative disorders, stroke, brain tumors, infections, and head trauma. Neuroimaging results in hallucinogen persisting perception disorder cases are typically negative. As noted earlier, reality testing remains intact (i.e., the individual is aware that the disturbance is linked to the

effect of the drug); if this is not the case, another disorder (e.g., psychotic disorder, another medical condition) might better explain the abnormal perceptions.

Comorbidity

Common comorbid mental disorders accompanying hallucinogen persisting perception disorder are panic disorder, alcohol use disorder, major depressive disorder, bipolar I disorder, and schizophrenia spectrum disorders.

Phencyclidine-Induced Mental Disorders

Other phencyclidine-induced mental disorders are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): phencyclidine-induced psychotic disorder (“Schizophrenia Spectrum and Other Psychotic Disorders”); phencyclidine-induced bipolar and related disorder (“Bipolar and Related Disorders”); phencyclidine-induced depressive disorder (“Depressive Disorders”); and phencyclidine-induced anxiety disorder (“Anxiety Disorders”). For phencyclidine-induced intoxication delirium and delirium induced by ketamine taken as prescribed, see the criteria and discussion of delirium in the chapter “Neurocognitive Disorders.” These phencyclidine-induced mental disorders are diagnosed instead of phencyclidine intoxication only when the symptoms are sufficiently severe to warrant independent clinical attention.

Hallucinogen-Induced Mental Disorders

The following other hallucinogen-induced mental disorders are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): other hallucinogen–

induced psychotic disorder (“Schizophrenia Spectrum and Other Psychotic Disorders”); other hallucinogen–induced bipolar and related disorder (“Bipolar and Related Disorders”); other hallucinogen–induced depressive disorder (“Depressive Disorders”); and other hallucinogen–induced anxiety disorder (“Anxiety Disorders”). For other hallucinogen intoxication delirium and delirium induced by other hallucinogens taken as prescribed, see the criteria and discussion of delirium in the chapter “Neurocognitive Disorders.” These hallucinogen-induced mental disorders are diagnosed instead of other hallucinogen intoxication only when the symptoms are sufficiently severe to warrant independent clinical attention.

Unspecified Phencyclidine-Related Disorder

F16.99

This category applies to presentations in which symptoms characteristic of a phencyclidine-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific phencyclidine-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Unspecified Hallucinogen-Related Disorder

F16.99

This category applies to presentations in which symptoms characteristic of a hallucinogen-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific hallucinogen-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Inhalant-Related Disorders

Inhalant Use Disorder

Inhalant Intoxication

Inhalant-Induced Mental Disorders

Unspecified Inhalant-Related Disorder

Inhalant Use Disorder

Diagnostic Criteria

A. A problematic pattern of use of a hydrocarbon-based inhalant substance leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. The inhalant substance is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control use of the inhalant substance.
3. A great deal of time is spent in activities necessary to obtain the inhalant substance, use it, or recover from its effects.
4. Craving, or a strong desire or urge to use the inhalant substance.
5. Recurrent use of the inhalant substance resulting in a failure to fulfill major role obligations at work, school, or home.
6. Continued use of the inhalant substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use.
7. Important social, occupational, or recreational activities are given up or reduced because of use of the inhalant substance.
8. Recurrent use of the inhalant substance in situations in which it is physically hazardous.
9. Use of the inhalant substance is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.
10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of the inhalant substance to achieve intoxication or desired effect.
    b. A markedly diminished effect with continued use of the same amount of the inhalant substance.

Specify **the particular inhalant:** When possible, the particular substance involved should be named (e.g., “solvent use disorder”).

Specify if:

In early remission: After full criteria for inhalant use disorder were previously met, none of the criteria for inhalant use disorder have been met for at least 3 months but for less than 12 months (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the inhalant substance,” may be met).

In sustained remission: After full criteria for inhalant use disorder were previously met, none of the criteria for inhalant use disorder have been met at any time during a period of 12 months or longer (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the inhalant substance,” may be met).

Specify if:

In a controlled environment: This additional specifier is used if the individual is in an environment where access to inhalant substances is restricted.

Code based on current severity/remission: If an inhalant intoxication or another inhalant-induced mental disorder is also present, do not use the

codes below for inhalant use disorder. Instead, the comorbid inhalant use disorder is indicated in the 4th character of the inhalant-induced disorder code (see the coding note for inhalant intoxication or a specific inhalant-induced mental disorder). For example, if there is comorbid inhalant-induced depressive disorder and inhalant use disorder, only the inhalant-induced depressive disorder code is given, with the 4th character indicating whether the comorbid inhalant use disorder is mild, moderate, or severe: F18.14 for mild inhalant use disorder with inhalant-induced depressive disorder or F18.24 for a moderate or severe inhalant use disorder with inhalant-induced depressive disorder.

Specify current severity/remission:

F18.10 Mild: Presence of 2–3 symptoms.

F18.11 Mild, In early remission

F18.11 Mild, In sustained remission

F18.20 Moderate: Presence of 4–5 symptoms.

F18.21 Moderate, In early remission

F18.21 Moderate, In sustained remission

F18.20 Severe: Presence of 6 or more symptoms.

F18.21 Severe, In early remission

F18.21 Severe, In sustained remission

Specifiers

"In a controlled environment" applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

The severity of individuals' inhalant use disorder is assessed by the number of diagnostic criteria endorsed. Changing severity of individuals' inhalant use disorder across time is reflected by reductions in the frequency (e.g., days used per month) and/or dose (e.g., tubes of glue per day) used, as assessed by the individual's self-report, report of others, clinician's observations, and biological testing (when practical).

Diagnostic Features

Examples of inhalant substances include volatile hydrocarbons, which comprise toxic gases from glues, fuels, paints, and other volatile compounds. When possible, the

particular substance involved should be named (e.g., "toluene use disorder"). However, most compounds that are inhaled are a mixture of several substances that can produce psychoactive effects, and it is often difficult to ascertain the exact substance responsible for the disorder. Unless there is clear evidence that a single, unmixed substance has been used, the general term *inhalant* should be used in recording the diagnosis. Disorders arising from inhalation of nitrous oxide or of amyl-, butyl-, or isobutylnitrite are considered as other (or unknown) substance use disorder.

Features of inhalant use disorder include repeated use of an inhalant substance despite the individual's knowing that the substance is causing serious problems for the individual (Criterion A9). Those problems are reflected in the diagnostic criteria.

Missing work or school or inability to perform typical responsibilities at work or school (Criterion A5), and continued use of the inhalant substance even though it causes arguments with family or friends, fights, and other social or interpersonal problems (Criterion A6), may be seen in inhalant use disorder. Limiting family contact, work or school obligations, or recreational activities (e.g., sports, games, hobbies) may also occur (Criterion A7). Use of inhalants when driving or operating dangerous equipment (Criterion A8) is also seen.

Tolerance (Criterion A10) is reported by about 10% of individuals who use inhalants. Because a clinically significant withdrawal syndrome has not been established with inhalant use, neither a diagnosis of inhalant withdrawal nor a corresponding diagnostic criterion for withdrawal complaints for inhalant use disorder is included. However, withdrawal symptoms may occur among inhalant users and individuals with moderate to severe inhalant use disorder, and these symptoms appear to be similar in frequency to withdrawal symptoms among those with moderate to severe cocaine use disorder.

Associated Features

A diagnosis of inhalant use disorder is supported by recurring episodes of intoxication with negative results in standard drug screens (which do not detect inhalants); possession, or lingering odors, of inhalant substances; peri-oral or peri-nasal “glue-sniffer’s rash”; association with other individuals known to use inhalants; membership in groups with prevalent inhalant use (e.g., some native or aboriginal communities, homeless children in street gangs); easy access to certain inhalant substances; paraphernalia possession; presence of the disorder’s characteristic medical complications (e.g., brain white matter pathology, rhabdomyolysis); and the presence of multiple other substance use disorders. Individuals with inhalant use disorder may present with symptoms of pernicious anemia, subacute combined degeneration of the spinal cord, major or mild neurocognitive disorder, brain atrophy, leukoencephalopathy, and many other nervous system disorders.

Prevalence

About 2.3% of American youth ages 12–17 years have used inhalants in the past 12 months, with 0.1% having a pattern of use that meets criteria for inhalant use disorder. Among U.S. adults, age 18 years and older, past 12-month prevalence of inhalant use is about 0.21%, with 0.04% having a pattern of use that meets criteria for an inhalant use disorder. Among youth, the prevalence of past 12-month inhalant use is highest among non-Hispanic Whites and individuals reporting more than one racialized identity and lowest among American Indians/Alaska Natives. Twelve-month prevalence rates of inhalant use and inhalant use disorder among adults are highest among non-Hispanic Whites and lowest among non-Hispanic Blacks and American Indians/Alaska Natives.

Development and Course

The declining prevalence in the United States of inhalant use and inhalant use disorder after adolescence (from 2.3% during adolescence to 0.1% in early adulthood for inhalant use and from 0.1% to 0.04% for inhalant use disorder) indicates that the disorder usually

remits in early adulthood. Inhalant use disorder is rare in prepubertal children, most common in adolescents and young adults, and uncommon in older persons. Calls to poison-control centers for “intentional abuse” of inhalants peak with calls involving

individuals at age 14 years. Those with inhalant use disorder extending into adulthood demonstrate earlier onset of inhalant use, use of multiple inhalants, and more frequent inhalant use.

Risk and Prognostic Factors

Temperamental. Predictors of inhalant use disorder include sensation seeking and impulsivity.

Environmental. Inhalant gases are widely and legally available, increasing the risk of misuse. Childhood maltreatment or trauma also is associated with youthful progression from inhalant non-use to inhalant use disorder.

Genetic and physiological. *Behavioral disinhibition* is a highly heritable general propensity to not constrain behavior in socially acceptable ways, to break social norms and rules, and to take dangerous risks, pursuing rewards excessively despite dangers of adverse consequences. Youths with strong behavioral disinhibition show risk factors for inhalant use disorder: early-onset substance use disorder, multiple substance involvement, and early conduct problems. Because behavioral disinhibition is under strong genetic influence, youths in families with substance use and antisocial behaviors are at elevated risk for inhalant use disorder.

Culture-Related Diagnostic Issues

Internationally, certain isolated Indigenous communities have experienced a high prevalence of inhalant problems. Also, in some low- and middle-income countries, groups of homeless children living on the streets have extensive inhalant use problems because of the effects of poverty and the availability and affordability of the substances, and as a way to cope with homelessness.

Sex- and Gender-Related Diagnostic Issues

Although the past 12-month prevalence of inhalant use disorder in the United States is almost identical among adolescent boys and girls, the disorder is very rare among adult women.

Diagnostic Markers

Urine, breath, or saliva tests may be valuable for assessing concurrent use of non-inhalant substances by individuals with inhalant use disorder. However, technical problems and the considerable expense of analyses make frequent biological testing for inhalants themselves impractical.

Association With Suicidal Thoughts or Behavior

In the United States, adolescent and adult inhalant use and inhalant use disorder are associated with suicidal thoughts and behavior, especially among individuals reporting symptoms of anxiety and depression and histories of trauma.

Functional Consequences of Inhalant Use Disorder

Because of inherent toxicity, use of inhalants can be fatal. Death can occur from anoxia, cardiac dysfunction, extreme allergic reaction, severe injury to the lungs, vomiting, accidents or injury, or central nervous system depression. Moreover, any inhaled volatile hydrocarbons may produce “sudden sniffing death” from cardiac arrhythmia. Inhalant use impairs neurobehavioral function and causes various neurological, gastrointestinal, cardiovascular, and pulmonary problems.

Long-term inhalant users are at increased risk for tuberculosis, HIV/AIDS, sexually transmitted diseases, depression, anxiety, bronchitis, asthma, and sinusitis.

Differential Diagnosis

Inhalant exposure (unintentional) from industrial or other accidents. A diagnosis of inhalant use disorder only applies if the inhalant exposure is intentional.

Inhalant intoxication, without meeting criteria for inhalant use disorder. Inhalant intoxication occurs frequently during inhalant use disorder but also may occur among individuals whose use does not meet criteria for inhalant use disorder.

Inhalant intoxication meeting criteria for inhalant use disorder, and inhalant-induced mental disorders. Inhalant use disorder is differentiated from inhalant intoxication and inhalant-induced mental disorders (e.g., inhalant-induced depressive disorder) in that inhalant use disorder describes a problematic pattern of inhalant use that involves impaired control over inhalant use, social impairment attributable to inhalant use, risky inhalant use (e.g., inhalant use despite medical complications), and pharmacological symptoms (the development of tolerance), whereas inhalant intoxication and inhalant-induced mental disorders describe psychiatric syndromes that develop in the context of heavy use. Inhalant intoxication and inhalant-induced mental disorders occur frequently in individuals with inhalant use disorder. In such cases, a diagnosis of inhalant intoxication or an inhalant-induced mental disorder should be given in addition to a diagnosis of inhalant use disorder, the presence of which is indicated in the diagnostic code.

Other substance use disorders, especially those involving sedating substances (e.g., alcohol, benzodiazepines, barbiturates). Inhalant use disorder commonly co-occurs with other substance use disorders, and the symptoms of the disorders may be similar and overlapping. To disentangle symptom patterns, it is helpful to inquire about which symptoms persisted during periods when some of the substances were not being used.

Comorbidity

Individuals with inhalant use disorder receiving clinical care often have numerous other substance use, mood, anxiety, and personality disorders. Inhalant use disorder commonly co-occurs with conduct disorder in adolescents and with antisocial personality disorder. Individuals with inhalant use disorder may have comorbid symptoms of hepatic or renal damage, rhabdomyolysis, methemoglobinemia, or symptoms of other gastrointestinal, cardiovascular, or pulmonary diseases.

Inhalant Intoxication

Diagnostic Criteria

A. Recent intended or unintended short-term, high-dose exposure to inhalant substances, including volatile hydrocarbons such as toluene or gasoline.

B. Clinically significant problematic behavioral or psychological changes (e.g., belligerence, assaultiveness, apathy, impaired judgment) that developed during, or shortly after, exposure to inhalants.

C. Two (or more) of the following signs or symptoms developing during, or shortly after, inhalant use or exposure:

   1. Dizziness.

2. Nystagmus.
3. Incoordination.

4. Slurred speech.
5. Unsteady gait.
6. Lethargy.
7. Depressed reflexes.
8. Psychomotor retardation.
9. Tremor.
10. Generalized muscle weakness.
11. Blurred vision or diplopia.
12. Stupor or coma.
13. Euphoria.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Coding note: The ICD-10-CM code depends on whether there is a comorbid inhalant use disorder. If a mild inhalant use disorder is comorbid, the ICD-10-CM code is **F18.120,** and if a moderate or severe inhalant use disorder is comorbid, the ICD-10-CM code is **F18.220.** If there is no comorbid inhalant use disorder, then the ICD-10-CM code is **F18.920.**

Note: For information on Development and Course, Risk and Prognostic Factors, Culture-Related Diagnostic Issues, and Diagnostic Markers, see the corresponding sections in Inhalant Use Disorder.

Diagnostic Features

The essential feature of inhalant intoxication is the presence of clinically significant problematic behavioral or psychological changes that develop during, or immediately after, intended or unintended inhalation of a volatile hydrocarbon substance. When possible, the particular substance involved should be named (e.g., toluene intoxication). Intoxication clears within a few minutes to a few hours after the exposure ends. Thus, inhalant intoxication usually occurs in brief episodes that may recur with further inhalant use.

Associated Features

Inhalant intoxication may be indicated by evidence of possession, or lingering odors, of inhalant substances (e.g., glue, paint thinner, gasoline, butane lighters); other features may include euphoria, relaxation, headache, rapid heartbeat, confusion, talkativeness, blurred vision, amnesia, slurred speech, irritability, nausea, fatigue, burning in eyes or throat, grandiosity, chest pain, auditory or visual hallucinations, and dissociation.

Prevalence

The prevalence of actual episodes of inhalant intoxication in the general population is unknown, but it is probable that a majority of inhalant users would at some time exhibit behavioral or psychological changes and symptoms that would meet criteria for inhalant intoxication. Therefore, the prevalence of inhalant use and the prevalence of

inhalant intoxication are likely similar. In 2017, inhalant use in the past year was reported by 0.6% of all Americans older than 12 years; the prevalence was highest in younger age groups (2.3% for individuals ages 12 –17 years, 1.6% for individuals ages 18–25 years, and 0.3% for individuals age 26 and older).

Sex- and Gender-Related Diagnostic Issues

Gender differences in the prevalence of inhalant intoxication in the general population are unknown. Regarding gender differences in the prevalence of inhalant use in the United

States, 0.8% of boys/men older than 12 years and 0.5% of girls/women older than 12 years have used inhalants in the previous year, but in the younger age groups differences are minimal or girls may have slightly higher prevalence (e.g., among adolescents ages 12–17 years, 2.4% of girls and 2.2% of boys have used inhalants in the past year).

Functional Consequences of Inhalant Intoxication

Use of inhaled substances in a closed container, such as a plastic bag over the head, may lead to unconsciousness, anoxia, and death. Separately, "sudden sniffing death," likely from cardiac arrhythmia or arrest, may occur with various volatile inhalants. The enhanced toxicity of certain volatile inhalants, such as butane or propane, also causes fatalities. Although inhalant intoxication itself is of short duration, it may produce persisting medical and neurological problems, especially if the intoxications are frequent. Clinically significant correlates of inhalant intoxication include reckless behaviors (e.g., taking foolish risks, getting into fights, having unprotected sex), antisocial behaviors (cruelty, damaging property, arrests), and having serious accidents.

Differential Diagnosis

Intoxication from other substances, especially from sedating substances (e.g., alcohol, benzodiazepines, barbiturates).
These disorders may have similar signs and symptoms, but intoxication attributable to other intoxicants may be identified via a toxicology screen. Differentiating the source of the intoxication may involve discerning evidence of inhalant exposure as described for inhalant use disorder. A diagnosis of inhalant intoxication may be suggested by possession or lingering odors of inhalant substances (e.g., glue, paint thinner, gasoline, butane lighters); paraphernalia possession (e.g., rags or bags for concentrating glue fumes); perioral or perinasal "glue-sniffer's rash"; reports from family or friends that the intoxicated individual possesses or uses inhalants; or apparent intoxication despite negative results on standard drug screens (which usually fail to identify inhalants).

Inhalant-induced mental disorders. Inhalant intoxication is distinguished from inhalant-induced mental disorders (e.g., inhalant-induced anxiety disorder, with onset during intoxication) because the symptoms (e.g., anxiety) in these latter disorders are in excess of those usually associated with inhalant intoxication, predominate in the clinical presentation, and are severe enough to warrant independent clinical attention.

Other toxic, metabolic, traumatic, neoplastic, or infectious disorders that impair brain function and cognition.
Numerous neurological and other medical conditions may produce the clinically significant behavioral or psychological changes (e.g., belligerence, assaultiveness, apathy, impaired judgment) that also characterize inhalant intoxication.

Comorbidity

Given the typical overlap of inhalant intoxication with inhalant use disorder, see "Comorbidity" under Inhalant Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Inhalant-Induced Mental Disorders

The following inhalant-induced mental disorders are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): inhalant-induced psychotic disorder ("Schizophrenia Spectrum and Other Psychotic Disorders"); inhalant-induced depressive

disorder ("Depressive Disorders"); inhalant-induced anxiety disorder ("Anxiety Disorders"); and inhalant-induced major or mild neurocognitive disorder ("Neurocognitive Disorders"). For inhalant intoxication delirium, see the criteria and discussion of delirium in the chapter "Neurocognitive Disorders." These inhalant-induced mental disorders are diagnosed instead of inhalant intoxication only when symptoms are sufficiently severe to warrant independent clinical attention.

Unspecified Inhalant-Related Disorder

F18.99

This category applies to presentations in which symptoms characteristic of an inhalant-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific inhalant-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Opioid-Related Disorders

Opioid Use Disorder

Opioid Intoxication

Opioid Withdrawal

Opioid-Induced Mental Disorders

Unspecified Opioid-Related Disorder

Opioid Use Disorder

Diagnostic Criteria

A. A problematic pattern of opioid use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. Opioids are often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control opioid use.
3. A great deal of time is spent in activities necessary to obtain the opioid, use the opioid, or recover from its effects.
4. Craving, or a strong desire or urge to use opioids.
5. Recurrent opioid use resulting in a failure to fulfill major role obligations at work, school, or home.
6. Continued opioid use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of opioids.
7. Important social, occupational, or recreational activities are given up or reduced because of opioid use.

8. Recurrent opioid use in situations in which it is physically hazardous.
9. Continued opioid use despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.
10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of opioids to achieve intoxication or desired effect.
    b. A markedly diminished effect with continued use of the same amount of an opioid.

    Note: This criterion is not considered to be met for those taking opioids solely under appropriate medical supervision.
11. Withdrawal, as manifested by either of the following:
    a. The characteristic opioid withdrawal syndrome (refer to Criteria A and B of the criteria set for opioid withdrawal).
    b. Opioids (or a closely related substance) are taken to relieve or avoid withdrawal symptoms.

    Note: This criterion is not considered to be met for those individuals taking opioids solely under appropriate medical supervision.

Specify if:

In early remission: After full criteria for opioid use disorder were previously met, none of the criteria for opioid use disorder have been met for at least 3 months but for less than 12 months (with the exception that Criterion A4, “Craving, or a strong desire or urge to use opioids,” may be met).

In sustained remission: After full criteria for opioid use disorder were previously met, none of the criteria for opioid use disorder have been met at any time during a period of 12 months or longer (with the exception that Criterion A4, “Craving, or a strong desire or urge to use opioids,” may be met).

Specify if:

On maintenance therapy: This additional specifier is used if the individual is taking a prescribed agonist medication such as methadone or buprenorphine and none of the criteria for opioid use disorder have

been met for that class of medication (except tolerance to, or withdrawal from, the agonist). This category also applies to those individuals being maintained on a partial agonist, an agonist/antagonist, or a full antagonist such as oral naltrexone or depot naltrexone.

In a controlled environment: This additional specifier is used if the individual is in an environment where access to opioids is restricted.

Code based on current severity/remission: If an opioid intoxication, opioid withdrawal, or another opioid-induced mental disorder is also present, do not use the codes below for opioid use disorder. Instead, the comorbid opioid use disorder is indicated in the 4th character of the opioid-induced disorder code (see the coding note for opioid intoxication, opioid withdrawal, or a specific opioid-induced mental disorder). For example, if there is comorbid opioid-induced depressive disorder and opioid use disorder, only the opioid-induced depressive disorder code is given, with the 4th character indicating whether the comorbid opioid use disorder is mild, moderate, or severe: F11.14 for mild opioid use disorder with opioid-induced depressive disorder or F11.24 for a moderate or severe opioid use disorder with opioid-induced depressive disorder.

Specify current severity/remission:

F11.10 Mild: Presence of 2–3 symptoms.

F11.11 Mild, In early remission

F11.11 Mild, In sustained remission

F11.20 Moderate: Presence of 4–5 symptoms.

F11.21 Moderate, In early remission

F11.21 Moderate, In sustained remission

F11.20 Severe: Presence of 6 or more symptoms.

F11.21 Severe, In early remission

F11.21 Severe, In sustained remission

Specifiers

The "on maintenance therapy" specifier applies as a further specifier of remission if the individual is both in remission and receiving maintenance therapy. "In a controlled environment" applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

Changing severity across time in an individual is also reflected by reductions in the frequency (e.g., days of use per month) and/or dose (e.g., injections or number of pills) of an opioid, as assessed by the individual's self-report, report of knowledgeable others, clinician's observations, and biological testing.

Diagnostic Features

The opioids include natural opioids (e.g., morphine, codeine), semisynthetics (e.g., heroin, oxycodone, hydrocodone, hydromorphone, oxymorphone), and synthetics with morphine-like action (e.g., methadone, meperidine, tramadol, fentanyl, carfentanil). Medications such as pentazocine and buprenorphine that have both opiate agonist and

antagonist effects are also included in this class because, especially at lower doses, their agonist properties produce similar physiological and behavioral effects as classic opioid agonists. Opioids are prescribed as analgesics, anesthetics, antidiarrheal agents, or cough suppressants. Heroin is one of the most commonly misused drugs of this class and is usually taken by injection, although it can be smoked or "snorted," especially when very pure heroin is available. Fentanyl is typically injected, both medically and nonmedically, and is used medically in transdermal and transmucosal formulations, whereas cough suppressants and antidiarrheal agents are taken orally. The other opioids are generally taken both by injection and orally.

Opioid use disorder can arise from prescription opioids or illicit opioids (e.g., heroin and, especially in recent years, fentanyl-related synthetic opioids). Opioid use disorder consists of signs and symptoms reflecting compulsive, prolonged self-administration of opioid substances either for a purpose other than a legitimate medical one or for use in a "non-medical" manner (i.e., greatly exceeding the amount prescribed for a medical condition). For example, an individual with adequate doses of prescribed analgesic opioid medication for pain relief who uses significantly more of the medication than prescribed, and not only because of persistent pain, is engaging in nonmedical opioid use and may have an opioid use disorder. Most individuals with opioid use disorder have tolerance and experience withdrawal on abrupt cessation or reduction in opioid use. Similar to processes that occur with other psychoactive substances, individuals with opioid use disorder often develop conditioned responses to drug-related stimuli (e.g., cue-reactive craving on seeing drug images or paraphernalia). These responses probably contribute to relapse, are difficult to extinguish, and typically persist long after withdrawal is completed.

Individuals with opioid use disorder tend to develop such regular patterns of compulsive drug use that daily activities are planned around obtaining and administering

opioids. Prescription opioids used nonmedically can be obtained from family or friends, from physicians by falsifying or exaggerating medical problems, by receiving simultaneous prescriptions from several physicians, or via purchase on the illegal market. Health care professionals with opioid use disorder can obtain opioids by writing prescriptions for themselves or by diverting opioids that have been prescribed for individuals or from pharmacy supplies.

Associated Features

An attempt to achieve opioid intoxication may result in fatal or nonfatal opioid overdose. Opioid overdose is characterized by unconsciousness, respiratory depression, and pinpoint pupils. However, opioid overdoses can also occur in the absence of intoxication-seeking drug use. Opioid overdoses have increased exponentially in the United States since 1999. Up to 2009, opioid overdoses were mainly due to prescribed opioids, but since 2010, overdoses due to heroin began a sharp rise, and additionally, since 2015, fatal overdoses due to synthetic opioids other than methadone (generally fentanyl) have outnumbered overdoses due to prescribed opioids.

Opioid use disorder can be associated with a history of drug-related crimes (e.g., possession or distribution of drugs, forgery, burglary, robbery, larceny, receiving stolen goods). Among health care professionals and individuals who have ready access to controlled substances, a different pattern of illegal activities may involve problems with state licensing boards, professional staffs of hospitals, or other administrative

agencies. Marital difficulties (including divorce), unemployment, and irregular employment can be associated with opioid use disorder at all socioeconomic levels.

Prevalence

The prevalence of nonmedical prescription opioid use among U.S. adults age 18 and older is 4.1%–4.7%, with rates of use higher in adults ages 18–25 than in those age 26 and older (5.5% vs. 3.4%, respectively). The prevalence of heroin use in the United States is 0.3%–0.4% and is higher among adults ages 18–25 (0.5%–0.7%) than in other age groups. In U.S. adolescents ages 12–17, 2.8%–3.9% use prescription opioids nonmedically, with higher rates in older adolescents than in younger adolescents. Heroin use in adolescents is quite low (< 0.05%–0.1%).

The prevalence of prescription opioid use disorder among U.S. adults age 18 and older (DSM-IV or DSM-5 criteria) is 0.6%–0.9%, and the prevalence of heroin use disorder (DSM-IV or DSM-5 criteria) is 0.1%–0.3%. Among those ages 12–17, prevalence of prescription opioid use disorder is 0.4%, and heroin use disorder is rare (essentially 0%). In the United States, rates of opioid use disorder (prescription opioids and heroin) are higher among men than women, among young adults than older adults, and among those with lower income or education. Among U.S. adults in 2012–2013, the prevalence of nonmedical prescription opioid use disorder varied by ethnoracial group: 1.42% in Native Americans, 1.04% in African Americans, 0.96% in non-Latinx Whites, 0.70% in Latinx, and 0.16% in Asian Americans or Pacific Islanders. Rates based on household surveys may underestimate national prevalence by omitting individuals in institutions and jail or prison, whose rates are likely to be much higher.

Globally in 2016, there were 26.8 million cases of DSM-IV opioid dependence, with an age-standardized prevalence of 353.0 cases per 100,000 people; prevalence of opioid dependence across geographic regions ranged from 0.14% to 0.46%.

Development and Course

Opioid use disorder can begin at any age. In the United States, problems associated with opioid use are most commonly first observed in the late teens or early 20s, with a longer interval between first opioid use and onset of disorder for prescription opioids than for

heroin. Early use can reflect a desire for relief from life stressors or psychological pain. Long-term studies show that once an opioid use disorder that requires treatment develops, it can continue over many years, with brief periods of abstinence in some individuals but long-term abstinence only in a minority. An exception occurred among U.S. soldiers who became dependent on opioids while serving in the Vietnam War; over 90% had long-term abstinence from opioids after returning to the United States, although many subsequently experienced problems with alcohol, amphetamines, or suicidal thoughts or behavior.

Risk and Prognostic Factors

In addition to an association with more frequent nonmedical prescription opioid use, adult prescription opioid use disorder is associated with most other substance use disorders. Opioid use disorder is highly associated with externalizing traits such as novelty-seeking, impulsivity, and disinhibition. Family, peer, and social environmental factors all increase the risk for opioid use disorder. Family and twin studies also indicate a strong genetic contribution to the risk for opioid use disorders, although identifying the specific genetic variants contributing to genetic risk has been slow. Peer

factors may relate to genetic predisposition in terms of how individuals select their environments, including their peers.

Culture-Related Diagnostic Issues

Individuals from socially oppressed ethnoracial groups were historically overrepresented among individuals with opioid use disorder. However, over time, opioid use disorder has become more common among White individuals, suggesting that the widespread availability of opioids and other social factors (e.g., changes in rates of poverty and unemployment) have an impact on prevalence. Consistent with these factors, despite small variations between ethnoracial groups in the psychometric performance of opioid use disorder criterion items, the criteria for opioid use disorder perform equally well across ethnoracial groups.

Sex- and Gender-Related Diagnostic Issues

Women with opioid use disorder appear more likely than men to have initiated opioid use in response to sexual abuse and violence, and they are more likely than men to be introduced to the drug by a partner. There is substantial evidence of telescoping among women in that they progress to a use disorder more quickly than men after first use; women also appear to be more ill when entering treatment facilities than are men, as noted in a large sample of heroin users in Italy.

Diagnostic Markers

Routine urine toxicology test results are often positive for opioid drugs in individuals with opioid use disorder. Urine test results remain positive for most opioids (e.g., heroin, morphine, codeine, oxycodone, propoxyphene) for 12–36 hours after administration. Some opioids, such as fentanyl and oxycodone, are not detected by standard urine tests (which test for morphine), but can be identified by more specialized procedures for several days after use. Similarly, methadone and buprenorphine (or buprenorphine/naloxone combinations) will not cause a positive result on routine tests for opiates; they require specific tests that can detect these substances for several days up to more than 1 week.

Although not specific markers of opioid use disorder, laboratory evidence of the presence of other substances (e.g., cocaine, marijuana, alcohol, amphetamines, benzodiazepines) is common in heroin users. In addition, screening test results for hepatitis A, B, and C virus are often positive in injection opioid users, either for hepatitis antigen (signifying active infection) or for hepatitis antibody (signifying past infection). Mildly elevated liver

function test results are common, either as a result of resolving hepatitis or from toxic injury to the liver due to contaminants that have been mixed with the injected opioid. HIV is also prevalent in injection opioid users. Subtle changes in cortisol secretion patterns and body temperature regulation have been observed for up to 6 months following opioid withdrawal.

Association With Suicidal Thoughts or Behavior

Opioid use disorder is associated with a heightened risk for suicide attempts and suicide. Some suicide risk factors overlap with risk factors for an opioid use disorder. In addition, repeated opioid intoxication or withdrawal may be associated with severe depressions that although temporary can be intense enough to lead to suicide attempts and suicide. Nonfatal accidental opioid overdose and attempted suicide are distinct

phenomena that can be difficult to differentiate but should not be mistaken for each other, if possible.

Findings from the Global Burden of Disease Study 2010 showed that among drugs of abuse, suicide is a common cause of death among regular users of opioids. Evidence suggests that suicides are undercounted or often misclassified in opioid-poisoning data. In a study of the Veterans Health Administration (VHA) national medical records, after adjustment for psychiatric comorbidity, opioid use disorder elevated the risk for suicide mortality, with greater increase in risk among women than among men. In another study also using VHA national medical records, among veterans prescribed opioids for chronic pain, suicide mortality increased with higher opioid doses, even after demographic and clinical factors were taken into account. A follow-up of a U.S. national cohort of adults with a history of an opioid overdose found that the standardized mortality ratio (SMR; the ratio between the observed number of deaths in a study population and the number of deaths that would be expected) was 25.9 for suicide, with a higher SMR for women than for men. A review posited that the reasons for the increased risk for suicide among opioid users were related to shared risk factors, namely, comorbid mental disorders and pain.

Functional Consequences of Opioid Use Disorder

Physiologically, opioid use is associated with a lack of mucous membrane secretions, causing dry mouth and nose. Slowing of gastrointestinal activity and a decrease in gut motility can produce severe constipation. Visual acuity may be impaired as a result of pupillary constriction with acute administration. In individuals who inject opioids, sclerosed veins (“tracks”) and puncture marks on the lower portions of the upper extremities are common. Veins sometimes become so severely sclerosed that peripheral edema develops, and individuals switch to injecting in veins in the legs, neck, or groin. When these veins become unusable, individuals often inject directly into their subcutaneous tissue (“skin-popping”), resulting in cellulitis, abscesses, and circular-appearing scars from healed skin lesions. Tetanus and *Clostridium botulinum* infections are rare but serious consequences of injecting opioids, especially with contaminated needles. Infections may also occur in other organs and include bacterial endocarditis, hepatitis, and HIV infection. Hepatitis C infections, for example, may occur in up to 90% of individuals who inject opioids. In addition, the prevalence of HIV infection is high among individuals who inject drugs, a large proportion of whom are individuals with opioid use disorder. For example, HIV infection rates are as high as 60% among heroin users in some areas of the United States and the Russian Federation. However, the incidence may be much lower in areas where access to clean injection material and paraphernalia is facilitated.

Tuberculosis is a particularly serious problem among individuals who use drugs intravenously, especially those who are dependent on heroin; infection is usually asymptomatic and evident only by the presence of a positive tuberculin skin test or tuberculosis blood test (interferon gamma release assay). However, many cases of active tuberculosis have been found, especially among those who are infected with HIV. These individuals

often have a newly acquired infection but also are likely to experience reactivation of a prior infection because of impaired immune function.

Individuals who sniff heroin or other opioids into the nose (insufflation, or “snorting”) often develop irritation of the nasal mucosa, sometimes accompanied by perforation of the nasal septum. Difficulties in sexual functioning are common. Males

often experience erectile dysfunction during intoxication or chronic use. Females commonly have disturbances of reproductive function and irregular menses.

Although acute opioid use produces analgesia, chronic use can produce hyperalgesia (opioid-induced hyperalgesia), a condition characterized by increased sensitivity to pain. Physiological dependence on opioids may occur in about half of the infants born to females with opioid use disorder. This can produce a severe withdrawal syndrome in the neonate requiring medical treatment and has increased markedly in prevalence.

The mortality rate in individuals with opioid use disorder is 6–20 times greater than in the general population. Fatal overdoses due to prescription opioids increased dramatically in the United States since 1999, with almost 400,000 such deaths occurring since then, and the rate of such overdoses is now five times higher than in 1999. Fatal overdoses due to heroin began a sharp increase in 2010, and since 2013, fatal overdoses due to synthetic opioids (e.g., fentanyl) increased so sharply that these rates were almost double the rates for prescription opioid or heroin overdoses by 2017. Nonfatal opioid overdoses resulting in hospitalization and emergency department visits have increased as well. Although not all risk factors for opioid use disorder and opioid overdose are the same, substantial overlap exists, making the risk for overdose one of the most serious potential consequences of opioid use disorder. Individuals with opioid use disorder are also at increased risk for mortality from many medical conditions (e.g., hepatitis, HIV infection, tuberculosis, cardiovascular disease). Death can also result from accidents, injuries, or other general medical complications.

Differential Diagnosis

Opioid intoxication, opioid withdrawal, and opioid-induced mental disorders. Opioid use disorder is differentiated from opioid intoxication, opioid withdrawal, and opioid-induced mental disorders (e.g., opioid-induced depressive disorder) in that opioid use disorder describes a problematic pattern of opioid use that involves impaired control over opioid use, social impairment attributable to opioid use, risky opioid use (e.g., continued opioid use despite medical complications), and pharmacological symptoms (the development of tolerance or withdrawal), whereas opioid intoxication, opioid withdrawal, and opioid-induced mental disorders describe psychiatric syndromes that occur in the context of heavy use. Opioid intoxication, opioid withdrawal, and opioid-induced mental disorders occur frequently in individuals with opioid use disorder. In such cases, a diagnosis of opioid intoxication, opioid withdrawal, or opioid-induced mental disorder should be given in addition to a diagnosis of opioid use disorder, the presence of which is indicated in the diagnostic code.

Other substance intoxication. Alcohol intoxication and sedative, hypnotic, or anxiolytic intoxication can cause a clinical picture that resembles that of opioid intoxication. A diagnosis of alcohol or sedative, hypnotic, or anxiolytic intoxication can usually be made based on the absence of pupillary constriction or the lack of a response to naloxone challenge. In some cases, intoxication may be due both to opioids and to alcohol or other sedatives. In these cases, the naloxone challenge will not reverse all of the sedative effects.

Other withdrawal disorders. The anxiety and restlessness associated with opioid withdrawal resemble symptoms seen in sedative-hypnotic withdrawal. However, opioid withdrawal is also accompanied by rhinorrhea, lacrimation, and pupillary dilation, which are not seen in sedative-type withdrawal. Dilated pupils are also seen in hallucinogen intoxication and stimulant intoxication. However, other signs or symptoms of opioid

withdrawal, such as nausea, vomiting, diarrhea, abdominal cramps, rhinorrhea, or lacrimation, are not present.

Independent mental disorders. Some of the effects of opioid use may resemble symptoms (e.g., depressed mood) of an independent mental disorder (e.g., persistent depressive disorder). Opioids are less likely to produce symptoms of mental disturbance than are most other drugs of abuse.

Comorbidity

Other than overdose, the most common medical comorbidities associated with opioid use disorder are viral (e.g., HIV, hepatitis C virus) and bacterial infections, particularly among injection heroin users. These infections are less common in prescription opioid use disorder.

Research with nationally representative samples of the U.S. population has found that opioid use disorder is often associated with other substance use disorders, especially those involving tobacco, alcohol, cannabis, stimulants, and benzodiazepines. Individuals with opioid use disorder are at risk for the development of persistent depressive disorder or major depressive disorder. These symptoms may represent an opioid-induced depressive disorder or an exacerbation of a preexisting independent depressive disorder. Periods of depression are especially common during chronic intoxication or in association with physical or psychosocial stressors related to the opioid use disorder. Insomnia is also common, especially during withdrawal. Opioid use disorder is also associated with bipolar I disorder, posttraumatic stress disorder, and antisocial, borderline, and schizotypal personality disorders. A history of conduct disorder in childhood or adolescence has also been identified as a significant risk factor for substance-related disorders, especially opioid use disorder. Further, prescription opioid use disorder and heroin use disorder are generally associated with *serious mental illness*, defined as a mental disorder other than a substance use disorder that results in serious functional impairment substantially limiting or interfering with major life activities.

Opioid Intoxication

Diagnostic Criteria

A. Recent use of an opioid.

B. Clinically significant problematic behavioral or psychological changes (e.g., initial euphoria followed by apathy, dysphoria, psychomotor agitation or retardation, impaired judgment) that developed during, or shortly after, opioid use.

C. Pupillary constriction (or pupillary dilation due to anoxia from severe overdose) and one (or more) of the following signs or symptoms developing during, or shortly after, opioid use:

   1. Drowsiness or coma.
   2. Slurred speech.
   3. Impairment in attention or memory.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Specify if:

With perceptual disturbances: This specifier may be noted in the rare instance in which hallucinations with intact reality testing or auditory, visual, or tactile illusions occur in the absence of a delirium.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid opioid use disorder and whether or not there are perceptual disturbances.

For opioid intoxication, without perceptual disturbances: If a mild opioid use disorder is comorbid, the ICD-10-CM code is **F11.120,** and if a moderate or severe opioid use disorder is comorbid, the ICD-10-CM code is **F11.220.** If there is no comorbid opioid use disorder, then the ICD-10-CM code is **F11.920.**

For opioid intoxication, with perceptual disturbances: If a mild opioid use disorder is comorbid, the ICD-10-CM code is **F11.122,** and if a moderate or severe opioid use disorder is comorbid, the ICD-10-CM code is **F11.222.** If there is no comorbid opioid use disorder, then the ICD-10-CM code is **F11.922.**

Diagnostic Features

The essential feature of opioid intoxication is the presence of clinically significant problematic behavioral or psychological changes (e.g., initial euphoria followed by apathy, dysphoria, psychomotor agitation or retardation, impaired judgment) that develop during, or shortly after, opioid use (Criteria A and B). Intoxication is accompanied by pupillary constriction (unless there has been a severe overdose with consequent anoxia and pupillary dilation) and one or more of the following signs: drowsiness (described as being "on the nod"), slurred speech, and impairment in attention or memory (Criterion C); drowsiness may progress to coma. Individuals with opioid intoxication may demonstrate inattention to the environment, even to the point of ignoring potentially harmful events. The signs or symptoms of opioid intoxication must not be attributable to another medical condition and are not better explained by another mental disorder (Criterion D).

Up to 2009, opioid overdoses were mainly due to prescribed opioids, but starting in 2010, overdoses due to heroin began a sharp rise, and additionally, since 2015, fatal overdoses due to synthetic opioids other than methadone (generally fentanyl) have outnumbered overdoses due to prescribed opioids.

Associated Features

Opioid intoxication can include decreases in respiratory rate and blood pressure, and mild hypothermia. The duration of opioid intoxication can vary as a function of the pharmacokinetics of the opioid ingested. Opioid intoxication may result in fatal or nonfatal opioid overdose. Opioid overdose is characterized by unconsciousness, respiratory depression, and pinpoint pupils. Fatal opioid overdoses have increased exponentially in the United States since 1999.

Development and Course

Opioid intoxication can occur in an individual who is opioid naïve, an individual who uses opioids sporadically, and an individual who is physically dependent on opioids. The dose of opioid consumed relative to the likelihood of experiencing opioid intoxication will vary as a function of the status and history of the individual's opioid

exposure (i.e., tolerance). Individuals often report that the qualitative pleasurable experience of opioid intoxication diminishes after repeated use of an opioid.

Differential Diagnosis

Other substance intoxication. Alcohol intoxication and sedative-hypnotic intoxication can cause a clinical picture that resembles opioid intoxication. A diagnosis of alcohol or sedative-hypnotic intoxication can usually be made based on the absence of pupillary constriction or the lack of a response to a naloxone challenge. In some cases, intoxication may be due both to opioids and to alcohol or other sedatives. In these cases, naloxone administration will not reverse all of the sedative effects. While response to administration of

naloxone can support the diagnosis of opioid intoxication, nonresponse may be due to the co-ingestion of an opioid with another drug (e.g., a benzodiazepine, alcohol) or to ingestion of a higher dose of and/or higher-potency opioid (e.g., fentanyl).

Opioid-induced mental disorders. Opioid intoxication is distinguished from opioid-induced mental disorders (e.g., opioid-induced depressive disorder, with onset during intoxication) because the symptoms (e.g., depressed mood) in the latter disorders are in excess of those usually associated with opioid intoxication, predominate in the clinical presentation, and are severe enough to warrant clinical attention.

Comorbidity

Given the typical overlap of opioid intoxication with opioid use disorder, see "Comorbidity" under Opioid Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Opioid Withdrawal

Diagnostic Criteria

A. Presence of either of the following:
   1. Cessation of (or reduction in) opioid use that has been heavy and prolonged (i.e., several weeks or longer).
   2. Administration of an opioid antagonist after a period of opioid use.

B. Three (or more) of the following developing within minutes to several days after Criterion A:
   1. Dysphoric mood.
   2. Nausea or vomiting.
   3. Muscle aches.
   4. Lacrimation or rhinorrhea.
   5. Pupillary dilation, piloerection, or sweating.
   6. Diarrhea.
   7. Yawning.
   8. Fever.
   9. Insomnia.

C. The signs or symptoms in Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication or withdrawal from another substance.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid opioid use disorder. If a mild opioid use disorder is comorbid, the ICD-10-CM code is **F11.13**, and if a moderate or severe opioid use disorder is comorbid, the ICD-10-CM code is **F11.23**. For opioid withdrawal occurring in the absence of an opioid use disorder (e.g., in a patient taking opioids solely under appropriate medical supervision), the ICD-10-CM code is **F11.93**.

Diagnostic Features

The essential feature of opioid withdrawal is the presence of a characteristic withdrawal syndrome that develops after the cessation of (or reduction in) prolonged opioid use

(Criterion A1). The opioids used may be illicit or licitly obtained drugs prescribed to treat pain. A withdrawal syndrome can also be precipitated by administration of an opioid antagonist (e.g., naloxone, naltrexone, nalmefene) after a period of opioid use (Criterion A2); it can also occur after administration of an opioid partial agonist (e.g., buprenorphine) to an individual currently using a full opioid agonist.

Opioid withdrawal has a characteristic pattern of signs and symptoms. The first of these are subjective and consist of complaints of anxiety, restlessness, and an "achy feeling" that is often located in the back and legs, along with irritability and increased sensitivity to pain. Three or more of the following must be present to make a diagnosis of opioid withdrawal: dysphoric mood; nausea or vomiting; muscle aches; lacrimation or rhinorrhea; pupillary dilation, piloerection, or increased sweating; diarrhea; yawning; fever; and insomnia (Criterion B). Piloerection and fever are associated with more severe withdrawal and are not often seen in routine clinical practice because individuals with opioid use disorder usually obtain substances before withdrawal becomes that far advanced. These symptoms of opioid withdrawal must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C). The symptoms must not be attributable to another medical condition and are not better explained by another mental disorder (Criterion D). Having symptoms that meet diagnostic criteria for opioid withdrawal alone is not sufficient for a diagnosis of opioid use disorder, but concurrent symptoms of craving and drug-seeking behavior are suggestive of comorbid opioid use disorder.

Associated Features

Opioid withdrawal may occur in any individual after cessation of repeated use of an opioid, whether in the setting of medical management of pain, during opioid agonist therapy for opioid use disorder, in the context of illicit use, or following attempts to self-treat symptoms of mental disorders with opioids. Opioid withdrawal is a distinct condition from opioid addiction or opioid use disorder and does not necessarily require the drug-seeking behaviors associated with opioid use disorder to be diagnosed. Thus, opioid withdrawal may occur in individuals without opioid use disorder and should not

be confused with it. Males with opioid withdrawal may experience piloerection, sweating, and spontaneous ejaculations while awake.

Prevalence

Among individuals from various U.S. clinical settings, opioid withdrawal occurred in 60% of individuals who had used heroin at least once in the prior 12 months. Individuals regularly using opioids (e.g., prescription opioids for pain, illicit opioids) for a period of time are at risk for developing physical dependence, including withdrawal, on cessation or marked reduction in use.

Development and Course

The speed and severity of withdrawal associated with opioids depend on the half-life of the opioid used. Most individuals who are physiologically dependent on short-acting drugs such as heroin begin to have withdrawal symptoms within 6–12 hours after the last dose. Symptoms may take 2–4 days to emerge in the case of longer-acting drugs such as methadone or buprenorphine. Acute withdrawal symptoms for a short-acting opioid such as heroin usually peak within 1–3 days and gradually subside over a period of 5–7 days. More chronic symptoms (e.g., anxiety, dysphoria, anhedonia, craving, insomnia) can last for weeks to months. The severity of opioid withdrawal also varies depending on the duration of opioid use. Opioid withdrawal symptoms among individuals receiving long-term prescription opioid treatment for pain can be minimized by tapering the drug slowly.

Among those with an opioid use disorder, opioid withdrawal and attempts to relieve withdrawal are typical. The course of withdrawal can be part of an escalating pattern in which an opioid is used to reduce withdrawal symptoms, in turn leading to recurrent episodes of withdrawal at a later time.

Differential Diagnosis

Other withdrawal disorders. The anxiety and restlessness associated with opioid withdrawal resemble symptoms seen in sedative-hypnotic withdrawal. However, opioid withdrawal is also accompanied by rhinorrhea, lacrimation, and pupillary dilation, which are not seen in sedative-type withdrawal.

Other substance intoxication. Dilated pupils are also seen in hallucinogen intoxication and stimulant intoxication. However, other signs or symptoms of opioid withdrawal, such as nausea, vomiting, diarrhea, abdominal cramps, rhinorrhea, and lacrimation, are not present.

Opioid-induced mental disorders. Opioid withdrawal is distinguished from opioid-induced mental disorders (e.g., opioid-induced depressive disorder, with onset during withdrawal) because the symptoms (e.g., depressed mood) in these latter disorders are in excess of those usually associated with opioid withdrawal, predominate in the clinical presentation, and are severe enough to warrant clinical attention.

Comorbidity

Given the typical overlap of opioid withdrawal with opioid use disorder, see “Comorbidity” under Opioid Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Opioid-Induced Mental Disorders

The following opioid-induced mental disorders are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): opioid-induced depressive disorder ("Depressive Disorders"); opioid-induced anxiety disorder ("Anxiety Disorders"); opioid-induced sleep disorder ("Sleep-Wake Disorders"); and opioid-induced sexual dysfunction ("Sexual Dysfunctions"). For opioid intoxication delirium, opioid withdrawal delirium, and delirium induced by opioids taken as prescribed, see the criteria and discussion of delirium in the chapter "Neurocognitive Disorders." These opioid-induced mental disorders are diagnosed instead of opioid intoxication or opioid withdrawal only when the symptoms are sufficiently severe to warrant independent clinical attention.

Unspecified Opioid-Related Disorder

F11.99

This category applies to presentations in which symptoms characteristic of an opioid-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific opioid-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Sedative-, Hypnotic-, or Anxiolytic-Related Disorders

Sedative, Hypnotic, or Anxiolytic Use Disorder

Sedative, Hypnotic, or Anxiolytic Intoxication

Sedative, Hypnotic, or Anxiolytic Withdrawal

Sedative-, Hypnotic-, or Anxiolytic-Induced Mental Disorders

Unspecified Sedative-, Hypnotic-, or Anxiolytic-Related Disorder

Sedative, Hypnotic, or Anxiolytic Use Disorder

Diagnostic Criteria

A. A problematic pattern of sedative, hypnotic, or anxiolytic use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

   1. Sedatives, hypnotics, or anxiolytics are often taken in larger amounts or over a longer period than was intended.
   2. There is a persistent desire or unsuccessful efforts to cut down or control sedative, hypnotic, or anxiolytic use.

3. A great deal of time is spent in activities necessary to obtain the sedative, hypnotic, or anxiolytic; use the sedative, hypnotic, or anxiolytic; or recover from its effects.
4. Craving, or a strong desire or urge to use the sedative, hypnotic, or anxiolytic.
5. Recurrent sedative, hypnotic, or anxiolytic use resulting in a failure to fulfill major role obligations at work, school, or home (e.g., repeated absences from work or poor work performance related to sedative, hypnotic, or anxiolytic use; sedative-, hypnotic-, or anxiolytic-related absences, suspensions, or expulsions from school; neglect of children or household).
6. Continued sedative, hypnotic, or anxiolytic use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of sedatives, hypnotics, or anxiolytics (e.g., arguments with a spouse about consequences of intoxication; physical fights).
7. Important social, occupational, or recreational activities are given up or reduced because of sedative, hypnotic, or anxiolytic use.
8. Recurrent sedative, hypnotic, or anxiolytic use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by sedative, hypnotic, or anxiolytic use).
9. Sedative, hypnotic, or anxiolytic use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the sedative, hypnotic, or anxiolytic.
10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of the sedative, hypnotic, or anxiolytic to achieve intoxication or desired effect.
    b. A markedly diminished effect with continued use of the same amount of the sedative, hypnotic, or anxiolytic.

    Note: This criterion is not considered to be met for individuals taking sedatives, hypnotics, or anxiolytics under medical supervision.
11. Withdrawal, as manifested by either of the following:
    a. The characteristic withdrawal syndrome for sedatives, hypnotics, or anxiolytics (refer to Criteria A and B of the criteria set for sedative, hypnotic, or anxiolytic withdrawal).
    b. Sedatives, hypnotics, or anxiolytics (or a closely related substance, such as alcohol) are taken to relieve or avoid withdrawal symptoms.

    Note: This criterion is not considered to be met for individuals taking sedatives, hypnotics, or anxiolytics under medical supervision.

Specify if:

In early remission: After full criteria for sedative, hypnotic, or anxiolytic use disorder were previously met, none of the criteria for sedative, hypnotic, or anxiolytic use disorder have been met for at least 3 months but for less than 12 months (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the sedative, hypnotic, or anxiolytic,” may be met).

In sustained remission: After full criteria for sedative, hypnotic, or anxiolytic use disorder were previously met, none of the criteria for sedative, hypnotic, or anxiolytic use disorder have been met at any time during a period of 12 months or longer (with the exception that Criterion A4, "Craving, or a strong desire or urge to use the sedative, hypnotic, or anxiolytic," may be met).

Specify if:

In a controlled environment: This additional specifier is used if the individual is in an environment where access to sedatives, hypnotics, or anxiolytics is restricted.

Code based on current severity/remission: If a sedative, hypnotic, or anxiolytic intoxication; sedative, hypnotic, or anxiolytic withdrawal; or another sedative-, hypnotic-, or anxiolytic-induced mental disorder is also present, do not use the codes below for sedative, hypnotic, or anxiolytic use disorder. Instead, the comorbid sedative, hypnotic, or anxiolytic use disorder is indicated in the 4th character of the sedative-, hypnotic-, or anxiolytic-induced disorder (see the coding note for sedative, hypnotic, or anxiolytic intoxication; sedative, hypnotic, or anxiolytic withdrawal; or specific sedative-, hypnotic-, or anxiolytic-induced mental disorder). For example, if there is comorbid sedative-, hypnotic-, or anxiolytic-induced depressive disorder and sedative, hypnotic, or anxiolytic use disorder, only the sedative-, hypnotic-, or anxiolytic-induced depressive disorder code is given, with the 4th character indicating whether the comorbid sedative, hypnotic, or anxiolytic use disorder is mild, moderate, or severe: F13.14 for mild sedative, hypnotic, or anxiolytic use disorder with sedative-, hypnotic-, or anxiolytic-induced depressive disorder or F13.24 for a moderate or severe sedative, hypnotic, or anxiolytic use disorder with sedative-, hypnotic-, or anxiolytic-induced depressive disorder.

Specify current severity/remission:

F13.10 Mild: Presence of 2–3 symptoms.

F13.11 Mild, In early remission

F13.11 Mild, In sustained remission

F13.20 Moderate: Presence of 4–5 symptoms.

F13.21 Moderate, In early remission

F13.21 Moderate, In sustained remission

F13.20 Severe: Presence of 6 or more symptoms.

F13.21 Severe, In early remission

F13.21 Severe, In sustained remission

Specifiers

"In a controlled environment" applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

Diagnostic Features

Sedative, hypnotic, or anxiolytic substances include benzodiazepines, benzodiazepine-like drugs (e.g., zolpidem, zaleplon), carbamates (e.g., glutethimide, meprobamate), barbiturates (e.g., secobarbital), and barbiturate-like hypnotics (e.g., glutethimide, methaqualone, propofol). This class of substances includes most prescription sleeping medications and most prescription antianxiety medications. Nonbenzodiazepine antianxiety agents (e.g., buspirone, gepirone) are not included in this class because they do not appear to be associated with significant misuse.

Like alcohol, these agents are brain depressants and can produce similar substance/medication-induced and substance use disorders. Sedative, hypnotic, or anxiolytic substances are available both by prescription and illegally. Some individuals who obtain these substances by prescription will develop a sedative, hypnotic, or anxiolytic use disorder, while others who misuse these substances or use them for intoxication will not develop a use disorder. In particular, sedatives, hypnotics, or anxiolytics with rapid onset or short to intermediate lengths of action may be taken for intoxication purposes, although longer-acting substances in this class may be taken for intoxication as well.

Craving (Criterion A4), either during periods of active use or during periods of abstinence, is a typical feature of sedative, hypnotic, or anxiolytic use disorder. Misuse of substances from this class may occur in conjunction with use of other substances. For example, individuals may use intoxicating doses of sedatives or benzodiazepines to "come down" from cocaine or amphetamines or use high doses of benzodiazepines in combination with methadone to "boost" its effects.

Repeated absences or poor work performance, school absences, suspensions or expulsions, and neglect of children or household (Criterion A5) may be related to sedative, hypnotic, or anxiolytic use disorder; the continued use of the substances despite arguments with a spouse about consequences of intoxication or despite physical fights (Criterion A6) may also occur. Limiting contact with family or friends, avoiding work or school, or stopping participation in hobbies, sports, or games (Criterion A7) and recurrent sedative, hypnotic, or anxiolytic use when driving an automobile or operating machinery when impaired by such use (Criterion A8) are also seen in sedative, hypnotic, or anxiolytic use disorder.

Very significant levels of tolerance and withdrawal can develop to sedative, hypnotic, or anxiolytic substances. There may be evidence of tolerance and withdrawal in the absence of a diagnosis of a sedative, hypnotic, or anxiolytic use disorder in an individual who has abruptly discontinued use of benzodiazepines that were taken for long periods of time at prescribed and therapeutic doses. In these cases, an additional diagnosis of sedative, hypnotic, or anxiolytic use disorder is made only if other criteria are met. That is, sedative, hypnotic, or anxiolytic medications may be prescribed for appropriate medical purposes, and depending on the dose regimen, these drugs may then produce tolerance and withdrawal. If these drugs are prescribed or recommended for appropriate medical purposes, and if they are used as prescribed, the resulting tolerance or withdrawal does not count toward the diagnosis of a substance use disorder. However, it is necessary to determine whether the drugs were inappropriately prescribed and used (e.g., falsifying medical symptoms to obtain the medication; using more medication than prescribed; obtaining the medication from several doctors without informing them).

Given the unidimensional nature of the symptoms of sedative, hypnotic, or anxiolytic use disorder, severity is based on the number of criteria endorsed.

Associated Features

Research with nationally representative samples of the U.S. population has found that sedative, hypnotic, or anxiolytic use disorder is often associated with other substance use disorders (e.g., alcohol, cannabis, opioid, stimulant use disorders). Sedatives are often used to alleviate the unwanted effects of these other substances. With repeated use of the sedative, hypnotic, or anxiolytic, tolerance develops to the sedative effects, and a progressively higher dose is used. However, tolerance to brain stem depressant effects develops much more slowly, and as the individual takes more substance to achieve euphoria or other desired effects, there may be a sudden onset of respiratory depression and hypotension, which may result in death. Intense or repeated sedative, hypnotic, or anxiolytic intoxication may be associated with severe depression that although temporary can lead to suicide attempt and suicide.

Prevalence

The 12-month prevalence of DSM-IV sedative, hypnotic, or anxiolytic use disorder in the United States is estimated to be 0.3% among adolescents ages 12–17 years and adults age 18 years and older, and this prevalence has remained stable nationally despite increases in rates of prescription of these medications. Rates of DSM-IV sedative, hypnotic, or anxiolytic use disorder in the United States have not been shown to vary consistently by gender, but data from other countries have generally found higher rates among girls and women than boys and men. The 12-month prevalence of DSM-IV sedative, hypnotic, or anxiolytic use disorder in the United States decreases as a function of age and is greatest among individuals ages 18–29 years (0.5%) and lowest among individuals 65 years and older (0.04%).

Twelve-month prevalence of sedative, hypnotic, or anxiolytic use, misuse (e.g., use without a prescription), or disorder varies across U.S. ethnoracial groups. For instance, 12-month prevalence estimates for sedative, hypnotic, or anxiolytic misuse across ethnoracial groups range from 0.6% to 2.5% for adolescents ages 12–17 years and 0.7% to 10.1% for adults.

Development and Course

The usual course of sedative, hypnotic, or anxiolytic use disorder involves individuals in their teens or 20s who escalate their occasional use of sedative, hypnotic, or anxiolytic agents to the point at which they develop problems that meet criteria for a diagnosis. This pattern may be especially likely among individuals who have other substance use disorders (e.g., alcohol, opioids, stimulants). An initial pattern of intermittent use socially (e.g., at parties) can lead to daily use and high levels of tolerance. Once this occurs, an increasing level of interpersonal difficulties can be expected, as well as increasingly severe episodes of cognitive dysfunction and physiological withdrawal.

The second and less frequently observed clinical course begins with an individual who originally obtained the medication by prescription from a physician, usually for the treatment of anxiety, insomnia, or somatic complaints. As either tolerance or a need for higher doses of the medication develops, there is a gradual increase in the dose and frequency of self-administration. The individual is likely to continue to justify use on the basis of original anxiety or insomnia symptoms, but substance-seeking behavior becomes more prominent, and the individual may seek out multiple physicians to obtain sufficient supplies of the medication. Tolerance can reach high levels, and withdrawal (including seizures and withdrawal delirium) may occur.

As with many substance use disorders, sedative, hypnotic, or anxiolytic use disorder generally has an onset during adolescence or early adult life. Although the risk for misuse and use disorder decreases with age after about age 30, side effects associated with psychoactive substances may increase as individuals age. In particular,

cognitive impairment increases as a side effect with age, and the metabolism of sedatives, hypnotics, or

anxiolytics decreases with age among older individuals. Both acute and chronic toxic effects of these substances, especially effects on cognition, memory, and motor coordination, are likely to increase with age as a consequence of pharmacodynamic and pharmacokinetic age-related changes. Individuals with major neurocognitive disorder are more likely to develop intoxication and impaired physiological functioning at lower doses. Because sedatives, hypnotics, and anxiolytics are often used in combination with other psychoactive substances, it can be difficult to ascertain whether the functional consequences are attributable to a single substance (e.g., sedative) or to the use of multiple substances.

Deliberate intoxication to achieve a "high" is most likely to be observed in teenagers and individuals in their 20s. Problems associated with sedatives, hypnotics, or anxiolytics are also seen in individuals in their 40s and older who escalate the dose of prescribed medications. In older individuals, intoxication can resemble a progressive major neurocognitive disorder.

Risk and Prognostic Factors

Temperamental. Impulsivity and novelty seeking are individual temperaments that relate to the propensity to develop a substance use disorder but may themselves be genetically determined. Personality disorders can also increase the risk of sedative, hypnotic, or anxiolytic misuse or use disorder.

Environmental. Because sedatives, hypnotics, or anxiolytics are all medications, a key risk factor relates to availability of the substances, both through an individual's own prescriptions and from prescriptions dispensed to family and friends. In the United States, the historical patterns of sedative, hypnotic, or anxiolytic misuse relate to broad prescribing patterns. For instance, a marked decrease in prescription of barbiturates was associated with an increase in benzodiazepine prescriptions. Peer factors may relate to genetic predisposition in terms of how individuals select their environment. Other individuals at heightened risk might include those with alcohol use disorder who may receive repeated prescriptions in response to their complaints of alcohol-related anxiety or insomnia.

Genetic and physiological. As with other substance use disorders, the risk for sedative, hypnotic, or anxiolytic use disorder has been found in U.S.-based twin registry studies to be related to individual, family, peer, social, and environmental factors. Within these domains, genetic factors play a particularly important role both directly and indirectly. Overall, across development, genetic factors seem to play a larger role in the onset of sedative, hypnotic, or anxiolytic use disorder as individuals age through puberty into adult life.

Course modifiers. In nationally representative U.S. studies, early onset of use is associated with greater likelihood for developing a sedative, hypnotic, or anxiolytic use disorder.

Culture-Related Diagnostic Issues

Prescription patterns (and availability) of this class of substances vary across countries and populations, which may lead to variations in prevalence of sedative, hypnotic, or anxiolytic use disorder. In the United States, use of benzodiazepines has been more frequently reported by non-Latinx Whites than Latinx or African Americans. However, risk of the disorder may vary within populations exposed to these substances. For

example, the 12-month prevalence of DSM-IV benzodiazepine use disorder among U.S. individuals who used benzodiazepines was higher among African Americans (3.0%) and non-Latinx "others" (2.6%) than among non-Latinx Whites (1.3%).

Sex- and Gender-Related Diagnostic Issues

Although estimates from individual studies vary, there appear to be no gender differences in the prevalence of sedative, hypnotic, or anxiolytic use disorder.

Diagnostic Markers

Almost all sedative, hypnotic, or anxiolytic substances can be identified through laboratory evaluations of urine or blood (the latter of which can quantify the amounts of these agents in the body). Urine test results are likely to remain positive for up to approximately 1 week after the use of long-acting substances, such as diazepam or flurazepam.

Association With Suicidal Thoughts or Behavior

U.S. epidemiological studies show that hypnotics are associated with suicide, but it is unclear if this association is attributable to underlying psychiatric conditions such as depression and insomnia, which are themselves risk factors for suicide.

Functional Consequences of Sedative, Hypnotic, or Anxiolytic Use Disorder

The social and interpersonal consequences of sedative, hypnotic, or anxiolytic use disorder mimic those of alcohol in terms of the potential for disinhibited behavior. Accidents, interpersonal difficulties, and interference with work or school performance are common outcomes. The disinhibiting effects of these agents, like alcohol, may potentially contribute to overly aggressive behavior and arguments or fights, with subsequent interpersonal and legal problems. Physical examination is likely to reveal evidence of a mild decrease in most aspects of autonomic nervous system functioning, including a slower pulse, a slightly decreased respiratory rate, and a slight drop in blood pressure (most likely to occur with postural changes).

Acute intoxication can result in accidental injuries and automobile accidents. There may be consequences of trauma (e.g., internal bleeding, a subdural hematoma) from accidents that occur while intoxicated. For elderly individuals, even short-term use of these sedating medications at prescribed doses may be associated with an increased risk for cognitive problems and falls. The association of sedative, hypnotic, or anxiolytic medications with increased risk of major neurocognitive disorder remains unclear.

At high doses, sedative, hypnotic, or anxiolytic substances can be lethal, particularly when mixed with other central nervous system depressants, such as opioids or alcohol, although the lethal dosage varies considerably among the specific substances. Intravenous use of these substances can result in medical complications related to the use of contaminated needles (e.g., hepatitis, HIV).

Accidental or deliberate overdoses, similar to those observed for alcohol use disorder or repeated alcohol intoxication, can occur. Overdoses may be associated with a deterioration in vital signs that signals an impending medical emergency (e.g., respiratory arrest from barbiturates). In contrast to their wide margin of safety when used alone, benzodiazepines taken in combination with opioids and alcohol can be particularly dangerous, and accidental overdoses are reported commonly in U.S. data. Accidental overdoses have also been reported in individuals who deliberately misuse

barbiturates and other nonbenzodiazepine sedatives (e.g., methaqualone), but because these agents are much less available than the benzodiazepines, the frequency of overdosing is low in most settings.

Differential Diagnosis

Sedative, hypnotic, or anxiolytic intoxication; sedative, hypnotic, or anxiolytic withdrawal; and sedative-, hypnotic-, or anxiolytic-induced mental disorders.
Sedative, hypnotic, or anxiolytic use disorder is differentiated from sedative, hypnotic, or anxiolytic intoxication; sedative, hypnotic, or anxiolytic withdrawal; and sedative-, hypnotic-, or anxiolytic-induced mental disorders (e.g., sedative-, hypnotic-, or anxiolytic-induced depressive disorder) in that sedative, hypnotic, or anxiolytic use disorder describes a problematic pattern

of sedative, hypnotic, or anxiolytic use that involves impaired control over such use; social impairment attributable to this use; risky sedative, hypnotic, or anxiolytic use (e.g., driving while intoxicated); and pharmacological symptoms (the development of tolerance or withdrawal); whereas sedative, hypnotic, or anxiolytic intoxication; sedative, hypnotic, or anxiolytic withdrawal; and sedative-, hypnotic-, or anxiolytic-induced mental disorders describe psychiatric syndromes that occur in the context of heavy use. Sedative, hypnotic, or anxiolytic intoxication; sedative, hypnotic, or anxiolytic withdrawal; and sedative-, hypnotic-, or anxiolytic-induced mental disorders occur frequently in individuals with sedative, hypnotic, or anxiolytic use disorder. In such cases, a diagnosis of sedative, hypnotic, or anxiolytic intoxication; sedative, hypnotic, or anxiolytic withdrawal; or a sedative-, hypnotic-, or anxiolytic-induced mental disorder should be given in addition to a diagnosis of sedative, hypnotic, and anxiolytic use disorder, the presence of which is indicated in the diagnostic code.

Other medical conditions. The slurred speech, incoordination, and other associated features characteristic of sedative, hypnotic, or anxiolytic intoxication could be the result of another medical condition (e.g., multiple sclerosis) or of a prior head trauma (e.g., a subdural hematoma).

Alcohol use disorder. Sedative, hypnotic, or anxiolytic use disorder must be differentiated from alcohol use disorder. The differential diagnosis is determined mostly through clinical history, although liver damage and other potential signs of chronic alcohol toxicity (e.g., cardiomyopathy) can also be more suggestive of alcohol use disorder than of sedative, hypnotic, or anxiolytic use disorder.

Clinically appropriate use of sedative, hypnotic, or anxiolytic medications. Individuals may continue to take benzodiazepine medication according to a physician's direction for a legitimate medical indication over extended periods of time. Even if physiological signs of tolerance or withdrawal are manifested, many of these individuals do not develop symptoms that meet the criteria for sedative, hypnotic, or anxiolytic use disorder because they are not preoccupied with obtaining the substance and its use does not interfere with their performance of usual social or occupational roles.

Comorbidity

Nonmedical use of sedative, hypnotic, or anxiolytic agents is associated with alcohol use disorder, tobacco use disorder, and, generally, illicit drug use. There may also be an overlap between sedative, hypnotic, or anxiolytic use disorder and antisocial personality disorder; depressive, bipolar, and anxiety disorders; and other substance use disorders, such as alcohol use disorder and illicit drug use disorders. Antisocial behavior and antisocial personality disorder are especially associated with sedative, hypnotic, or anxiolytic use disorder when the substances are obtained illegally.

Comorbidity with other substance use disorders and other psychiatric disorders increases the risk of transition from sedative, hypnotic, or anxiolytic use to use disorder and decreases the probability of remission.

Sedative, Hypnotic, or Anxiolytic Intoxication

Diagnostic Criteria

A. Recent use of a sedative, hypnotic, or anxiolytic.
B. Clinically significant maladaptive behavioral or psychological changes (e.g., inappropriate sexual or aggressive behavior, mood lability, impaired judgment) that developed during, or shortly after, sedative, hypnotic, or anxiolytic use.

C. One (or more) of the following signs or symptoms developing during, or shortly after, sedative, hypnotic, or anxiolytic use:
    1. Slurred speech.
    2. Incoordination.
    3. Unsteady gait.
    4. Nystagmus.
    5. Impairment in cognition (e.g., attention, memory).
    6. Stupor or coma.
D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Coding note: The ICD-10-CM code depends on whether there is a comorbid sedative, hypnotic, or anxiolytic use disorder. If a mild sedative, hypnotic, or anxiolytic use disorder is comorbid, the ICD-10-CM code is **F13.120,** and if a moderate or severe sedative, hypnotic, or anxiolytic use disorder is comorbid, the ICD-10-CM code is **F13.220.** If there is no comorbid sedative, hypnotic, or anxiolytic use disorder, then the ICD-10-CM code is **F13.920.**

Note: For information on Development and Course; Risk and Prognostic Factors; Culture-Related Diagnostic Issues; Diagnostic Markers; Functional Consequences of Sedative, Hypnotic, or Anxiolytic Intoxication; and Comorbidity, see the corresponding sections in Sedative, Hypnotic, or Anxiolytic Use Disorder.

Diagnostic Features

The essential feature of sedative, hypnotic, or anxiolytic intoxication is the presence of clinically significant maladaptive behavioral or psychological changes (e.g., inappropriate sexual or aggressive behavior, mood lability, impaired judgment, impaired social or occupational functioning) that develop during, or shortly after, use of a sedative, hypnotic, or anxiolytic (Criteria A and B). As with other brain depressants, such as alcohol, these behaviors may be accompanied by slurred speech, incoordination (at levels that can interfere with driving abilities and with performing usual activities to the point of causing falls or automobile accidents), an unsteady gait,

nystagmus, impairment in cognition (e.g., attentional or memory problems), and stupor or coma (Criterion C). Memory impairment is a prominent feature of sedative, hypnotic, or anxiolytic intoxication and is most often characterized by an anterograde amnesia that resembles "alcoholic blackouts," which can be disturbing to the individual. The symptoms must not be attributable to another medical condition and are not better explained by another mental disorder (Criterion D). Intoxication may occur in individuals who are receiving these substances by prescription, are borrowing the medication from friends or relatives, or are deliberately taking the substance to achieve intoxication. Because sedatives, hypnotics, and anxiolytics are often used in combination with other psychoactive substances, it can be difficult to ascertain whether the functional consequences are attributable to a sedative, hypnotic, or anxiolytic or to the use of multiple substances.

Associated Features

Associated features include taking more medication than prescribed, taking multiple different medications, or mixing sedative, hypnotic, or anxiolytic agents with alcohol, which can markedly increase the effects of these agents.

Prevalence

The prevalence of sedative, hypnotic, or anxiolytic intoxication in the general population is unknown. However, it is probable that most nonmedical users of sedatives, hypnotics,

or anxiolytics would at some time have signs or symptoms that meet criteria for sedative, hypnotic, or anxiolytic intoxication; if so, then the prevalence of nonmedical sedative, hypnotic, or anxiolytic use in the general population may be similar to the prevalence of sedative, hypnotic, or anxiolytic intoxication. For example, in 2018, tranquilizers or sedative were used nonmedically in the United States by 2.4% of individuals age 12 or older and 4.9% of those ages 18–25.

Differential Diagnosis

Alcohol use disorder. Because the clinical presentations may be identical, distinguishing sedative, hypnotic, or anxiolytic intoxication from alcohol use disorder requires evidence for recent ingestion of sedative, hypnotic, or anxiolytic medications by self-report, informant report, or toxicological testing. Many individuals who misuse sedatives, hypnotics, or anxiolytics may also misuse alcohol and other substances, and so multiple intoxication diagnoses are possible.

Alcohol intoxication. Alcohol intoxication may be distinguished from sedative, hypnotic, or anxiolytic intoxication by the smell of alcohol on the breath. Otherwise, the features of the two disorders may be similar.

Sedative-, hypnotic-, or anxiolytic-induced mental disorders. Sedative, hypnotic, or anxiolytic intoxication is distinguished from sedative-, hypnotic-, or anxiolytic-induced mental disorders (e.g., sedative-, hypnotic-, or anxiolytic-induced anxiety disorder, with onset during withdrawal) because the symptoms (e.g., anxiety) in the latter disorders are in excess of those usually associated with sedative, hypnotic, or anxiolytic intoxication; predominate in the clinical presentation; and are severe enough to warrant clinical attention.

Neurocognitive disorders. In situations of cognitive impairment, traumatic brain injury, and delirium from other causes, sedatives, hypnotics, or anxiolytics may be intoxicating at quite low dosages. The differential diagnosis in these complex settings

is based on the predominant syndrome. An additional diagnosis of sedative, hypnotic, or anxiolytic intoxication may be appropriate even if the substance has been ingested at a low dosage in the setting of these other (or similar) co-occurring conditions.

Comorbidity

Given the typical overlap of sedative, hypnotic, or anxiolytic intoxication with sedative, hypnotic, or anxiolytic use disorder, see "Comorbidity" under Sedative, Hypnotic, or Anxiolytic Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Sedative, Hypnotic, or Anxiolytic Withdrawal

Diagnostic Criteria

A. Cessation of (or reduction in) sedative, hypnotic, or anxiolytic use that has been prolonged.

B. Two (or more) of the following, developing within several hours to a few days after the cessation of (or reduction in) sedative, hypnotic, or anxiolytic use described in Criterion A:

1. Autonomic hyperactivity (e.g., sweating or pulse rate greater than 100 bpm).
2. Hand tremor.

3. Insomnia.
4. Nausea or vomiting.
5. Transient visual, tactile, or auditory hallucinations or illusions.
6. Psychomotor agitation.
7. Anxiety.
8. Grand mal seizures.

C. The signs or symptoms in Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication or withdrawal from another substance.

Specify if:

With perceptual disturbances: This specifier may be noted when hallucinations with intact reality testing or auditory, visual, or tactile illusions occur in the absence of a delirium.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid sedative, hypnotic, or anxiolytic use disorder and whether or not there are perceptual disturbances.

For sedative, hypnotic, or anxiolytic withdrawal, without perceptual disturbances: If a mild sedative, hypnotic, or anxiolytic use disorder is comorbid, the ICD-10-CM code is **F13.130**, and if a moderate or severe sedative, hypnotic, or anxiolytic use disorder is comorbid, the ICD-10-CM code is **F13.230**. If there is no comorbid

sedative, hypnotic, or anxiolytic use disorder (e.g., in a patient taking sedatives, hypnotics, or anxiolytics solely under appropriate medical supervision), then the ICD-10-CM code is **F13.930**.

For sedative, hypnotic, or anxiolytic withdrawal, with perceptual disturbances: If a mild sedative, hypnotic, or anxiolytic use disorder is comorbid, the ICD-10-CM code is **F13.132**, and if a moderate or severe sedative, hypnotic, or anxiolytic use disorder is comorbid, the ICD-10-CM code is **F13.232**. If there is no comorbid sedative, hypnotic, or anxiolytic use disorder (e.g., in a patient taking sedatives, hypnotics, or anxiolytics solely under appropriate medical supervision), then the ICD-10-CM code is **F13.932**.

Note: For information on Development and Course; Risk and Prognostic Factors; Culture-Related Diagnostic Issues; Functional Consequences of Sedative, Hypnotic, or Anxiolytic Withdrawal; and Comorbidity, see the corresponding sections in Sedative, Hypnotic, or Anxiolytic Use Disorder.

Diagnostic Features

The essential feature of sedative, hypnotic, or anxiolytic withdrawal is the presence of a characteristic syndrome that develops after a marked decrease in or cessation of intake after several weeks or more of regular use (Criteria A and B). This withdrawal syndrome is characterized by two or more symptoms (similar to alcohol withdrawal) that include autonomic hyperactivity (e.g., increases in heart rate, respiratory rate, blood pressure, or body temperature, along with sweating); a tremor of the hands; insomnia; nausea, sometimes accompanied by vomiting; anxiety; and psychomotor agitation. A grand mal seizure may occur in perhaps as many as 20%–30% of individuals undergoing untreated withdrawal from these substances. In severe withdrawal, visual, tactile, or auditory hallucinations or illusions can occur but are usually in the context of a withdrawal delirium. If the individual's reality testing is intact (i.e., knows the substance is causing the hallucinations) and the illusions occur in a clear sensorium, the specifier "with perceptual disturbances"

can be noted. When hallucinations occur in the absence of intact reality testing, a diagnosis of substance/medication-induced psychotic disorder should be considered. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C). The symptoms must not be attributable to another medical condition and are not better explained by another mental disorder (e.g., alcohol withdrawal, generalized anxiety disorder) (Criterion D). Relief of withdrawal symptoms with administration of any sedative-hypnotic agent would support a diagnosis of sedative, hypnotic, or anxiolytic withdrawal.

Associated Features

The timing and severity of the withdrawal syndrome will differ depending on the specific substance and its pharmacokinetics and pharmacodynamics. For example, withdrawal from shorter-acting substances that are rapidly absorbed and that have no active metabolites (e.g., triazolam) can begin within hours after the substance is stopped; withdrawal from substances with long-acting metabolites (e.g., diazepam) may not begin for 1–2 days or longer. The withdrawal syndrome produced by substances in this class may be characterized by the development of a delirium that can be life-threatening. There may be evidence of tolerance and withdrawal in the absence

of a diagnosis of a benzodiazepine use disorder in an individual who has abruptly discontinued benzodiazepines that were taken for long periods of time at prescribed and therapeutic doses.

The time course of the withdrawal syndrome is generally predicted by the half-life of the substance. Medications whose actions typically last about 10 hours or less (e.g., lorazepam, oxazepam, temazepam) produce withdrawal symptoms within 6–8 hours of decreasing blood levels that peak in intensity on the second day and improve markedly by the fourth or fifth day. For substances with longer half-lives (e.g., diazepam), symptoms may not develop for more than 1 week, peak in intensity during the second week, and decrease markedly during the third or fourth week. There may be additional longer-term symptoms at a much lower level of intensity that persist for several months.

The longer the substance has been taken and the higher the dosages used, the more likely there will be severe withdrawal. However, withdrawal has been reported with as little as 15 mg of diazepam (or its equivalent in other benzodiazepines) when taken daily for several months. Doses of approximately 40 mg of diazepam (or its equivalent) daily are more likely to produce clinically relevant withdrawal symptoms, and even higher doses (e.g., 100 mg of diazepam) are more likely to be followed by withdrawal seizures or delirium. Sedative, hypnotic, or anxiolytic withdrawal delirium is characterized by disturbances in consciousness and cognition, with visual, tactile, or auditory hallucinations. When present, sedative, hypnotic, or anxiolytic withdrawal delirium should be diagnosed instead of withdrawal.

Prevalence

The prevalence of sedative, hypnotic, or anxiolytic withdrawal is unknown.

Diagnostic Markers

Seizures and autonomic instability in the setting of a history of prolonged exposure to sedative, hypnotic, or anxiolytic medications suggest a high likelihood of sedative, hypnotic, or anxiolytic withdrawal.

Differential Diagnosis

Other medical conditions. The symptoms of sedative, hypnotic, or anxiolytic withdrawal may be mimicked by other medical conditions (e.g., hypoglycemia, diabetic ketoacidosis). If seizures are a feature of the sedative, hypnotic, or anxiolytic withdrawal, the

differential diagnosis includes the various causes of seizures (e.g., infections, head injury, poisonings).

Essential tremor. Essential tremor, a neurological condition that frequently runs in families, may erroneously suggest the tremulousness associated with sedative, hypnotic, or anxiolytic withdrawal.

Alcohol withdrawal. Alcohol withdrawal produces a syndrome very similar to that of sedative, hypnotic, or anxiolytic withdrawal. The differential diagnosis is determined mostly through clinical history, although liver damage and other potential signs of chronic alcohol toxicity (e.g., cardiomyopathy) can also be more suggestive of alcohol withdrawal than of sedative, hypnotic, or anxiolytic withdrawal.

Sedative-, hypnotic-, or anxiolytic-induced mental disorders. Sedative, hypnotic, or anxiolytic withdrawal is distinguished from sedative-, hypnotic-, or anxiolytic-induced mental disorders (e.g., sedative-, hypnotic-, or anxiolytic-induced anxiety disorder,

with onset during withdrawal) because the symptoms (e.g., anxiety) in the latter disorders are in excess of those usually associated with sedative, hypnotic, or anxiolytic withdrawal; predominate in the clinical presentation; and are severe enough to warrant clinical attention.

Anxiety disorders. Recurrence or worsening of an underlying anxiety disorder produces a syndrome similar to sedative, hypnotic, or anxiolytic withdrawal, although the most extreme manifestations of withdrawal, such as delirium tremens or true seizures, are not symptoms of any anxiety disorder. Withdrawal would be suspected with an abrupt reduction in the dosage of a sedative, hypnotic, or anxiolytic medication. When a taper is under way, distinguishing the withdrawal syndrome from the underlying anxiety disorder can be difficult. As with alcohol, lingering withdrawal symptoms (e.g., anxiety, moodiness, trouble sleeping) can be mistaken for independent anxiety or depressive disorders (e.g., generalized anxiety disorder).

Comorbidity

Given the typical overlap of sedative, hypnotic, or anxiolytic withdrawal with sedative, hypnotic, or anxiolytic use disorder, see "Comorbidity" under Sedative, Hypnotic, or Anxiolytic Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Sedative-, Hypnotic-, or Anxiolytic-Induced Mental Disorders

The following sedative-, hypnotic-, or anxiolytic-induced mental disorders are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): sedative-, hypnotic-, or anxiolytic-induced psychotic disorder ("Schizophrenia Spectrum and Other Psychotic Disorders"); sedative-, hypnotic-, or anxiolytic-induced bipolar and related disorder ("Bipolar and Related Disorders"); sedative-, hypnotic-, or anxiolytic-induced depressive disorder ("Depressive Disorders"); sedative-, hypnotic-, or anxiolytic-induced anxiety disorder ("Anxiety Disorders"); sedative-, hypnotic-, or anxiolytic-induced sleep disorder ("Sleep-Wake Disorders"); sedative-, hypnotic-, or anxiolytic-induced sexual dysfunction ("Sexual Dysfunctions"); and sedative-, hypnotic-, or anxiolytic-induced major or mild neurocognitive disorder ("Neurocognitive Disorders"). For sedative, hypnotic, or anxiolytic intoxication delirium; sedative, hypnotic, or anxiolytic withdrawal delirium; and delirium induced by sedatives, hypnotics, or anxiolytics taken as prescribed, see the

criteria and discussion of delirium in the chapter "Neurocognitive Disorders." These sedative-, hypnotic-, or anxiolytic-induced mental disorders are diagnosed instead of sedative, hypnotic, or anxiolytic intoxication or sedative, hypnotic, or anxiolytic withdrawal only when the symptoms are sufficiently severe to warrant independent clinical attention.

Unspecified Sedative-, Hypnotic-, or Anxiolytic-Related Disorder

F13.99

This category applies to presentations in which symptoms characteristic of a sedative-, hypnotic-, or anxiolytic-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific sedative-, hypnotic-, or anxiolytic-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Stimulant-Related Disorders

Stimulant Use Disorder

Stimulant Intoxication

Stimulant Withdrawal

Stimulant-Induced Mental Disorders

Unspecified Stimulant-Related Disorder

Stimulant Use Disorder

Diagnostic Criteria

A. A pattern of amphetamine-type substance, cocaine, or other stimulant use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. The stimulant is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control stimulant use.
3. A great deal of time is spent in activities necessary to obtain the stimulant, use the stimulant, or recover from its effects.
4. Craving, or a strong desire or urge to use the stimulant.
5. Recurrent stimulant use resulting in a failure to fulfill major role obligations at work, school, or home.
6. Continued stimulant use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the stimulant.
7. Important social, occupational, or recreational activities are given up or reduced because of stimulant use.

8. Recurrent stimulant use in situations in which it is physically hazardous.
9. Stimulant use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the stimulant.
10. Tolerance, as defined by either of the following:

a. A need for markedly increased amounts of the stimulant to achieve intoxication or desired effect.
b. A markedly diminished effect with continued use of the same amount of the stimulant.

Note: This criterion is not considered to be met for those taking stimulant medications solely under appropriate medical supervision, such as medications for attention-deficit/hyperactivity disorder or narcolepsy.

11. Withdrawal, as manifested by either of the following:
    a. The characteristic withdrawal syndrome for the stimulant (refer to Criteria A and B of the criteria set for stimulant withdrawal).
    b. The stimulant (or a closely related substance) is taken to relieve or avoid withdrawal symptoms.

    Note: This criterion is not considered to be met for those taking stimulant medications solely under appropriate medical supervision, such as medications for attention-deficit/hyperactivity disorder or narcolepsy.

Specify if:

In early remission: After full criteria for stimulant use disorder were previously met, none of the criteria for stimulant use disorder have been met for at least 3 months but for less than 12 months (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the stimulant,” may be met).

In sustained remission: After full criteria for stimulant use disorder were previously met, none of the criteria for stimulant use disorder have been met at any time during a period of 12 months or longer (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the stimulant,” may be met).

Specify if:

In a controlled environment: This additional specifier is used if the individual is in an environment where access to stimulants is restricted.

Code based on current severity/remission: If an amphetamine-type substance intoxication, amphetamine-type substance withdrawal, or amphetamine-type substance-induced mental disorder is also present, do not use the codes below for amphetamine-type substance use disorder. Instead, the comorbid amphetamine-type substance use disorder is indicated in the 4th character of the amphetamine-type substance-induced disorder code (see the coding note for amphetamine-type substance intoxication, amphetamine-type substance withdrawal, or a specific amphetamine-type substance-induced mental disorder). For example, if there is comorbid amphetamine-induced depressive disorder and amphetamine use disorder, only the amphetamine-induced depressive disorder code is given, with the 4th character indicating whether the comorbid amphetamine use disorder is mild, moderate, or severe: F15.14 for mild amphetamine use disorder with amphetamine-induced depressive disorder or F15.24 for a moderate or severe amphetamine use disorder with amphetamine-induced depressive disorder. (The instructions for amphetamine-type substance also apply to other or unspecified stimulant intoxication, other or unspecified stimulant withdrawal, and other or unspecified stimulant-induced mental disorder.) Similarly, if there is comorbid cocaine-induced depressive disorder and cocaine use disorder,

only the cocaine-induced depressive disorder code is given, with the 4th character indicating whether the comorbid cocaine use disorder is mild, moderate,

or severe: F14.14 for a mild cocaine use disorder with cocaine-induced depressive disorder or F14.24 for a moderate or severe cocaine use disorder with cocaine-induced depressive disorder.

Specify current severity/remission:

Mild: Presence of 2–3 symptoms.

F15.10 Amphetamine-type substance

F14.10 Cocaine

F15.10 Other or unspecified stimulant

Mild, In early remission

F15.11 Amphetamine-type substance

F14.11 Cocaine

F15.11 Other or unspecified stimulant

Mild, In sustained remission

F15.11 Amphetamine-type substance

F14.11 Cocaine

F15.11 Other or unspecified stimulant

Moderate: Presence of 4–5 symptoms.

F15.20 Amphetamine-type substance

F14.20 Cocaine

F15.20 Other or unspecified stimulant

Moderate, In early remission

F15.21 Amphetamine-type substance

F14.21 Cocaine

F15.21 Other or unspecified stimulant

Moderate, In sustained remission

F15.21 Amphetamine-type substance

F14.21 Cocaine

F15.21 Other or unspecified stimulant

Severe: Presence of 6 or more symptoms.

F15.20 Amphetamine-type substance

F14.20 Cocaine

F15.20 Other or unspecified stimulant

Severe, In early remission

F15.21 Amphetamine-type substance

F14.21 Cocaine

F15.21 Other or unspecified stimulant

Severe, In sustained remission

F15.21 Amphetamine-type substance

F14.21 Cocaine

F15.21 Other or unspecified stimulant

Specifiers

"In a controlled environment" applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

Diagnostic Features

Stimulants are a type of psychoactive substance that increases activity in the brain and can temporarily elevate alertness, mood, and awareness. Stimulants covered in this chapter include amphetamine and prescription stimulants with similar effects (e.g., methylphenidate) and cocaine. Substance-related disorders involving certain other substances with stimulant properties are classified in other sections of this chapter. These include caffeine (in caffeine-related disorders), nicotine (in tobacco-related disorders), and MDMA (3,4-methylenedioxymethamphetamine; in other hallucinogen-related disorders), which has both stimulant and hallucinogenic effects.

Given that the effects of amphetamine-type substances are similar to those of cocaine, amphetamine-related disorders and cocaine-related disorders are grouped under the single rubric "stimulant-related disorders." Amphetamine-type substances (and other or unspecified stimulants) and cocaine have different ICD-10-CM codes (e.g., F15.10 mild amphetamine-type substance use disorder, F14.10 mild cocaine use disorder). The particular stimulant used by the individual is recorded in the diagnosis (e.g., "methamphetamine withdrawal," "methylphenidate use disorder," "cocaine intoxication").

The amphetamine-type substances include stimulants with a substituted phenylethylamine structure, such as amphetamine, dextroamphetamine, and methamphetamine. Also included are substances that are structurally different but have similar effects, such as methylphenidate, modafinil, and armodafinil. These amphetamine-type substances are usually taken orally or intravenously, although methamphetamine is also taken by the nasal route. In addition to the synthetic amphetamine-type compounds, there are naturally occurring, plant-derived stimulants such as *khât*, as well as synthetic chemical *khât* analogs, called *cathinones*.

Amphetamines and other stimulants may be obtained by prescription for the treatment of obesity, attention-deficit/hyperactivity disorder, and narcolepsy. Consequently, prescribed stimulants may be diverted into the illegal market.

Cocaine, a naturally occurring substance produced by the coca plant, is consumed in several preparations (e.g., coca leaves, coca paste, cocaine hydrochloride, and cocaine alkaloids such as freebase and crack) that differ in potency because of varying levels of purity and speed of onset. However, in all of the forms, cocaine is the active ingredient. Cocaine hydrochloride powder is usually "snorted" through the nostrils or dissolved in water and injected intravenously. Crack and other cocaine alkaloids are easily vaporized and inhaled, and thus their effects have an extremely rapid onset.

Individuals exposed to amphetamine-type substances or cocaine can develop stimulant use disorder as rapidly as 1 week, although the onset is not always this rapid. Regardless of the route of administration, tolerance occurs with repeated use. Withdrawal symptoms, particularly hypersomnia, increased appetite, and dysphoria, can occur and can enhance craving. Most individuals with stimulant use disorder have experienced tolerance or withdrawal.

Use patterns and course are similar for disorders involving amphetamine-type substances and cocaine, as both are potent central nervous system stimulants with similar psychoactive and sympathomimetic effects. Amphetamine-type substances are longer acting than cocaine and thus are used fewer times per day. Usage may be chronic or episodic, with binges punctuated by brief non-use periods. Aggressive or violent behavior is common when high doses are smoked, ingested, or administered intravenously. Intense temporary anxiety resembling panic disorder or generalized anxiety disorder, as well as paranoid ideation and psychotic episodes that resemble schizophrenia, is seen with high-dose use.

Withdrawal states are associated with temporary but intense depressive symptoms that can resemble a major depressive episode; the depressive symptoms usually resolve

within 1 week. Tolerance to amphetamine-type substances develops and leads to escalation of the dose. Conversely, some users of amphetamine-type substances develop sensitization, characterized by enhanced effects.

Associated Features

When injected or smoked, stimulants typically produce an instant feeling of well-being, confidence, and euphoria. Dramatic behavioral changes can rapidly develop with stimulant use disorder. Chaotic behavior, social isolation, aggressive behavior, and sexual dysfunction can result from long-term stimulant use disorder.

Individuals with acute intoxication may present with rambling speech, headache, transient ideas of reference, and tinnitus. There may be paranoid ideation, auditory hallucinations in a clear sensorium, and tactile hallucinations, which the individual usually recognizes as drug effects. Threats or acting out of aggressive behavior may occur. Depression, suicidal thoughts, irritability, anhedonia, emotional lability, or disturbances in attention and concentration commonly occur during withdrawal. Mental disturbances associated with cocaine use usually resolve hours to days after cessation of use but can persist for 1 month. Physiological changes during stimulant withdrawal are opposite to those of the intoxication phase, sometimes including bradycardia. Temporary depressive symptoms may meet symptomatic and duration criteria for major depressive episode. Histories consistent with repeated panic attacks, social anxiety disorder–like behavior, and generalized anxiety–like syndromes are common, as are eating disorders. One extreme instance of stimulant toxicity is stimulant-induced psychotic disorder, a disorder that resembles schizophrenia, with delusions and hallucinations.

Individuals with stimulant use disorder often develop conditioned responses to drug-related stimuli (e.g., craving on seeing any white powderlike substance). These responses contribute to relapse, are difficult to extinguish, and persist after detoxification.

Depressive symptoms with suicidal thoughts or behavior can occur and are generally the most serious problems seen during stimulant withdrawal.

Prevalence

Stimulant use disorder: amphetamine-type substances. Estimated 12-month prevalence of amphetamine-type substance use disorder in the United States is 0.4% among individuals 12 years and older. Twelve-month prevalence is 0.1% among individuals ages 12–17 years, 0.5% among those ages 18–25, and 0.4% among those age 26 and older. Rates are 0.5% for men and 0.2% for women, overall. Rates are approximately 0.4% among Hispanics and non-Hispanic Whites and 0.1% among African Americans and Asian Americans. Prevalence estimates for American Indian/Alaskan Natives and

Native Hawaiian/Pacific Islander populations are difficult to determine, given small sample sizes, but there is some evidence for higher rates in American Indians/Alaskan Natives.

Among U.S. adults, 6.6% (annual average) used prescription stimulants overall; 4.5% used without misuse, 1.9% misused without use disorders, and 0.2% had use disorders. While non-Hispanic Whites are more likely to use prescription stimulants nonmedically, Hispanics tend to use them more frequently and have higher rates of prescription stimulant use disorder.

Stimulant use disorder: cocaine. Estimated 12-month prevalence of cocaine use disorder in the United States is 0.4% among individuals 12 years and older. Rates are 0.1% among individuals ages 12–17 years, 0.7% among those ages 18–25 years, and 0.3% among those age 26 and older. Rates are 0.5% for men and 0.2% for women, overall. Rates are 0.4% among African Americans and non-Hispanic Whites, 0.3% in Hispanics, and < 0.1% among Asian Americans.

Development and Course

In the United States, stimulant use disorder occurs throughout all levels of society and is more common among individuals ages 18–25 years compared with individuals ages 12–17 or 26 years and older. On average, first regular use among individuals in treatment occurs at approximately age 23 years. For primary methamphetamine treatment admissions, the average age is 34 years, and for primary cocaine treatment admissions, the average age is 44 years for smoked cocaine and 37 years for other routes.

Some persons begin stimulant use to control weight or to improve performance in school, work, or athletics. Initial use may include obtaining medications such as methylphenidate or amphetamine salts prescribed to others for the treatment of attention-deficit/hyperactivity disorder. Among primary treatment admissions for amphetamine-type substance use in the United States, 61% reported smoking, 26% reported injecting, and 9% reported snorting, suggesting that stimulant use disorder can develop from multiple modes of administration.

Patterns of stimulant administration include episodic or daily (or almost daily) use. Episodic use (e.g., intense use over a weekend or on one or more weekdays) tends to be separated by 2 or more days of nonuse. “Binges” involve continuous high-dose use over hours or days and are often associated with physical dependence. Binges usually terminate only when stimulant supplies are depleted or exhaustion ensues. Chronic daily use may involve high or low doses, often with an increase in dose over time.

Stimulant smoking and intravenous use are associated with rapid progression to severe-level stimulant use disorder, often occurring over weeks to months. Intranasal use of cocaine and oral use of amphetamine-type substances result in more gradual progression occurring over months to years. With continued use, there is a diminution of pleasurable effects because of tolerance and an increase in dysphoric effects.

Risk and Prognostic Factors

Temperamental. Comorbid bipolar disorder, schizophrenia, antisocial personality disorder, and other substance use disorders are risk factors for developing stimulant use disorder and for relapse to cocaine use in treatment samples. Higher stress reactivity has been correlated with frequency of cocaine use in some U.S. treatment samples. Conduct disorder in childhood and antisocial personality disorder are associated with the development of stimulant-related disorders. In the United States, previous use of another substance, being male, having a Cluster B personality disorder, family history of substance use disorder, and being separated, divorced, or widowed all result in

increased risk of using cocaine. Men who have sex with men are also at higher risk for methamphetamine use.

Environmental. Predictors of cocaine use among a cohort of U.S. teenagers include prenatal cocaine exposure, postnatal cocaine use by parents, and exposure to community violence during childhood. Research in industrialized countries suggests that exposure to intimate partner violence or childhood mistreatment often co-occurs with stimulant use, especially in women. In a cohort of U.S. women followed up longitudinally, socioeconomic status, including food insecurity, had a dose-dependent effect on risk of stimulant use. For youth, especially girls, risk factors include living in an unstable home environment, having a psychiatric condition, criminal behavior, and associating with dealers and users.

Culture-Related Diagnostic Issues

The prevalence of cocaine use in the United States increased between 2001–2002 and 2012–2013 among non-Latinx Whites, African Americans, and Latinx, but the prevalence of cocaine use disorder increased only among Whites. Despite small variations, cocaine and other stimulant use disorder diagnostic criteria perform equally across gender and

ethnoracial groups. In limited data on prevalence estimates, it appears that American Indian/Alaskan Native populations are at higher risk for methamphetamine use disorder, and, to a lesser degree, cocaine use disorder, than are non-Hispanic Whites, while native Hawaiian/Pacific Islanders appear to have similar risks to non-Hispanic Whites.

Approximately 64% of individuals admitted to publicly funded substance abuse treatment programs for primary methamphetamine/amphetamine-related disorders are non-Hispanic White, followed by 20% of Hispanic origin, 3% Asian and Pacific Islander, and 6% non-Hispanic Black. Among individuals admitted for primary treatment related to smoked cocaine, 51% were non-Hispanic Black, 35% non-Hispanic White, 8% Hispanic, and 1% Asian/Pacific Islander. For admissions related to other routes of cocaine administration, 47% were non-Hispanic White, 31% were non-Hispanic Black, 17% were of Hispanic origin, and 1% were Asian/Pacific Islander. Rates of disorders in clinical samples should be interpreted with caution because they may be affected by differential access to and utilization of services, pathways to care, criminalization, stigma, and racial bias in diagnosis and referral for treatment.

Sex- and Gender-Related Diagnostic Issues

In the United States, women with cocaine use disorder more frequently have comorbid psychiatric disorders, such as depression and posttraumatic stress disorder (PTSD), compared with men. Gonadal hormones affect a male's responses to cocaine. Females with cocaine use disorder and higher levels of progesterone have lower stress-induced and cue-induced cocaine craving and lower cue-induced changes in blood pressure than females with cocaine use disorder and lower levels of progesterone. This may explain why use of cocaine in pregnant females is lower than in nonpregnant females.

Diagnostic Markers

Benzoylecgonine, a metabolite of cocaine, typically remains in the urine for 1–3 days after a single dose and may be present for 7–12 days in individuals using repeated high doses. Mildly elevated liver function tests can be present in cocaine injectors or users with concomitant alcohol use. There are no neurobiological markers of diagnostic

utility. Discontinuation of chronic cocaine use may be associated with electroencephalographic changes, suggesting persistent abnormalities; alterations in secretion patterns of prolactin; and downregulation of dopamine receptors.

Short-half-life amphetamine-type substances (e.g., methamphetamine) can be detected for 1–3 days, and possibly up to 4 days depending on dosage and metabolism. Hair samples can be used to detect presence of amphetamine-type substances for up to 90 days. Other laboratory findings, as well as physical findings and other medical conditions (e.g., weight loss, malnutrition; poor hygiene), are similar for both cocaine and amphetamine-type substance use disorder.

Association With Suicidal Thoughts or Behavior

Few data on the association of stimulant use disorders and suicide are available because most studies examining suicidal thoughts and behavior examine use of stimulants rather than stimulant use disorders. One systematic review found that regular or problem amphetamine use (examining primarily individuals who inject amphetamines and/or individuals admitted to treatment for use of amphetamines) is associated with increased suicide mortality. A general population study of adults in the United States found an association of prescription stimulant use disorder with suicidal thoughts. In a study of individuals admitted to substance use treatment, those with cocaine use disorder were much more likely to report suicidal thoughts than those with other substance use disorders. In a study of both men and women in the U.S. Veterans Administration health care system,

cocaine and amphetamine use disorders were each associated with increased rates of suicide deaths.

Functional Consequences of Stimulant Use Disorder

Various medical conditions may occur depending on the route of administration. Intranasal users often develop sinusitis, irritation, bleeding of the nasal mucosa, and a perforated nasal septum. Individuals who smoke stimulants are at increased risk for respiratory problems (e.g., coughing, bronchitis, and pneumonitis). Injectors have puncture marks and “tracks,” most commonly on their forearms. Risk of HIV and hepatitis C infection increases with frequent intravenous injections and unsafe sexual activity. Other sexually transmitted diseases, hepatitis B, and tuberculosis and other lung infections are also seen. Weight loss and malnutrition are common.

Chest pain may be a common symptom during stimulant intoxication. Myocardial infarction, palpitations and arrhythmias, sudden death from respiratory or cardiac arrest, and stroke have been associated with stimulant use among young and otherwise healthy individuals. Pneumothorax can result from performing Valsalva-like maneuvers done to better absorb inhaled smoke. Cocaine use is associated with irregularities in placental blood flow, abruptio placentae, premature labor and delivery, and an increased prevalence of infants with very low birth weights.

Individuals with stimulant use disorder may become involved in theft, prostitution, or drug dealing in order to acquire drugs or money for drugs. Traumatic injuries due to violent behavior are common among individuals trafficking drugs.

Neurocognitive impairment is common among both methamphetamine and cocaine users, including deficits related to attention, impulsivity, verbal learning/memory, working memory, and executive functioning. Transient psychosis and seizure have also been reported with chronic use of either cocaine or methamphetamine, possibly related to patterns of use or the exacerbation of preexisting vulnerabilities. Amphetamine use can cause toxic effects related to elevated body temperature, and there is some

evidence that chronic use causes neuroinflammation and neurotoxicity in dopaminergic neurons. Oral health problems include “meth mouth” with gum disease, tooth decay, and mouth sores related to the toxic effects of smoking the drug and to bruxism while intoxicated. Adverse pulmonary effects appear to be less common for amphetamine-type substances because they are smoked fewer times per day, although methamphetamine use is still associated with a risk of pulmonary arterial hypertension. Emergency department visits are common for stimulant-related mental disorder symptoms, injury, skin infections, and dental pathology. In the United States, diagnosis of a stimulant use disorder is associated with a 20% increase in 30-day readmission rates in assessment of follow-up after hospitalization for “any cause” (a standard measure of overall hospital quality of care).

Differential Diagnosis

Phencyclidine intoxication. Intoxication with phencyclidine (PCP or “angel dust”) or synthetic “designer drugs” such as mephedrone (known by different names, including “bath salts”) may cause a similar clinical picture and can only be distinguished from stimulant intoxication by the presence of cocaine or amphetamine-type substance metabolites in a urine or plasma sample.

Stimulant intoxication, stimulant withdrawal, and stimulant-induced mental disorders. Stimulant use disorder is differentiated from stimulant intoxication, stimulant withdrawal, and stimulant-induced mental disorders (e.g., stimulant-induced depressive disorder) in that stimulant use disorder describes a problematic pattern of stimulant use that involves impaired control over stimulant use, social impairment attributable to stimulant use, risky stimulant use (e.g., continued stimulant use despite medical complications), and pharmacological symptoms (the development of tolerance or withdrawal), whereas stimulant

intoxication, stimulant withdrawal, and stimulant-induced mental disorders describe psychiatric syndromes that occur in the context of heavy use. Stimulant intoxication, stimulant withdrawal, and stimulant-induced mental disorders occur frequently in individuals with stimulant use disorder. In such cases, a diagnosis of stimulant intoxication, stimulant withdrawal, or a stimulant-induced mental disorder should be given in addition to a diagnosis of stimulant use disorder, the presence of which is indicated in the diagnostic code.

Independent mental disorders. Some of the effects of stimulant use may resemble symptoms of independent mental disorders, such as psychosis (schizophrenia) and low mood (major depressive disorder). Discerning whether these behaviors occurred before the intake of the drug is important in the differentiation of acute drug effects from a preexisting mental disorder.

Comorbidity

Stimulant-related disorders often co-occur with other substance use disorders, especially those involving substances with sedative properties, which are often taken to reduce insomnia, nervousness, and other unpleasant side effects. Individuals admitted to treatment for cocaine use are likely to also use heroin, PCP, or alcohol, and individuals admitted for amphetamine-type substance use disorder are likely to use marijuana, heroin, or alcohol. Stimulant use disorder may be associated with posttraumatic stress disorder, antisocial personality disorder, attention-deficit/hyperactivity disorder, and gambling disorder. Cardiopulmonary problems are often present in individuals seeking treatment for cocaine-related problems, with chest pain being the most common. Medical problems occur in response to adulterants used

as “cutting” agents. Cocaine users who ingest cocaine cut with levamisole, an antimicrobial and veterinary medication, may experience agranulocytosis and febrile neutropenia.

Stimulant Intoxication

Diagnostic Criteria

A. Recent use of an amphetamine-type substance, cocaine, or other stimulant.

B. Clinically significant problematic behavioral or psychological changes (e.g., euphoria or affective blunting; changes in sociability; hypervigilance; interpersonal sensitivity; anxiety, tension, or anger; stereotyped behaviors; impaired judgment) that developed during, or shortly after, use of a stimulant.

C. Two (or more) of the following signs or symptoms, developing during, or shortly after, stimulant use:

1. Tachycardia or bradycardia.
2. Pupillary dilation.
3. Elevated or lowered blood pressure.
4. Perspiration or chills.
5. Nausea or vomiting.
6. Evidence of weight loss.
7. Psychomotor agitation or retardation.
8. Muscular weakness, respiratory depression, chest pain, or cardiac arrhythmias.
9. Confusion, seizures, dyskinesias, dystonias, or coma.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Specify **the particular intoxicant** (i.e., amphetamine-type substance, cocaine, or other stimulant).

Specify if:

With perceptual disturbances: This specifier may be noted when hallucinations with intact reality testing or auditory, visual, or tactile illusions occur in the absence of a delirium.

Coding note: The ICD-10-CM code depends on whether the stimulant is an amphetamine-type substance, cocaine, or other stimulant; whether there is a comorbid amphetamine-type substance, cocaine, or other stimulant use disorder; and whether or not there are perceptual disturbances.

For amphetamine-type substance, cocaine, or other stimulant intoxication, without perceptual disturbances: If a mild amphetamine-type substance or other stimulant use disorder is comorbid, the ICD-10-CM code is **F15.120,** and if a moderate or severe amphetamine-type substance or other stimulant use disorder is comorbid, the ICD-10-CM code is **F15.220.** If there is no comorbid

amphetamine-type substance or other stimulant use disorder, then the ICD-10-CM code is **F15.920.** Similarly, if a mild cocaine use disorder is comorbid, the ICD-10-CM code is **F14.120,** and if a moderate or severe cocaine use disorder is comorbid, the ICD-10-CM code is **F14.220.** If there is no comorbid cocaine use disorder, then the ICD-10-CM code is **F14.920.**

For amphetamine-type substance, cocaine, or other stimulant intoxication, with perceptual disturbances: If a mild amphetamine-type substance or other stimulant use disorder is comorbid, the ICD-10-CM code is **F15.122,** and if a moderate or severe amphetamine-type substance or other stimulant use disorder is comorbid, the ICD-10-CM code is **F15.222.** If there is no comorbid amphetamine-type substance or other stimulant use disorder, then the ICD-10-CM code is **F15.922.** Similarly, if a mild cocaine use disorder is comorbid, the ICD-10-CM code is **F14.122,** and if a moderate or severe cocaine use disorder is comorbid, the ICD-10-CM code is **F14.222.** If there is no comorbid cocaine use disorder, then the ICD-10-CM code is **F14.922.**

Diagnostic Features

The essential feature of stimulant intoxication, related to amphetamine-type substances and cocaine, is the presence of clinically significant behavioral or psychological changes that develop during, or shortly after, use of stimulants (Criteria A and B). Auditory hallucinations may be prominent, as may paranoid ideation, and these symptoms must be distinguished from an independent psychotic disorder such as schizophrenia. Stimulant intoxication usually begins with a “high” feeling and includes one or more of the following: euphoria with enhanced vigor, gregariousness, hyperactivity, restlessness, hypervigilance, interpersonal sensitivity, talkativeness, anxiety, tension, alertness, grandiosity, stereotyped and repetitive behavior, anger, impaired judgment, and, in the case of chronic intoxication, affective blunting with fatigue or sadness and social withdrawal. These behavioral and psychological changes are accompanied by two or more of the following signs and symptoms that develop during or shortly after stimulant use: tachycardia or bradycardia; pupillary dilation; elevated or lowered blood pressure; perspiration or chills; nausea or vomiting; evidence of weight loss; psychomotor agitation or retardation; muscular weakness, respiratory depression, chest pain, or cardiac arrhythmias; and confusion, seizures, dyskinesias, dystonias, or coma (Criterion C). Intoxication, either acute or chronic, is often associated with impaired social or occupational functioning. Severe intoxication can lead to convulsions, cardiac arrhythmias, hyperpyrexia, and death. For the diagnosis of stimulant intoxication to be made, the symptoms must not be attributable to another medical condition and are not better explained by another mental disorder

(Criterion D). While stimulant intoxication occurs in individuals with stimulant use disorders, intoxication is not a criterion for stimulant use disorder, which is confirmed by the presence of 2 of the 11 diagnostic criteria for use disorder.

Associated Features

The magnitude and direction of the behavioral and physiological changes depend on many variables, including the dose used and the characteristics of the individual using the substance or the context (e.g., tolerance, rate of absorption, chronicity of use, context in which taken). Stimulant effects such as euphoria, increased pulse and blood

pressure, and psychomotor activity are most commonly seen. Depressant effects such as sadness, bradycardia, decreased blood pressure, and decreased psychomotor activity are less common and generally emerge only with chronic high-dose use.

Prevalence

Although prevalence of stimulant intoxication is not known, prevalence of stimulant use can be used as a proxy. Many individuals who use stimulants may not have symptoms that fully meet the criteria for stimulant intoxication, which requires "clinically significant problematic behavioral or psychological changes." Thus, rates of stimulant use can be considered the upper bounds of the likely prevalence of stimulant intoxication.

Estimated 12-month prevalence of cocaine use in the United States is 2.2% for individuals age 12 and older (0.5% among individuals ages 12–17-years, 6.2% among individuals ages 18–25 years, and 1.7% among individuals age 26 and older); 3% of men/boys and 1.4% of women/girls used cocaine in the last 12 months. Twelve-month prevalence of cocaine use is 2.3% among Whites, 2.2% among Hispanics, 1.7% among African Americans, and 1% among Asian Americans.

Estimated 12-month prevalence of methamphetamine use in the United States is 0.6% for individuals age 12 and older (0.2% among individuals ages 12–17 years, 1.1% among individuals ages 18–25 years, and 0.6% among individuals age 26 and older). Twelve-month prevalence of methamphetamine use is 0.8% among men/boys and 0.4% among women/girls. Twelve-month prevalence of methamphetamine use is 0.7% among Whites, 0.6% among Hispanics, 0.2% among African Americans, and 0.1% among Asian Americans. Small sample sizes make estimating rates among American Indians/Alaskan Natives difficult.

Differential Diagnosis

Stimulant-induced mental disorders. Stimulant intoxication is distinguished from stimulant-induced mental disorders (e.g., stimulant-induced anxiety disorder, with onset during intoxication) because the symptoms (e.g., anxiety) in the latter disorders are in excess of those usually seen in stimulant intoxication, predominate in the clinical presentation, and meet full criteria for the relevant disorder.

Independent mental disorders. Salient mental disturbances associated with stimulant intoxication should be distinguished from the symptoms of schizophrenia, bipolar and depressive disorders, generalized anxiety disorder, and panic disorder as described in this manual.

Comorbidity

Given the typical overlap of stimulant intoxication with stimulant use disorder, see "Comorbidity" under Stimulant Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Stimulant Withdrawal

Diagnostic Criteria

A. Cessation of (or reduction in) prolonged amphetamine-type substance, cocaine, or other stimulant use.

B. Dysphoric mood and two (or more) of the following physiological changes, developing within a few hours to several days after Criterion A:

1. Fatigue.
2. Vivid, unpleasant dreams.
3. Insomnia or hypersomnia.
4. Increased appetite.
5. Psychomotor retardation or agitation.

C. The signs or symptoms in Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

D. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication or withdrawal from another substance.

Specify **the particular substance that causes the withdrawal syndrome** (i.e., amphetamine-type substance, cocaine, or other stimulant).

Coding note: The ICD-10-CM code depends on whether the stimulant is an amphetamine-type substance, cocaine, or other stimulant and on whether or not there is a comorbid amphetamine-type substance, cocaine, or other stimulant use disorder. If mild amphetamine-type substance or other stimulant use disorder is comorbid, the ICD-10-CM code is **F15.13**. If moderate or severe amphetamine-type substance or other stimulant use disorder is comorbid, the ICD-10-CM code is **F15.23**. For amphetamine-type substance or other stimulant withdrawal occurring in the absence of amphetamine-type substance or other stimulant use disorder (e.g., in a patient taking amphetamine solely under appropriate medical supervision), the ICD-10-CM code is **F15.93**. If mild cocaine use disorder is comorbid, the ICD-10-CM code is **F14.13**. If moderate or severe cocaine use disorder is comorbid, the ICD-10-CM code is **F14.23**. For cocaine withdrawal occurring in the absence of a cocaine use disorder, the ICD-10-CM code is **F14.93**.

Diagnostic Features

The essential feature of stimulant withdrawal is the presence of a characteristic withdrawal syndrome that develops within a few hours to several days after the cessation of (or marked reduction in) stimulant use (generally high dose) that has been prolonged (Criterion A). The withdrawal syndrome is characterized by the development of dysphoric mood accompanied by two or more of the following physiological changes: fatigue, vivid and unpleasant dreams, insomnia or hypersomnia, increased appetite, and psychomotor retardation or agitation (Criterion B). Bradycardia is often present and is a reliable measure of stimulant withdrawal.

Anhedonia and drug craving can often be present but are not part of the diagnostic criteria. These symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C). The symptoms must not be attributable to another medical condition and are not better explained by another mental disorder (Criterion D).

Associated Features

Acute withdrawal symptoms (“a crash”) are often seen after periods of repetitive high-dose use (“runs” or “binges”). These symptoms are characterized by intense and unpleasant feelings of lassitude and depression and increased appetite, generally requiring several days of rest and recuperation. Depressive symptoms with suicidal thoughts or behavior can occur and are generally the most serious problems seen during “crashing” or other forms of stimulant withdrawal. Many individuals with stimulant use disorder may experience a withdrawal syndrome at some point.

Differential Diagnosis

Stimulant-induced mental disorders. Stimulant withdrawal is distinguished from stimulant-induced mental disorders (e.g., stimulant-induced depressive disorder, with onset during withdrawal) because the symptoms (e.g., depressed mood) in these latter disorders are in excess of those usually associated with stimulant withdrawal, predominate in the clinical presentation, and are severe enough to warrant clinical attention.

Comorbidity

Given the typical overlap of stimulant withdrawal with stimulant use disorder, see “Comorbidity” under Stimulant Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Stimulant-Induced Mental Disorders

The following stimulant-induced mental disorders (which include amphetamine-type substance–, cocaine-, and other stimulant–induced mental disorders) are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): stimulant-induced psychotic disorder (“Schizophrenia Spectrum and Other Psychotic Disorders”); stimulant-induced bipolar and related disorder (“Bipolar and Related Disorders”); stimulant-induced depressive disorder (“Depressive Disorders”); stimulant-induced anxiety disorder (“Anxiety Disorders”); stimulant-induced obsessive-compulsive disorder (“Obsessive-Compulsive and Related Disorders”); stimulant-induced sleep disorder (“Sleep-Wake Disorders”); stimulant-induced sexual dysfunction (“Sexual Dysfunctions”); and stimulant-induced mild neurocognitive disorder (“Neurocognitive Disorders”). For stimulant intoxication delirium and delirium induced by stimulants taken as prescribed, see the criteria and discussion of delirium in the chapter “Neurocognitive Disorders.” These stimulant-induced mental disorders are diagnosed instead of stimulant intoxication or stimulant withdrawal only when the symptoms are sufficiently severe to warrant independent clinical attention.

Unspecified Stimulant-Related Disorder

This category applies to presentations in which symptoms characteristic of a stimulant-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific stimulant-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Coding note: The ICD-10-CM code depends on whether the stimulant is an amphetamine-type substance, cocaine, or other stimulant. The ICD-10-CM code for an unspecified amphetamine-type substance or other

stimulant–related disorder is **F15.99.** The ICD-10-CM code for an unspecified cocaine-related disorder is **F14.99.**

Tobacco-Related Disorders

Tobacco Use Disorder

Tobacco Withdrawal

Tobacco-Induced Mental Disorders

Unspecified Tobacco-Related Disorder

Tobacco Use Disorder

Diagnostic Criteria

A. A problematic pattern of tobacco use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. Tobacco is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control tobacco use.
3. A great deal of time is spent in activities necessary to obtain or use tobacco.
4. Craving, or a strong desire or urge to use tobacco.
5. Recurrent tobacco use resulting in a failure to fulfill major role obligations at work, school, or home (e.g., interference with work).
6. Continued tobacco use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of tobacco (e.g., arguments with others about tobacco use).
7. Important social, occupational, or recreational activities are given up or reduced because of tobacco use.
8. Recurrent tobacco use in situations in which it is physically hazardous (e.g., smoking in bed).
9. Tobacco use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by tobacco.
10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of tobacco to achieve the desired effect.
    b. A markedly diminished effect with continued use of the same amount of tobacco.
11. Withdrawal, as manifested by either of the following:
    a. The characteristic withdrawal syndrome for tobacco (refer to Criteria A and B of the criteria set for tobacco withdrawal).

b. Tobacco (or a closely related substance, such as nicotine) is taken to relieve or avoid withdrawal symptoms.

Specify if:

In early remission: After full criteria for tobacco use disorder were previously met, none of the criteria for tobacco use disorder have been met for at least 3 months but for less than 12 months (with the exception that Criterion A4, "Craving, or a strong desire or urge to use tobacco," may be met).

In sustained remission: After full criteria for tobacco use disorder were previously met, none of the criteria for tobacco use disorder have been met at any time during a period of 12 months or longer (with the exception that Criterion A4, "Craving, or a strong desire or urge to use tobacco," may be met).

Specify if:

On maintenance therapy: The individual is taking a long-term maintenance medication, such as nicotine replacement medication, and no criteria for tobacco use disorder have been met for that class of medication (except tolerance to, or withdrawal from, the nicotine replacement medication).

In a controlled environment: This additional specifier is used if the individual is in an environment where access to tobacco is restricted.

Code based on current severity/remission: If a tobacco withdrawal or tobacco-induced sleep disorder is also present, do not use the codes below for tobacco use disorder. Instead, the comorbid tobacco use disorder is indicated in the 4th character of the tobacco-induced disorder code (see the coding note for tobacco withdrawal or tobacco-induced sleep disorder). For example, if there is comorbid tobacco-induced sleep disorder and tobacco use disorder, only the tobacco-induced sleep disorder code is given, with the 4th character indicating whether the comorbid tobacco use disorder is moderate or severe: F17.208 for moderate or severe tobacco use disorder with tobacco-induced sleep disorder. It is not permissible to code a comorbid mild tobacco use disorder with a tobacco-induced sleep disorder.

Specify current severity/remission:

Z72.0 Mild: Presence of 2–3 symptoms.

F17.200 Moderate: Presence of 4–5 symptoms.

F17.201 Moderate, In early remission

F17.201 Moderate, In sustained remission

F17.200 Severe: Presence of 6 or more symptoms.

F17.201 Severe, In early remission

F17.201 Severe, In sustained remission

Specifiers

"On maintenance therapy" applies as a specifier to be added to "in remission" if the individual is both in remission and on maintenance therapy. "In a controlled environment" applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled

environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

Diagnostic Features

Tobacco use disorder can develop with use of all forms of tobacco (e.g., cigarettes, chewing tobacco, snuff, pipes, cigars, electronic nicotine delivery devices such as electronic cigarettes [e-cigarettes]) and with prescription nicotine-containing medications (nicotine gum and patch). The relative ability of these products to produce tobacco use disorder or to induce withdrawal is associated with the rapidity of the route of administration (smoked over oral over transdermal) and the nicotine content of the product. The name of this substance category was changed from "nicotine" in prior editions of DSM to "tobacco" in DSM-5 on the basis of harms from addiction being associated mostly with tobacco and much less with nicotine.

Tobacco use disorder is common among individuals who use cigarettes and smokeless tobacco daily, is less common among individuals who use e-cigarettes, and is uncommon

among those who do not use tobacco daily or use nicotine medications. Tolerance to tobacco is exemplified by the disappearance of nausea and dizziness after intake and by a more intense effect of tobacco the first time it is used during the day. Cessation of tobacco use can produce a well-defined withdrawal syndrome. Many individuals with tobacco use disorder use tobacco to relieve or to avoid withdrawal symptoms (e.g., after being in a situation where use is restricted). Many individuals with tobacco use disorder have tobacco-related physical symptoms or diseases and continue to smoke. The large majority report craving when they do not smoke for several hours. Spending excessive time using tobacco can be exemplified by chain-smoking (i.e., smoking one cigarette after another with no time between cigarettes). Because tobacco sources are readily and legally available, and because tobacco intoxication is very rare, spending a great deal of time attempting to procure tobacco or recovering from its effects is uncommon. Giving up important social, occupational, or recreational activities can occur when an individual forgoes an activity because it occurs in tobacco use–restricted areas. Use of tobacco rarely results in failure to fulfill major role obligations (e.g., interference with work or home responsibilities), but persistent social or interpersonal problems (e.g., having arguments with others about tobacco use, avoiding social situations because of others' disapproval of tobacco use) or use that is physically hazardous (e.g., smoking in bed, smoking around flammable chemicals) occur at an intermediate prevalence. Although these criteria are less often endorsed by tobacco users, if endorsed, they can indicate a more severe disorder.

Associated Features

Smoking within 30 minutes of waking, smoking daily, smoking more cigarettes per day, and waking at night to smoke are associated with tobacco use disorder. Environmental cues can evoke craving and withdrawal. Serious medical conditions often occur, including lung and other cancers, cardiac and pulmonary disease, perinatal problems, cough, shortness of breath, and accelerated skin aging.

Prevalence

Although cigarettes are the most commonly used tobacco product, use of other tobacco products (especially e-cigarettes) has become more common. In the United States, 19% of adults used a tobacco product in the last year, 19% used more than one product,

14% used cigarettes, 4% used cigars, 3% used e-cigarettes, and 2% used smokeless tobacco. One fourth (24%) of current U.S. smokers are nondaily smokers.

The 12-month prevalence of DSM-5 tobacco use disorder in the United States in 2012–2013 was 20% among adults age 18 years and older and 29.6% among Native Americans, 22.3% among non-Latinx Whites, 20.1% among African Americans, 12.2% among Latinx, and 11.2% among Asian Americans and Pacific Islanders. Prevalence was higher among men; those who were young, unmarried, less educated, poor, or residing in the southern United States; and those with almost any psychiatric disorder. The prevalence among current daily smokers is approximately 50%.

Global comparisons show that in all geographic regions of the world, the age-standardized prevalence of daily tobacco smoking is higher in men than in women, but the gender ratio varies greatly, from 16.9:1 in East Asia to 1.2:1 in Australasia.

Development and Course

About 20% of U.S. high school seniors report having ever smoked cigarettes, and about 5% have used in the past 30 days. Among adolescents who smoke cigarettes at least monthly, most of these individuals will become daily tobacco users in the future. Initiation of smoking after age 21 years is rare. Some of the tobacco use disorder criteria symptoms (e.g., craving) occur soon after beginning tobacco use, suggesting the addiction process begins with initial use; however, fulfilling DSM criteria usually occurs over several years.

Nondaily smoking has become more prevalent since the late 1990s in the United States, especially among individuals ages 18–34 years, Blacks, Hispanics, and individuals with at least a college education.

Risk and Prognostic Factors

Temperamental. Individuals with externalizing personality traits are more likely to initiate tobacco use. Children with attention-deficit/hyperactivity disorder or conduct disorder, and adults with depressive, bipolar, anxiety, personality, psychotic, or other substance use disorders, are at higher risk for starting and continuing tobacco use and of tobacco use disorder.

Environmental. Persons with low incomes and low educational levels are more likely to initiate tobacco use and are less likely to stop.

Genetic and physiological. Genetic factors contribute to the onset of tobacco use, the continuation of tobacco use, and the development of tobacco use disorder, with a degree of heritability equivalent to that observed with other substance use disorders (i.e., about 50%). Some of this risk is specific to tobacco, and some is common with the vulnerability to developing any substance use disorder.

Culture-Related Diagnostic Issues

Acceptance of tobacco use varies across cultural contexts. Age-standardized prevalence of daily tobacco smoking varies greatly by geographic region, ranging from 4.7% in Western Sub-Saharan Africa to 24.2% in Eastern Europe. The degree to which these geographic differences are the result of income, education, and tobacco control activities in a country is unclear. Prevalence of tobacco use in the United States varies by age, gender, and ethnoracial background, with lower rates of smoking onset and progression to daily smoking among Black youth, especially young women. Liver enzyme polymorphisms that vary across ethnoracial groups can affect nicotine metabolism, contributing to variation in smoking behavior. Higher tobacco use

disorder prevalence is also associated with exposure to racism and ethnic discrimination. Prevalence of DSM-IV nicotine dependence is higher among adult lesbian, gay, and bisexual individuals than among heterosexuals, possibly also due to an association with exposure to sexual orientation–related discrimination. Among individuals with DSM-IV nicotine dependence, lower income and education are associated with disorder persistence.

Sex- and Gender-Related Diagnostic Issues

The ratio of men to women among U.S. smokers is approximately 1.4:1 and has been stable between 2004 and 2014. This ratio is generally consistent across various income and educational levels. The ratio diminishes in older age groups as fewer men are smoking as age increases. The literature from several U.S. settings suggests that negative reinforcement (i.e., that smoking relieves negative affect) is a greater motivator in women than in men. Menstrual cycle effects on smoking are found inconsistently, but tobacco withdrawal appears worse in the luteal than the follicular phase of the cycle. Pregnant females smoke at a lower rate than nonpregnant females but relapse back to smoking rapidly after delivery.

Diagnostic Markers

The following biomarkers can be used to measure the extent of tobacco or nicotine use: carbon monoxide in the breath and nicotine and its metabolite cotinine in blood, saliva, or urine; however, these are only weakly associated with tobacco use disorder.

Association With Suicidal Thoughts or Behavior

National U.S. survey data show that past-year cigarette use is associated with a two- to threefold increased risk of suicidal thoughts and behavior, with earlier age at first tobacco use increasing risk. Evidence from the U.S. Veterans Health Administration shows that even after adjustment for covariates, tobacco use disorder is associated with an increased risk of suicide. A large study of twins in Finland found that the relationship between tobacco use and suicide increased in a dose-response manner, and that for identical twins discordant for tobacco use, tobacco use was associated with a sixfold increased risk for suicide.

Functional Consequences of Tobacco Use Disorder

Medical consequences of tobacco use often begin when tobacco users are in their 40s and usually become progressively more debilitating over time. One-half of smokers who do not stop using tobacco will die early from a tobacco-related illness, and smoking-related morbidity occurs in more than one-half of tobacco users. Most medical conditions result from exposure to carbon monoxide, tars, and other non-nicotine components of tobacco. The major predictor of reversibility is duration of smoking. Secondhand smoke increases the risk of heart disease and cancer by 30%. Long-term use of nicotine medications does not appear to cause medical harm.

Comorbidity

The most common medical conditions from smoking are cardiovascular illnesses, chronic obstructive pulmonary disease, and cancers. Smoking also increases perinatal problems, such as low birth weight and miscarriage. Prevalence of smoking is almost twice as high in individuals with major depressive disorder; although the prevalence of smoking in the United States is higher among individuals with low socioeconomic status, the increased prevalence of smoking among those with depression is

independent of socioeconomic status. The most common psychiatric comorbidities associated with smoking are alcohol and other substance, depressive, bipolar, anxiety, personality, and attention-deficit/hyperactivity disorders. In the United States, individuals with a psychiatric disorder are three times more likely than others to have tobacco use disorder. Adults with DSM-5 tobacco use disorder are significantly more likely than other adults to have comorbid psychiatric disorders, including other DSM-5 substance use disorders, major depressive disorder, bipolar I disorder, panic disorder, generalized anxiety disorder, posttraumatic stress disorder, and borderline and antisocial personality disorders.

Tobacco Withdrawal

Diagnostic Criteria F17.203

A. Daily use of tobacco for at least several weeks.
B. Abrupt cessation of tobacco use, or reduction in the amount of tobacco used, followed within 24 hours by four (or more) of the following signs or symptoms:
    1. Irritability, frustration, or anger.
    2. Anxiety.
    3. Difficulty concentrating.
    4. Increased appetite.
    5. Restlessness.
    6. Depressed mood.
    7. Insomnia.

C. The signs or symptoms in Criterion B cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
D. The signs or symptoms are not attributed to another medical condition and are not better explained by another mental disorder, including intoxication or withdrawal from another substance.

Coding note: The ICD-10-CM code for tobacco withdrawal is **F17.203**. Note that the ICD-10-CM code indicates the comorbid presence of a moderate or severe tobacco use disorder, reflecting the fact that tobacco withdrawal can only occur in the presence of a moderate or severe tobacco use disorder.

Diagnostic Features

Withdrawal symptoms impair the ability to stop tobacco use. The symptoms after abstinence from tobacco are in large part due to nicotine deprivation. Tobacco withdrawal is common among daily tobacco users who stop or reduce their use of tobacco. Symptoms are more intense among individuals who smoke cigarettes and also use smokeless tobacco or electronic cigarettes daily. This symptom intensity is likely attributable to the more rapid onset and higher levels of nicotine with cigarette smoking. Significant withdrawal among those who are nondaily cigarette users or use only nicotine medications is uncommon.

Typically, heart rate decreases by 5–12 bpm in the first few days after stopping smoking, and weight increases an average of 4–7 lb (2–3 kg) over the first year after stopping smoking. Tobacco withdrawal can produce clinically significant mood changes and functional impairment. Because of conditioning effects, withdrawal can be prompted by environmental cues such as seeing others smoking. Gradual reduction of tobacco decreases the severity of withdrawal.

Associated Features

Craving for tobacco or nicotine is very common during abstinence and has a large effect on the ability to remain abstinent. Abstinence can increase impulsivity and anhedonia and can decrease positive affect. Abstinence from tobacco or nicotine also appears to increase craving for sweet or sugary foods and impairs performance on tasks requiring vigilance. Smoking increases the metabolism of many medications used to treat mental disorders; thus, cessation of smoking can increase the blood levels of these medications, and this can produce clinically significant outcomes. This effect appears to be due not to nicotine but rather to other compounds in tobacco.

Prevalence

Approximately 50% of daily smokers who quit for 2 or more days will have four or more symptoms of tobacco withdrawal. The most commonly endorsed signs and symptoms are anxiety, irritability, and difficulty concentrating. The least commonly endorsed symptoms are depression and insomnia.

Development and Course

Tobacco withdrawal usually begins within 24 hours of stopping or cutting down tobacco use, peaks at 2–3 days after abstinence, and usually lasts 2–3 weeks. Tobacco withdrawal symptoms can occur among adolescent tobacco users, even prior to daily tobacco use. Prolonged symptoms beyond 1 month can occur but are uncommon.

Risk and Prognostic Factors

Temperamental. Smokers with depressive disorders, bipolar disorders, anxiety disorders, attention-deficit/hyperactivity disorder, and other substance use disorders have more severe withdrawal.

Genetic and physiological. Genotype can influence the probability of withdrawal upon abstinence.

Diagnostic Markers

The following biomarkers can be used to measure the extent of tobacco or nicotine use but are only weakly associated with tobacco withdrawal: carbon monoxide in the breath and nicotine and its metabolite cotinine in blood, saliva, or urine.

Functional Consequences of Tobacco Withdrawal

Tobacco withdrawal can cause significant distress and difficulty functioning in a minority of smokers, but this may be uncommon. Withdrawal impairs the ability to stop or control tobacco use. Whether tobacco withdrawal can prompt the development of a new mental disorder or recurrence of a mental disorder is debatable, but if this occurs, it would be in a small minority of tobacco users.

Differential Diagnosis

The symptoms of tobacco withdrawal overlap with those of other substance withdrawal syndromes (e.g., alcohol withdrawal; sedative, hypnotic, or anxiolytic withdrawal; stimulant withdrawal; caffeine withdrawal; opioid withdrawal); caffeine intoxication; anxiety, depressive, bipolar, and sleep disorders; and medication-induced akathisia. Admission to smoke-free inpatient units or voluntary smoking cessation can induce withdrawal symptoms that mimic, intensify, or disguise other disorders or adverse effects of medications used to treat mental disorders (e.g., irritability thought to be due to alcohol withdrawal could be due to tobacco withdrawal). Reduction in symptoms with the use of nicotine confirms the diagnosis.

Comorbidity

Given the typical overlap of tobacco withdrawal with tobacco use disorder, see "Comorbidity" under Tobacco Use Disorder for more details about co-occurring conditions that are likely to be encountered.

Tobacco-Induced Mental Disorders

Tobacco-induced sleep disorder is discussed in the chapter "Sleep-Wake Disorders" (see "Substance/Medication-Induced Sleep Disorder").

Unspecified Tobacco-Related Disorder

F17.209

This category applies to presentations in which symptoms characteristic of a tobacco-related disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific tobacco-related disorder or any of the disorders in the substance-related and addictive disorders diagnostic class.

Other (or Unknown) Substance–Related Disorders

Other (or Unknown) Substance Use Disorder

Other (or Unknown) Substance Intoxication

Other (or Unknown) Substance Withdrawal

Other (or Unknown) Substance–Induced Mental Disorders

Unspecified Other (or Unknown) Substance–Related Disorder

Other (or Unknown) Substance Use Disorder

Diagnostic Criteria

A. A problematic pattern of use of an intoxicating substance not able to be classified within the alcohol; caffeine; cannabis; hallucinogen (phencyclidine and others); inhalant; opioid; sedative, hypnotic, or anxiolytic; stimulant; or tobacco categories and leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period:

1. The substance is often taken in larger amounts or over a longer period than was intended.
2. There is a persistent desire or unsuccessful efforts to cut down or control use of the substance.
3. A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects.
4. Craving, or a strong desire or urge to use the substance.
5. Recurrent use of the substance resulting in a failure to fulfill major role obligations at work, school, or home.
6. Continued use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use.
7. Important social, occupational, or recreational activities are given up or reduced because of use of the substance.
8. Recurrent use of the substance in situations in which it is physically hazardous.
9. Use of the substance is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.
10. Tolerance, as defined by either of the following:
    a. A need for markedly increased amounts of the substance to achieve intoxication or desired effect.
    b. A markedly diminished effect with continued use of the same amount of the substance.
11. Withdrawal, as manifested by either of the following:
    a. The characteristic withdrawal syndrome for other (or unknown) substance (refer to Criteria A and B of the criteria sets for other [or unknown] substance withdrawal).

    b. The substance (or a closely related substance) is taken to relieve or avoid withdrawal symptoms.

Specify if:

In early remission: After full criteria for other (or unknown) substance use disorder were previously met, none of the criteria for other (or unknown) substance use disorder have been met for at least 3 months but for less than 12 months (with the exception that Criterion A4, “Craving, or a strong desire or urge to use the substance,” may be met).

In sustained remission: After full criteria for other (or unknown) substance use disorder were previously met, none of the criteria for other (or unknown) substance use disorder have been met at any time during a period of 12 months or longer (with the exception that Criterion

A4, “Craving, or a strong desire or urge to use the substance,” may be met).

Specify if:

In a controlled environment: This additional specifier is used if the individual is in an environment where access to the substance is restricted.

Code based on current severity/remission: If an other (or unknown) substance intoxication, other (or unknown) substance withdrawal, or other (or unknown) substance–induced mental disorder is present, do not use the codes below for other (or unknown) substance use disorder. Instead, the comorbid other (or unknown) substance use disorder is indicated in the 4th character of the other (or unknown) substance–induced disorder code (see the coding note for other [or unknown] substance intoxication, other [or unknown] substance withdrawal, or specific other [or unknown] substance–induced mental disorder). For example, if there is comorbid other (or unknown) substance–induced depressive disorder and other (or unknown) substance use disorder, only the other (or unknown) substance–induced depressive disorder code is given, with the 4th character indicating whether the comorbid other (or unknown) substance use disorder is mild, moderate, or severe: F19.14 for other (or unknown) substance use disorder with other (or unknown) substance–induced depressive disorder or F19.24 for a moderate or severe other (or unknown) substance use disorder with other (or unknown) substance–induced depressive disorder.

Specify current severity/remission:

F19.10 Mild: Presence of 2–3 symptoms.

F19.11 Mild, In early remission

F19.11 Mild, In sustained remission

F19.20 Moderate: Presence of 4–5 symptoms.

F19.21 Moderate, In early remission

F19.21 Moderate, In sustained remission

F19.20 Severe: Presence of 6 or more symptoms.

F19.21 Severe, In early remission

F19.21 Severe, In sustained remission

Specifiers

“In a controlled environment” applies as a further specifier of remission if the individual is both in remission and in a controlled environment (i.e., in early remission in a controlled environment or in sustained remission in a controlled environment). Examples of these environments are closely supervised and substance-free jails, therapeutic communities, and locked hospital units.

Diagnostic Features

The diagnostic class other (or unknown) substance–related disorders applies to substances that are not included within any of the nine substance classes presented earlier in this chapter (i.e., to alcohol; caffeine; cannabis; hallucinogens [phencyclidine and others]; inhalants; opioids; sedatives, hypnotics, or anxiolytics; stimulants; or tobacco). Such substances include anabolic steroids; nonsteroidal anti-inflammatory

drugs; corticosteroids; antiparkinsonian medications; antihistamines; nitrous oxide; amyl-, butyl-, or isobutyl-nitrites; betel nut, which is chewed in many geographic regions to produce mild euphoria and a floating sensation; and kava (from a South Pacific pepper plant), which produces mild euphoria, sedation, incoordination, and weight loss, as well as health effects (e.g., mild hepatitis, lung abnormalities). Note that gaseous substances are included with the inhalant category only if they are hydrocarbon agents; other gaseous substances (including nitrous oxide mentioned above) are included in the other (or unknown) substance category. Unknown substance–related disorders are associated with unidentified substances, such as intoxications in which the individual cannot identify the ingested drug, or substance use disorders involving either new, black market drugs not yet identified or familiar drugs illegally sold under false names.

Note that substances included within the scope of one of the substance classes should be coded within that respective substance class and are inappropriate to include in the "other substance" category. For example, the following substances are explicitly included in specific substance classes and should not be included in the "other substance" category: synthetic cannabinoids are included in the cannabis category; propofol is included in the sedative, hypnotic, or anxiolytic category; and cathinones (including *khât* plant agents and synthetic chemical derivatives) are included in the stimulant category.

Other (or unknown) substance use disorder is a mental disorder in which repeated use of an other or unknown substance typically continues, despite the individual's knowing that the substance is causing serious problems for the individual. Those problems are reflected in the diagnostic criteria. When the substance is known but does not fit within any of the other nine substance classes, it should be reflected when recording and coding the name of the disorder (e.g., "nitrous oxide use disorder," using the applicable code for other [or unknown] substance use disorder).

Associated Features

A diagnosis of other (or unknown) substance use disorder is supported by any of the following: the individual's reported use of a substance that is not among the nine classes listed in this chapter; recurring episodes of intoxication with negative results in standard drug screens, which may not detect new or rarely used substances; and the presence of symptoms characteristic of an unidentified substance that has newly appeared in the individual's community.

Because of access to nitrous oxide ("laughing gas"), membership in certain populations may be associated with frequent use of the substance and possibly with a diagnosis of nitrous oxide use disorder. The role of this gas as an anesthetic agent leads to misuse by some medical and dental professionals, and its use as a propellant for commercial products (e.g., whipped cream dispensers) contributes to misuse by food service workers. Nitrous oxide misuse by adolescents and young adults is significant, and some individuals with very frequent use may present with serious medical complications and mental conditions, including myeloneuropathy, spinal cord subacute combined degeneration, peripheral neuropathy, and psychosis.

Use of amyl-, butyl-, and isobutyl (and similar) nitrite gases is prevalent among homosexual men and some adolescents, especially those with conduct disorder.

Substance use disorders generally are associated with elevated risks of suicide, but there is no evidence of unique risk factors for suicide with other (or unknown) substance use disorder.

Prevalence

Based on extremely limited data, the prevalence of most other (or unknown) substance use disorders is likely lower than that of use disorders involving the nine substance classes in this chapter. For certain gaseous substances, prevalence of use is not rare (lifetime prevalence in the U.S. household population for individuals age 12 and older is estimated at 4.6% for nitrous oxide and 2.5% for nitrites), but how often the patterns of use qualify for a use disorder is unknown.

Development and Course

No single pattern of development or course characterizes the pharmacologically varied other (or unknown) substance use disorders. Often unknown substance use disorders will be reclassified when the unknown substance eventually is identified.

Risk and Prognostic Factors

Risk and prognostic factors for other (or unknown) substance use disorders are thought to be similar to those for most substance use disorders and include the presence of any other substance use disorders, conduct disorder, or antisocial personality disorder in the individual or the individual's family; early onset of substance problems; easy availability of the substance in the individual's environment; childhood maltreatment or trauma; and evidence of limited early self-control and behavioral disinhibition.

Culture-Related Diagnostic Issues

Certain cultures may be associated with other (or unknown) substance use disorders involving specific indigenous substances within the cultural region, such as betel nut.

Diagnostic Markers

Urine, breath, or saliva tests may correctly identify a commonly used substance falsely sold as a novel product. However, routine clinical tests usually cannot identify truly unusual or new substances, which may require testing in specialized laboratories.

Differential Diagnosis

Use of other or unknown substances without meeting criteria for other (or unknown) substance use disorder.
Use of unknown substances is not rare among adolescents, but most use does not meet the diagnostic standard of two or more criteria for other (or unknown) substance use disorder in a 12-month period.

Substance use disorders. Other (or unknown) substance use disorder may co-occur with various substance use disorders that involve any of the nine substance classes presented earlier in this chapter, and the symptoms of the disorders may be similar and overlapping. To disentangle symptom patterns, it is helpful to inquire about which symptoms persisted during periods when some of the substances were not being used.

Other (or unknown) substance intoxication, other (or unknown) substance withdrawal, and other (or unknown) substance–induced mental disorders.
Other (or unknown) substance use disorder is differentiated from other (or unknown) substance intoxication, other (or unknown) substance withdrawal, and other-(or unknown) substance-induced mental disorders (e.g., corticosteroid-induced bipolar and related disorder) in that other (or unknown) substance use disorder describes a problematic pattern of use of the other (or unknown) substance that involves impaired control over the use of the substance, social impairment attributable to use of the substance, risky use of the substance (e.g., continued use despite medical complications), and pharmacological symptoms (the development of

tolerance or withdrawal), whereas other (or unknown) substance intoxication, other (or unknown) substance withdrawal, and other (or unknown) substance-induced mental disorders describe psychiatric syndromes that occur in the context of heavy use. Other (or unknown) substance intoxication, other (or unknown) substance withdrawal, and other (or unknown) substance–induced mental disorders may occur in individuals with other (or unknown) substance use disorder. In such cases, a diagnosis of other (or unknown) substance intoxication, other (or unknown) substance withdrawal, or other (or unknown) substance–induced mental disorder should be given in addition to a diagnosis of other (or unknown) substance use disorder, the presence of which is indicated in the diagnostic code.

Comorbidity

Substance use disorders, including other (or unknown) substance use disorder, are commonly comorbid with one another, with conduct disorder in adolescence, and with antisocial personality disorder.

Other (or Unknown) Substance Intoxication

Diagnostic Criteria

A. The development of a reversible substance-specific syndrome attributable to recent ingestion of (or exposure to) a substance that is not listed elsewhere or is unknown.

B. Clinically significant problematic behavioral or psychological changes that are attributable to the effect of the substance on the central nervous system (e.g., impaired motor coordination, psychomotor agitation or retardation, euphoria, anxiety, belligerence, mood lability, cognitive impairment, impaired judgment, social withdrawal) and develop during, or shortly after, use of the substance.

C. The signs or symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including intoxication with another substance.

Specify if:

With perceptual disturbances: This specifier may be noted when hallucinations with intact reality testing or auditory, visual, or tactile illusions occur in the absence of a delirium.

Coding note: The ICD-10-CM code depends on whether there is a comorbid other (or unknown) substance use disorder involving the same substance and whether or not there are perceptual disturbances.

For other (or unknown) substance intoxication, without perceptual disturbances: If a mild other (or unknown) substance use disorder is comorbid, the ICD-10-CM code is **F19.120**, and if a moderate or severe other (or unknown) substance use disorder is comorbid, the ICD-10-CM code is **F19.220**. If there is no comorbid other (or unknown) substance use disorder, then the ICD-10-CM code is **F19.920**.

For other (or unknown) substance intoxication, with perceptual disturbances: If a mild other (or unknown) substance use disorder is comorbid, the ICD-10-CM code is **F19.122**, and if a moderate or severe other (or unknown) substance use disorder is comorbid, the ICD-10-CM

code is **F19.222**. If there is no comorbid other (or unknown) substance use disorder, then the ICD-10-CM code is **F19.922**.

Note: For information on Risk and Prognostic Factors, Culture-Related Diagnostic Issues, and Diagnostic Markers, see the corresponding sections in Other (or Unknown) Substance Use Disorder.

Diagnostic Features

The essential feature of other (or unknown) substance intoxication is the presence of clinically significant behavioral or psychological changes that develop during, or immediately after, use of either a) a substance not included within one of the nine substance classes presented in this chapter (i.e., alcohol; caffeine; cannabis; phencyclidine and other hallucinogens; inhalants; opioids; sedatives, hypnotics, or anxiolytics; stimulants; or tobacco) or b) an unknown substance. If the substance is known, it should be reflected in the name of the disorder upon coding (e.g., "kava intoxication").

Application of the diagnostic criteria for other (or unknown) substance intoxication is very challenging. Criterion A requires development of a reversible "substance-specific syndrome," but if the substance is unknown, that syndrome usually will be unknown. To resolve this conflict, clinicians may ask the individual or obtain collateral history as to whether the individual has experienced a similar episode after using substances with the same "street" name or from the same source. Similarly, hospital emergency departments sometimes recognize over a few days numerous presentations of a severe, unfamiliar intoxication syndrome from a newly available, previously unknown substance. Because of the great variety of intoxicating substances, Criterion B can provide only broad examples of signs and symptoms from some intoxications, with no threshold for the number of symptoms required for a diagnosis; clinical judgment guides those decisions. Criterion C requires ruling out other medical conditions, mental disorders, or intoxications.

Prevalence

The prevalence of other (or unknown) substance intoxication is unknown.

Development and Course

Intoxications usually appear and then peak minutes to hours after use of the substance, but the onset and course vary with the substance and the route of administration. Generally, substances used by pulmonary inhalation and intravenous injection have the most rapid onset of action, whereas those ingested by mouth and requiring metabolism to an active product are much slower. (For example, after ingestion of certain mushrooms, the first signs of an eventually fatal intoxication may not appear for a few days.) Intoxication effects usually resolve within hours to a very few days. However, the body may completely eliminate an anesthetic gas such as nitrous oxide just minutes after use ends. At the other extreme, some "hit-and-run" intoxicating substances poison systems, leaving permanent impairments. For example, MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine), a contaminating by-product in the synthesis of a certain opioid, kills dopaminergic cells and induces permanent parkinsonism in individuals who had sought opioid intoxication.

Functional Consequences of Other (or Unknown) Substance Intoxication

Impairment from intoxication with any substance may have serious consequences, including dysfunction at work, social indiscretions, problems in interpersonal relationships, failure to fulfill role obligations, traffic accidents, fighting, high-risk behaviors (i.e., having unprotected sex), and substance or medication overdose. The pattern of consequences will vary with the particular substance.

Differential Diagnosis

Use of other or unknown substance, without meeting criteria for other (or unknown) substance intoxication.
The individual used an other or unknown substance(s), but the dose was insufficient to produce symptoms that meet the diagnostic criteria required for the diagnosis.

Substance intoxication or other substance/medication-induced mental disorders. Familiar substances may be sold in the black market as novel products, and individuals may experience intoxication from those substances. History, toxicology screens, or chemical testing of the substance itself may help to identify it. Other substance intoxication is distinguished from other substance/medication-induced mental disorders (e.g., corticosteroid-induced anxiety disorder) because the symptoms (e.g., anxiety) in these latter disorders are in excess of those (if known) usually associated with the specific substance intoxication, predominate in the clinical presentation, and are severe enough to warrant clinical attention.

Other toxic, metabolic, traumatic, neoplastic, vascular, or infectious disorders that impair brain function and cognition.
Numerous neurological and other medical conditions may produce rapid onset of signs and symptoms mimicking those of intoxications, including the examples in Criterion B. Paradoxically, drug withdrawals also must be ruled out; for example, lethargy may indicate withdrawal from one drug or intoxication with another substance.

Comorbidity

As with all substance-related disorders, conduct disorder in adolescence, antisocial personality disorder, and other substance use disorders tend to co-occur with other (or unknown) substance intoxication.

Other (or Unknown) Substance Withdrawal

Diagnostic Criteria

A. Cessation of (or reduction in) use of a substance that has been heavy and prolonged.
B. The development of a substance-specific syndrome shortly after the cessation of (or reduction in) substance use.
C. The substance-specific syndrome causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
D. The symptoms are not attributable to another medical condition and are not better explained by another mental disorder, including withdrawal from another substance.
E. The substance involved cannot be classified under any of the other substance categories (alcohol; caffeine; cannabis; opioids; sedatives, hypnotics, or anxiolytics; stimulants; or tobacco) or is unknown.

Specify if:

With perceptual disturbances: This specifier may be noted when hallucinations with intact reality testing or auditory, visual, or tactile illusions occur in the absence of a delirium.

Coding note: The ICD-10-CM code depends on whether or not there is a comorbid other (or unknown) substance use disorder and whether or not there are perceptual disturbances.

For other (or unknown) substance withdrawal, without perceptual disturbances: If a mild other (or unknown) substance use disorder is comorbid, the ICD-10-CM code is **F19.130**, and if a moderate or severe other (or unknown) substance use disorder is comorbid, the ICD-10-CM code is **F19.230**. If there is no comorbid other (or unknown) substance use disorder (e.g., in a patient taking an other [or unknown] substance solely under appropriate medical supervision), then the ICD-10-CM code is **F19.930**.

For other (or unknown) substance withdrawal, with perceptual disturbances: If a mild other (or unknown) substance use disorder is comorbid, the ICD-10-CM code is

F19.132, and if a moderate or severe other (or unknown) substance use disorder is comorbid, the ICD-10-CM code is **F19.232**. If there is no comorbid other (or unknown) substance use disorder (e.g., in a patient taking an other [or unknown] substance solely under appropriate medical supervision), then the ICD-10-CM code is **F19.932**.

Note: For information on Risk and Prognostic Factors and Diagnostic Markers, see the corresponding sections in Other (or Unknown) Substance Use Disorder.

Diagnostic Features

Other (or unknown) substance withdrawal is a clinically significant syndrome that develops during or within a few hours to days after reducing or terminating dosing with a substance (Criteria A and B). Although recent dose reduction or termination usually is clear in the history, other diagnostic procedures are very challenging if the drug is unknown. Criterion B requires development of a "substance-specific syndrome" (i.e., the individual's signs and symptoms must correspond with the known withdrawal syndrome for the recently stopped drug)—a requirement that rarely can be met with an unknown substance. Consequently, clinical judgment must guide such decisions when this information is limited. Criterion D requires ruling out other medical conditions, mental disorders, or withdrawals from familiar substances. When the substance is known, it should be reflected in the name of the disorder upon coding (e.g., "betel nut withdrawal").

Prevalence

The prevalence of other (or unknown) substance withdrawal is unknown.

Development and Course

Withdrawal signs commonly appear some hours after use of the substance is terminated, but the onset and course vary greatly, depending on the dose typically used and the rate of elimination of the specific substance from the body. At peak severity,

withdrawal symptoms from some substances involve only moderate levels of discomfort, whereas withdrawal from other substances may be fatal. Withdrawal-associated dysphoria often motivates relapse to substance use. Withdrawal symptoms slowly abate over days, weeks, or months, depending on the particular drug and doses to which the individual became tolerant.

Functional Consequences of Other (or Unknown) Substance Withdrawal

Withdrawal from any substance may have serious consequences, including physical signs and symptoms (e.g., malaise, vital sign changes, abdominal distress, headache), intense drug craving, anxiety, depression, agitation, psychotic symptoms, or cognitive impairments. These consequences may lead to problems such as dysfunction at work, problems in interpersonal relationships, failure to fulfill role obligations, traffic accidents, fighting, high-risk behavior (e.g., having unprotected sex), suicide attempts, and substance or medication overdose. The pattern of consequences will vary with the particular substance.

Differential Diagnosis

Dose reduction after extended dosing, but not meeting the criteria for other (or unknown) substance withdrawal.
The individual used other (or unknown) substances, but the dose that was used was insufficient to produce symptoms that meet the criteria required for the withdrawal diagnosis.

Substance withdrawal or other substance/medication-induced mental disorders. Familiar substances may be sold in the black market as novel products, and individuals may experience withdrawal when discontinuing those substances. History, toxicology screens, or

chemical testing of the substance itself may help to identify it. Other substance withdrawal is distinguished from other substance/medication-induced mental disorders (e.g., venlafaxine-induced anxiety disorder, with onset during withdrawal) because the symptoms (e.g., anxiety) in these latter disorders are in excess of symptoms (if known) usually associated with the specific substance withdrawal, predominate in the clinical presentation, and are severe enough to warrant clinical attention.

Other toxic, metabolic, traumatic, neoplastic, vascular, or infectious disorders that impair brain function and cognition.
Numerous neurological and other medical conditions may produce rapid onset of signs and symptoms mimicking those of withdrawals. Paradoxically, drug intoxications also must be ruled out; for example, lethargy may indicate withdrawal from one drug or intoxication with another substance.

Comorbidity

As with all substance-related disorders, conduct disorder in adolescence, antisocial personality disorder, and other substance use disorders are likely to co-occur with other (or unknown) substance withdrawal.

Other (or Unknown) Substance–Induced Mental Disorders

Because the category of other or unknown substances is inherently ill-defined, the extent and range of these substance-induced mental disorders are uncertain.

Nevertheless, other (or unknown) substance–induced mental disorders are possible and are described in other chapters of the manual with disorders with which they share phenomenology (see the substance/medication-induced mental disorders in these chapters): other (or unknown) substance–induced psychotic disorder ("Schizophrenia Spectrum and Other Psychotic Disorders"); other (or unknown) substance–induced bipolar and related disorder ("Bipolar and Related Disorders"); other (or unknown) substance–induced depressive disorder ("Depressive Disorders"); other (or unknown) substance–induced anxiety disorders ("Anxiety Disorders"); other (or unknown) substance–induced obsessive-compulsive disorder ("Obsessive-Compulsive and Related Disorders"); other (or unknown) substance–induced sleep disorder ("Sleep-Wake Disorders"); other (or unknown) substance–induced sexual dysfunction ("Sexual Dysfunctions"); and other (or unknown) substance/medication–induced major or mild neurocognitive disorder ("Neurocognitive Disorders"). For other (or unknown) substance–induced intoxication delirium, other (or unknown) substance–induced withdrawal delirium, and delirium induced by other (or unknown) substance taken as prescribed, see the criteria and discussion of delirium in the chapter "Neurocognitive Disorders." These other (or unknown) substance–induced mental disorders are diagnosed instead of other (or unknown) substance intoxication or other (or unknown) substance withdrawal only when the symptoms are sufficiently severe to warrant independent clinical attention.

Unspecified Other (or Unknown) Substance–Related Disorder

F19.99

This category applies to presentations in which symptoms characteristic of an other (or unknown) substance–related disorder that cause clinically significant distress or

impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific other (or unknown) substance–related disorder or any of the disorders in the substance-related disorders diagnostic class.

Non-Substance-Related Disorders

Gambling Disorder

Diagnostic Criteria **F63.0**

A. Persistent and recurrent problematic gambling behavior leading to clinically significant impairment or distress, as indicated by the individual exhibiting four (or more) of the following in a 12-month period:

1. Needs to gamble with increasing amounts of money in order to achieve the desired excitement.

2. Is restless or irritable when attempting to cut down or stop gambling.
3. Has made repeated unsuccessful efforts to control, cut back, or stop gambling.
4. Is often preoccupied with gambling (e.g., having persistent thoughts of reliving past gambling experiences, handicapping or planning the next venture, thinking of ways to get money with which to gamble).
5. Often gambles when feeling distressed (e.g., helpless, guilty, anxious, depressed).
6. After losing money gambling, often returns another day to get even ("chasing" one's losses).
7. Lies to conceal the extent of involvement with gambling.
8. Has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling.
9. Relies on others to provide money to relieve desperate financial situations caused by gambling.

B. The gambling behavior is not better explained by a manic episode.

Specify if:

Episodic: Meeting diagnostic criteria at more than one time point, with symptoms subsiding between periods of gambling disorder for at least several months.

Persistent: Experiencing continuous symptoms, to meet diagnostic criteria for multiple years.

Specify if:

In early remission: After full criteria for gambling disorder were previously met, none of the criteria for gambling disorder have been met for at least 3 months but for less than 12 months.

In sustained remission: After full criteria for gambling disorder were previously met, none of the criteria for gambling disorder have been met during a period of 12 months or longer.

Specify current severity:

Mild: 4–5 criteria met.

Moderate: 6–7 criteria met.

Severe: 8–9 criteria met.

Note: Although some behavioral conditions that do not involve ingestion of substances have similarities to substance-related disorders, only one disorder —gambling disorder—has sufficient data to be included in this section.

Specifiers

Severity is based on the number of criteria endorsed. Individuals with mild gambling disorder may exhibit only 4–5 of the criteria, with the most frequently endorsed criteria usually related to preoccupation with gambling and "chasing" losses. Individuals with moderately severe gambling disorder exhibit more of the criteria (i.e., 6–7). Individuals with the most severe form will exhibit all or most of the nine criteria (i.e., 8–9). Jeopardizing relationships or career opportunities because of gambling and relying on others to provide money for gambling losses are typically the least often endorsed criteria and most often occur among those with more severe gambling disorder.

Furthermore, individuals presenting for treatment of gambling disorder typically have moderate to severe forms of the disorder.

Diagnostic Features

Gambling involves risking something of value in the hopes of obtaining something of greater value. In many cultures, individuals gamble on games and events, and most do so without experiencing problems. However, some individuals develop substantial impairment related to their gambling behaviors. The essential feature of gambling disorder is persistent and recurrent maladaptive gambling behavior that disrupts personal, family, and/or vocational pursuits (Criterion A). Gambling disorder is defined as a cluster of four or more of the symptoms listed in Criterion A occurring at any time in the same 12-month period.

A pattern of "chasing one's losses" may develop, with an urgent need to continue gambling (often with placing larger bets or taking greater risks) to undo a loss or series of losses. The individual may abandon a gambling strategy and try to win back losses all at once. Although many gamblers may "chase" for short periods of time, it is the frequent, and often long-term, "chase" that is characteristic of gambling disorder (Criterion A6). Individuals may lie to family members, therapists, or others to conceal the extent of involvement with gambling; these instances of deceit may also include, but are not limited to, covering up illegal behaviors such as forgery, fraud, theft, or embezzlement to obtain money with which to gamble (Criterion A7). Individuals may also engage in "bailout" behavior, turning to family or others for help with a desperate financial situation that was caused by gambling (Criterion A9).

In some cases, symptoms meeting diagnostic criteria for gambling disorder may occur as a direct physiological consequence of taking dopaminergic medications, such as those used to treat Parkinson's disease. When such symptoms are induced by a medication, these cases would be diagnosed as gambling disorder.

Associated Features

Distortions in thinking (e.g., denial, superstitions, a sense of power and control over the outcome of chance events, overconfidence) may be present in individuals with gambling disorder. Many individuals with gambling disorder believe that money is both the cause of and the solution to their problems. Some individuals with gambling disorder are impulsive, competitive, energetic, restless, and easily bored; they may be overly concerned with the approval of others and may be generous to the point of extravagance when winning. Other individuals with gambling disorder are depressed and lonely, and they may gamble when feeling helpless, guilty, or depressed.

Prevalence

The past-year prevalence rate of gambling disorder is about 0.2%–0.3% in the general U.S. population, with a range of 0.1%–0.7% observed across international studies. In the

general U.S. population, the lifetime prevalence rate is about 0.4%–1.0%. For women, the lifetime prevalence rate of gambling disorder is about 0.2%, and for men it is about 0.6%. The 12-month prevalence of DSM-5 gambling disorder varies among ethnoracial groups in the United States: it is 0.52% in African Americans, 0.25% in Latinx, and 0.23% in non-Latinx Whites.

Development and Course

The onset of gambling disorder can occur during adolescence or young adulthood, but in other individuals it manifests during middle or even older adulthood. Generally, gambling disorder develops over the course of years, although the progression appears to be more rapid in women than in men. National data from the United States and Canada show that most individuals who develop a gambling disorder evidence a pattern of gambling that gradually increases in both frequency and amount of wagering. Certainly, milder forms can develop into more severe cases. Most individuals with gambling disorder report that one or two types of gambling are most problematic for them, although some individuals participate in many forms of gambling. Individuals are likely to engage in certain types of gambling (e.g., buying scratch tickets daily) more frequently than others (e.g., playing slot machines or blackjack at the casino weekly). Frequency of gambling can be related more to the type of gambling than to the severity of the overall gambling disorder. For example, purchasing a single scratch ticket each day may not be problematic, while less frequent casino, sports, or card gambling may be part of a gambling disorder. Similarly, amounts of money spent wagering are not in themselves indicative of gambling disorder. Some individuals can wager thousands of dollars per month and not have a problem with gambling, while others may wager much smaller amounts but experience substantial gambling-related difficulties.

Gambling patterns may be regular or episodic, and gambling disorder can be persistent or in remission. Gambling can increase during periods of stress or depression and during periods of substance use or abstinence. There may be periods of heavy gambling and severe problems, times of total abstinence, and periods of nonproblematic gambling. Gambling disorder is sometimes associated with spontaneous, long-term remissions. Nevertheless, some individuals underestimate their vulnerability to develop gambling disorder or to relapse following remission. When in a period of remission, they may incorrectly assume that they will have no problem regulating gambling and that they can engage in some forms of gambling nonproblematically, only to experience a relapse of gambling disorder.

Early expression of gambling disorder is more common among young men (ages 18–21 years) than among young women. Individuals who begin gambling in youth often do so with family members or friends. Development of early-life gambling disorder appears to be associated with impulsivity and substance abuse. Internet gambling has been linked to risky and problematic gambling among youth and may be conducted in a more isolative (i.e., nonpeer) fashion. Some video gaming characteristics (e.g., loot boxes or loot crates containing prizes determined by chance that may be of higher or lower value or desirability) overlap with gambling behavior and may influence the course of gambling disorder. Many high school and college students who develop gambling disorder grow out of the disorder over time, although it remains a lifelong problem for some. Mid- and later-life onset of gambling disorder is more common among women than among men.

There are age and gender variations in the type of gambling activities and the prevalence rates of gambling disorder. Gambling disorder in the United States is more common among younger and middle-age individuals than among older adults. Among U.S. young adults (ages 18–21 years), the disorder is more prevalent in young men than in young women. Younger individuals prefer different forms of gambling (e.g., sports betting), whereas older adults are more likely to develop problems with slot machine and bingo gambling. Although the proportions of individuals who seek treatment for gambling disorder are low across all age groups in the United States, younger individuals are especially unlikely to present for treatment.

Risk and Prognostic Factors

Temperamental. Gambling that begins in childhood or early adolescence is associated with increased rates of gambling disorder. Gambling disorder also appears to aggregate with antisocial personality disorder, depressive and bipolar disorders, and other substance use disorders, particularly alcohol use disorder.

Genetic and physiological. Gambling disorder can aggregate in families, and this effect appears to relate to both environmental and genetic factors. Gambling problems are more frequent in monozygotic than in dizygotic twins. Gambling disorder is also more prevalent among first-degree relatives of individuals with moderate to severe alcohol use disorder than among the general population.

Course modifiers. Many individuals, including adolescents and young adults, are likely to resolve their problems with gambling disorder over time, although a strong predictor of future gambling problems is previous gambling problems. Psychopathology, including attention-deficit/hyperactivity and anxiety disorders, has been found to be associated with increased risk of onset of gambling disorder among those who gamble and with persistence of gambling disorder symptoms over time.

Culture-Related Diagnostic Issues

Types of gambling activities vary across cultural contexts and ethnoracial groups (e.g., pai gow, cockfights, blackjack, horse racing). Some Indigenous populations in Canada, New Zealand, and the United States have high prevalence rates of gambling problems, possibly related to limited economic opportunities, the expectation that gambling may help advance social goals, and the location of casinos on some U.S. tribal lands. U.S.-born individuals have higher rates of gambling problems than first-generation immigrants to the United States. Endorsement of specific disorder criteria may vary across ethnoracial groups. For example, among individuals with gambling problems, Asian Americans may be less likely than other groups to endorse being preoccupied with gambling (Criterion A4), while African Americans and Latinx may be more likely to endorse repeated unsuccessful efforts to control gambling (Criterion A3).

Sex- and Gender-Related Diagnostic Issues

Men develop gambling disorder at higher rates than women, although this gender gap may be narrowing. Data from treatment-seeking populations have suggested that women may develop gambling problems more rapidly after the onset of gambling (so-called telescoping), although general population data suggest that men progress more rapidly to disordered gambling than women do. Although women seek treatment sooner than men do, rates of treatment seeking in U.S. national surveys are low (< 10%) among individuals with gambling disorder regardless of gender.

Women may gamble as a maladaptive approach to negative affect, whereas men may gamble more for the thrill of it. Compared with men, women may also experience more shame related to gambling. Men tend to wager on different forms of gambling than women, with cards, sports, and horse race gambling more prevalent among men, and slot machine and bingo gambling more common among women. Women with gambling disorder are more likely than men with gambling disorder to have depressive, bipolar, and anxiety disorders.

Association With Suicidal Thoughts or Behavior

In a U.S. study, up to half of individuals in treatment for gambling disorder in Connecticut reported suicidal thoughts, and about 17% reported attempted suicide. A nationwide register study in Sweden showed that compared with individuals without gambling disorder, individuals ages 20–74 years with gambling disorder have a 15-fold increased suicide mortality rate.

Functional Consequences of Gambling Disorder

Areas of psychosocial, health, and mental health functioning may be adversely affected by gambling disorder. Specifically, individuals with gambling disorder may, because of their involvement with gambling, jeopardize or lose important relationships with family members or friends. Such problems may occur from repeatedly lying to others to cover up the extent of gambling or from requesting money that is used for gambling or to pay off gambling debts. Employment or educational activities may likewise be adversely impacted by gambling disorder; absenteeism or poor work or school performance can occur with gambling disorder, as individuals may gamble during work or school hours or be preoccupied with gambling or its adverse consequences when they should be working or studying. Individuals with gambling disorder in a U.S. national sample had poor general health and utilized medical services at high rates.

Differential Diagnosis

Nondisordered gambling. Gambling disorder must be distinguished from professional and social gambling. In professional gambling, risks are limited and discipline is central. Social gambling typically occurs with friends or colleagues and lasts for a limited period of time, with acceptable losses. Some persons can experience problems associated with gambling (e.g., short-term chasing behavior and loss of control) that do not meet the full criteria for gambling disorder.

Manic episode. Loss of judgment and excessive gambling may occur during a manic episode. An additional diagnosis of gambling disorder should be given only if the gambling behavior is not better explained by manic episodes (e.g., a history of maladaptive gambling behavior at times other than during a manic episode). Alternatively, an individual with gambling disorder may, during a period of gambling, exhibit behavior that resembles a manic episode, but once the individual is away from the gambling, these manic-like features dissipate.

Personality disorders. Problems with gambling may occur in individuals with antisocial personality disorder and other personality disorders. If the criteria are met for both disorders, both can be diagnosed.

Gambling symptoms due to dopaminergic medications. Some individuals taking dopaminergic medications (e.g., for Parkinson‘s disease) may experience urges to gamble that might be distressing or impairing enough to meet criteria for gambling disorder. In such cases, a diagnosis of gambling disorder would be warranted.

Comorbidity

Gambling disorder is associated with poor general health. In addition, some specific medical conditions, such as tachycardia and angina, are more common among individuals with gambling disorder than in the general population, even when other substance use disorders, including tobacco use disorder, are controlled for. In U.S. national surveys, individuals with gambling disorder have high rates of comorbidity with other mental disorders, such as substance use disorders, depressive disorders, anxiety disorders, and personality disorders. In some individuals, other mental disorders may precede gambling disorder and be either absent or present during the manifestation of gambling disorder. Gambling disorder may also occur prior to the onset of other mental disorders, especially bipolar and related disorders, anxiety disorders, and substance use disorders. In a U.S. national survey, in approximately three-quarters of cases of individuals with gambling disorder and another mental disorder, other psychopathology preceded the gambling disorder.

Neurocognitive Disorders

The neurocognitive disorders (NCDs) begin with delirium, followed by the syndromes of major NCD, mild NCD, and their etiological subtypes. The major or mild NCD subtypes are NCD due to Alzheimer's disease; vascular NCD; NCD with Lewy bodies; NCD due to Parkinson's disease; frontotemporal NCD; NCD due to traumatic brain injury; NCD due to HIV infection; substance/medication-induced NCD; NCD due to Huntington's disease; NCD due to prion disease; NCD due to another medical condition; NCD due to multiple etiologies; and unspecified NCD. The NCD category encompasses the group of disorders in which the primary clinical deficit is in cognitive function, and that are acquired rather than developmental. Although cognitive deficits are present in many if not all mental disorders (e.g., schizophrenia, bipolar disorders), only disorders whose core features are cognitive are included in the NCD category. The NCDs are those in which impaired cognition has not been present since birth or very early life, and thus represents a decline from a previously attained level of functioning.

The NCDs are unique among DSM-5 categories in that these are syndromes for which the underlying pathology, and frequently the etiology as well, can potentially be determined. The various underlying disease entities have all been the subject of extensive research, clinical experience, and expert consensus on diagnostic criteria. The DSM-5 criteria for these disorders have been developed in close consultation with the expert groups for each of the disease entities and align as closely as possible with the current consensus criteria for each of them. The potential utility of biomarkers is also discussed in relation to diagnosis. Dementia is subsumed under the newly named entity *major neurocognitive disorder*, although the term *dementia* is not precluded from use in the etiological subtypes in which that term is standard. Furthermore, DSM-5 recognizes a less severe

level of cognitive impairment, *mild neurocognitive disorder,* which can also be a focus of care. Diagnostic criteria are provided for both these syndromic entities, followed by diagnostic criteria for the different etiological subtypes. Several of the NCDs frequently coexist with one another, and their relationships may be multiply characterized under different chapter subheadings, including "Differential Diagnosis" (e.g., NCD due to Alzheimer's disease vs. vascular NCD), "Risk and Prognostic Factors" (e.g., vascular pathology increasing the clinical expression of Alzheimer's disease), or "Comorbidity" (e.g., mixed Alzheimer's disease–vascular pathology).

The term *dementia* is retained in DSM-5 for continuity and may be used in settings where physicians and patients are accustomed to this term. Although dementia is the customary term for disorders like the degenerative dementias that usually affect older adults, the term *neurocognitive disorder* is widely used and often preferred for conditions affecting younger individuals, such as impairment secondary to traumatic brain injury or HIV infection. Furthermore, the major NCD definition is somewhat broader than the term *dementia,* in that a diagnosis of major NCD can be made if there is a significant cognitive decline in only one cognitive domain, whereas a diagnosis of dementia in ICD-10 and ICD-11 (and formerly in DSM-IV) requires multiple cognitive deficits. Thus, cases that would qualify in ICD-10 and ICD-11 (and formerly DSM-IV) for a diagnosis of amnestic disorder (memory impairment in the absence of other cognitive deficits) are diagnosed as major NCD in DSM-5.

Neurocognitive Domains

The criteria for the various NCDs are based on defined cognitive domains. Table 1 provides for each of the key domains a working definition, examples of symptoms or observations regarding impairments in everyday activities, and examples of assessments. The domains thus defined, along with guidelines for clinical thresholds, form the basis on which the NCDs, their levels, and their subtypes may be diagnosed.

TABLE 1 Neurocognitive domains

Cognitive domain	Examples of symptoms or observations	Examples of assessments
Complex attention (sustained attention, divided attention, selective attention, processing speed)	*Major:* Has increased difficulty in environments with multiple stimuli (TV, radio, conversation); is easily distracted by competing events in the environment. Is unable to attend unless input is restricted and simplified. Has difficulty holding new information in mind, such as recalling phone numbers or addresses just given, or reporting what was just said. Is unable to perform mental calculations. All thinking takes longer than usual, and components to be processed must be simplified to one or a few. *Mild:* Normal tasks take longer than previously. Begins to find errors in routine tasks; finds work needs more double-checking than previously. Thinking is easier when not competing with other things (radio, TV, other conversations, cell phone, driving).	*Sustained attention:* Maintenance of attention over time (e.g., pressing a button every time a tone is heard, and over a period of time). *Selective attention:* Maintenance of attention despite competing stimuli or distractors: hearing numbers and letters read and asked to count only letters. *Divided attention:* Attending to two tasks within the same time period: rapidly tapping while learning a story being read. Processing speed can be quantified on any task by timing it (e.g., time to put together a design of blocks; time to match symbols with numbers; speed in responding, such as counting speed or serial 3 speed).

Cognitive domain	Examples of symptoms or observations	Examples of assessments
Executive function (planning, decision-making, working memory, responding to feedback/error correction, overriding habits/inhibition, mental flexibility)	*Major:* Abandons complex projects. Needs to focus on one task at a time. Needs to rely on others to plan instrumental activities of daily living or make decisions. *Mild:* Increased effort required to complete multistage projects. Has increased difficulty multitasking or difficulty resuming a task interrupted by a visitor or phone call. May complain of increased fatigue from the extra effort required to organize, plan, and make decisions. May report that large social gatherings are more taxing or less enjoyable because of increased effort required to follow shifting conversations.	*Planning:* Ability to find the exit to a maze; interpret a sequential picture or object arrangement. *Decision-making:* Performance of tasks that assess process of deciding in the face of competing alternatives (e.g., simulated gambling). *Working memory:* Ability to hold information for a brief period and to manipulate it (e.g., adding up a list of numbers or repeating a series of numbers or words backward). *Feedback/error utilization:* Ability to benefit from feedback to infer the rules for solving a problem. *Overriding habits/inhibition:* Ability to choose a more complex and effortful solution to be correct (e.g., looking away from the direction indicated by an arrow; naming the color of a word's font rather than naming the word). *Mental/cognitive flexibility:* Ability to shift between two concepts, tasks, or response rules (e.g., from number to letter, from verbal to key-press response, from adding numbers to ordering numbers, from ordering objects by size to ordering by color).

Cognitive domain	Examples of symptoms or observations	Examples of assessments
670 Learning and memory (immediate memory, recent memory [including free recall, cued recall, and recognition memory], very-long-term memory [semantic; autobiographical], implicit learning)	*Major:* Repeats self in conversation, often within the same conversation. Cannot keep track of short list of items when shopping or of plans for the day. Requires frequent reminders to orient to task at hand. *Mild:* Has difficulty recalling recent events, and relies increasingly on list making or calendar. Needs occasional reminders or re-reading to keep track of characters in a movie or novel. Occasionally may repeat self over a few weeks to the same person. Loses track of whether bills have already been paid. **Note:** Except in severe forms of major neurocognitive disorder, semantic, autobiographical, and implicit learning are relatively preserved, compared with recent memory.	*Immediate memory span:* Ability to repeat a list of words or digits. **Note:** Immediate memory sometimes subsumed under "working memory" (see "Executive Function"). *Recent memory:* Assesses the process of encoding new information (e.g., word lists, a short story, or diagrams). The aspects of recent memory that can be tested include 1) free recall (the individual is asked to recall as many words, diagrams, or elements of a story as possible); 2) cued recall (examiner aids recall by providing semantic cues such as "List all the food items on the list" or "Name all of the children from the story"); and 3) recognition memory (examiner asks about specific items—e.g., "Was 'apple' on the list?" or "Did you see this diagram or figure?"). Other aspects of memory that can be assessed include semantic memory (memory for facts), autobiographical memory (memory for personal events or people), and implicit (procedural) learning (unconscious learning of skills).

Cognitive domain	Examples of symptoms or observations	Examples of assessments
Language (expressive language [including naming, word finding, fluency, and grammar, and syntax] and receptive language)	*Major:* Has significant difficulties with expressive or receptive language. Often uses general-use phrases such as “that thing” and “you know what I mean,” and prefers general pronouns rather than names. With severe impairment, may not even recall names of closer friends and family. Idiosyncratic word usage, grammatical errors, and spontaneity of output and economy of utterances occur. Stereotypy of speech occurs; echolalia and automatic speech typically precede mutism. *Mild:* Has noticeable word-finding difficulty. May substitute general for specific terms. May avoid use of specific names of acquaintances. Grammatical errors involve subtle omission or incorrect use of articles, prepositions, auxiliary verbs, etc.	*Expressive language:* Confrontational naming (identification of objects or pictures); fluency (e.g., name as many items as possible in a semantic [e.g., animals] or phonemic [e.g., words starting with “f”] category in 1 minute). *Grammar and syntax* (e.g., omission or incorrect use of articles, prepositions, auxiliary verbs): Errors observed during naming and fluency tests are compared with norms to assess frequency of errors and compare with normal slips of the tongue. *Receptive language:* Comprehension (word definition and object-pointing tasks involving animate and inanimate stimuli): performance of actions/activities according to verbal command.

Cognitive domain	Examples of symptoms or observations	Examples of assessments
671 Perceptual-motor (includes abilities subsumed under the terms *visual perception, visuoconstructional, perceptual-motor, praxis,* and *gnosis*)	*Major:* Has significant difficulties with previously familiar activities (using tools, driving motor vehicle), navigating in familiar environments; is often more confused at dusk, when shadows and lowering levels of light change perceptions. *Mild:* May need to rely more on maps or others for directions. Uses notes and follows others to get to a new place. May find self lost or turned around when not concentrating on task. Is less precise in parking. Needs to expend greater effort for spatial tasks such as carpentry, assembly, sewing, or knitting.	*Visual perception:* Line bisection tasks can be used to detect basic visual defect or attentional neglect. Motor-free perceptual tasks (including facial recognition) require the identification and/or matching of figures—best when tasks cannot be verbally mediated (e.g., figures are not objects); some require the decision of whether a figure can be "real" or not based on dimensionality. *Visuoconstructional:* Assembly of items requiring hand-eye coordination, such as drawing, copying, and block assembly. *Perceptual-motor:* Integrating perception with purposeful movement (e.g., inserting blocks into a form board without visual cues; rapidly inserting pegs into a slotted board). *Praxis:* Integrity of learned movements, such as ability to imitate gestures (wave goodbye) or pantomime use of objects to command ("Show me how you would use a hammer"). *Gnosis:* Perceptual integrity of awareness and recognition, such as recognition of faces and colors.

Cognitive domain	Examples of symptoms or observations	Examples of assessments
Social cognition (recognition of emotions, theory of mind)	*Major:* Behavior clearly out of acceptable social range; shows insensitivity to social standards of modesty in dress or of political, religious, or sexual topics of conversation. Focuses excessively on a topic despite group's disinterest or direct feedback. Behavioral intention without regard to family or friends. Makes decisions without regard to safety (e.g., inappropriate clothing for weather or social setting). Typically, has little insight into these changes. *Mild:* Has subtle changes in behavior or attitude, often described as a change in personality, such as less ability to recognize social cues or read facial expressions, decreased empathy, increased extraversion or introversion, decreased inhibition, or subtle or episodic apathy or restlessness.	*Recognition of emotions:* Identification of emotion in images of faces representing a variety of both positive and negative emotions. *Theory of mind:* Ability to consider another person's mental state (thoughts, desires, intentions) or experience—story cards with questions to elicit information about the mental state of the individuals portrayed, such as "Where will the girl look for the lost bag?" or "Why is the boy sad?"

Delirium

Diagnostic Criteria

A. A disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) accompanied by reduced awareness of the environment.

B. The disturbance develops over a short period of time (usually hours to a few days), represents a change from baseline attention and awareness, and tends to fluctuate in severity during the course of a day.

C. An additional disturbance in cognition (e.g., memory deficit, disorientation, language, visuospatial ability, or perception).

D. The disturbances in Criteria A and C are not better explained by another preexisting, established, or evolving neurocognitive disorder and do not occur in the context of a severely reduced level of arousal, such as coma.

E. There is evidence from the history, physical examination, or laboratory findings that the disturbance is a direct physiological consequence of another medical condition, substance intoxication or withdrawal (i.e., due to a drug of abuse or to a medication), or exposure to a toxin, or is due to multiple etiologies.

Specify if:

Acute: Lasting a few hours or days.

Persistent: Lasting weeks or months.

Specify if:

Hyperactive: The individual has a hyperactive level of psychomotor activity that may be accompanied by mood lability, agitation, and/or refusal to cooperate with medical care.

Hypoactive: The individual has a hypoactive level of psychomotor activity that may be accompanied by sluggishness and lethargy that approaches stupor.

Mixed level of activity: The individual has a normal level of psychomotor activity even though attention and awareness are disturbed. Also includes individuals whose activity level rapidly fluctuates.

Specify whether:

Substance intoxication delirium: This diagnosis should be made instead of substance intoxication when the symptoms in Criteria A and C predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance] intoxication delirium are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. If a mild substance use disorder is comorbid with the substance intoxication delirium, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance intoxication delirium (e.g., "mild cocaine use disorder with cocaine intoxication delirium"). If a moderate or severe substance use disorder is comorbid with the substance intoxication delirium, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after a one-time heavy use of the substance), then the 4th position character is "9," and the clinician should record only the substance intoxication delirium.

	ICD-10-CM		
Substance intoxication delirium	**With mild use disorder**	**With moderate or severe use disorder**	**Without use disorder**
Alcohol	F10.121	F10.221	F10.921
Cannabis	F12.121	F12.221	F12.921
Phencyclidine	F16.121	F16.221	F16.921

	ICD-10-CM		
Other hallucinogen	F16.121	F16.221	F16.921
Inhalant	F18.121	F18.221	F18.921
Opioid	F11.121	F11.221	F11.921
Sedative, hypnotic, or anxiolytic	F13.121	F13.221	F13.921
Amphetamine-type substance (or other stimulant)	F15.121	F15.221	F15.921
Cocaine	F14.121	F14.221	F14.921
Other (or unknown) substance	F19.121	F19.221	F19.921

Substance withdrawal delirium: This diagnosis should be made instead of substance withdrawal when the symptoms in Criteria A and C predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

Coding note: The ICD-10-CM codes for the [specific substance] withdrawal delirium are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. If a mild substance use disorder is comorbid with the substance withdrawal delirium, the 4th position character is “1,” and the clinician should record “mild [substance] use disorder” before the substance withdrawal delirium (e.g., “mild alcohol use disorder with alcohol withdrawal delirium”). If a moderate or severe substance use disorder is comorbid with the substance withdrawal delirium, the 4th position character is “2,” and the clinician should record “moderate [substance] use disorder” or “severe [substance] use disorder,” depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder (e.g., after regular use of an anxiolytic substance taken as prescribed), then the 4th position character is “9,” and the clinician should record only the substance withdrawal delirium.

	ICD-10-CM		
Substance withdrawal delirium	**With mild use disorder**	**With moderate or severe use disorder**	**Without use disorder**
Alcohol	F10.131	F10.231	F10.931
Opioid	F11.188	F11.288	F11.988
Sedative, hypnotic, or anxiolytic	F13.131	F13.231	F13.931
Other (or unknown) substance	F19.131	F19.231	F19.931

Medication-induced delirium: This diagnosis applies when the symptoms in Criteria A and C arise as a side effect of a medication taken as prescribed.

Code [specific medication]–induced delirium: **F11.921** opioid taken as prescribed (or **F11.988** if during withdrawal from opioid taken as prescribed); **F12.921** pharmaceutical cannabis receptor agonist taken as prescribed; **F13.921** sedative, hypnotic, or anxiolytic taken as prescribed (or **F13.931** if during withdrawal from sedative, hypnotic, or anxiolytic taken as prescribed); **F15.921** amphetamine-type substance or other stimulant taken as prescribed; **F16.921** ketamine or other hallucinogen taken as prescribed or for medical reasons; **F19.921** for medications that do not fit into any of the classes (e.g., dexamethasone) and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown (or **F19.931** if during withdrawal from medications that do not fit into any of the classes, taken as prescribed).

F05 Delirium due to another medical condition: There is evidence from the history, physical examination, or laboratory findings that the disturbance is attributable to the physiological consequences of another medical condition.

Coding note: Include the name of the other medical condition in the name of the delirium (e.g., F05 delirium due to hepatic encephalopathy). The other medical condition should also be coded and listed separately immediately before the delirium due to another medical condition (e.g., K72.90 hepatic encephalopathy; F05 delirium due to hepatic encephalopathy).

F05 Delirium due to multiple etiologies: There is evidence from the history, physical examination, or laboratory findings that the delirium has more than one etiology (e.g., more than one etiological medical condition; another medical condition plus substance intoxication or medication side effect).

Coding note: Use multiple separate codes reflecting specific delirium etiologies (e.g., K72.90 hepatic encephalopathy; F05 delirium due to hepatic failure; F10.231 alcohol withdrawal delirium). Note that the etiological medical condition both appears as a separate code that precedes the delirium code and is substituted into the delirium due to another medical condition rubric.

Recording Procedures

Substance intoxication delirium. The name of the substance intoxication delirium begins with the specific substance (e.g., cocaine) that is presumed to be causing the delirium. The diagnostic code is selected from the table included in the criteria set, which is based on the drug class and presence or absence of a comorbid substance use disorder. For substances that do not fit into any of the classes (e.g., dexamethasone), the code for "other substance" should be used; and in cases in which a substance is judged to be an etiological factor but the specific class of substance is unknown, the category "unknown substance" should be used.

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the name of the substance intoxication delirium, followed by the course (i.e., acute, persistent), followed by the specifier indicating level of psychomotor

activity (i.e., hyperactive, hypoactive, mixed level of activity). For example, in the case of acute hyperactive intoxication delirium occurring in a man with a severe cocaine use disorder, the diagnosis is F14.221 severe cocaine use disorder with cocaine intoxication delirium, acute, hyperactive. A separate diagnosis of the comorbid severe cocaine use disorder is not given. If the intoxication delirium occurs without a comorbid substance use disorder (e.g., after a one-time heavy use of the substance), no accompanying substance use disorder is noted (e.g., F16.921 phencyclidine intoxication delirium, acute, hypoactive).

Substance withdrawal delirium. The name of the substance withdrawal delirium begins with the specific substance (e.g., alcohol) that is presumed to be causing the withdrawal delirium. The diagnostic code is selected from substance-specific codes in the coding note included in the criteria set. When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word "with," followed by the substance withdrawal delirium, followed by the course (i.e., acute, persistent), followed by the specifier indicating level of psychomotor activity (i.e., hyperactive, hypoactive, mixed level of activity). For example, in the case of acute hyperactive withdrawal delirium occurring in a man with a severe alcohol use disorder, the diagnosis is F10.231 severe alcohol use disorder with alcohol withdrawal delirium, acute, hyperactive. A separate diagnosis of the comorbid severe alcohol use disorder is not given.

Medication-induced delirium. The name of the medication-induced delirium begins with the specific substance (e.g., dexamethasone) that is presumed to be causing the delirium. The name of the disorder is followed by the course (i.e., acute, persistent), followed by the specifier indicating level of psychomotor activity (i.e., hyperactive, hypoactive, mixed level of activity). For example, in the case of acute hyperactive medication-induced delirium occurring in a man using dexamethasone as prescribed, the diagnosis is F19.921 dexamethasone-induced delirium, acute, hyperactive.

Specifiers

Regarding course, in hospital settings, delirium usually lasts about 1 week, but some symptoms often persist even after individuals are discharged from the hospital.

Individuals with delirium may rapidly switch between hyperactive and hypoactive states. The hyperactive state may be more common or more frequently recognized and often is associated with medication side effects and drug withdrawal. The hypoactive state may be more frequent in older adults and is often unrecognized among older individuals in emergency departments and hospitals.

Diagnostic Features

The essential feature of delirium is an acute impairment of consciousness characterized by a disturbance in attention accompanied by reduced awareness of the environment, both core features of normal consciousness. Because these deficits reflect an altered state of consciousness affecting many higher cerebral cortical functions of the cerebral cortex, they are accompanied by a change from baseline in other cognitive functions that cannot be better explained by a preexisting or evolving neurocognitive disorder (NCD). The disturbance in attention (Criterion A) is manifested by reduced ability to direct, focus, sustain, and shift attention. Questions must be repeated because the individual's attention wanders, or the individual may perseverate with an answer to a previous question rather than appropriately shift attention. The individual is easily distracted by irrelevant stimuli. The disturbance in awareness affects both internal thinking and insight as well as difficulty making sense of what is happening in the external environment.

The disturbance develops over a short period of time, usually hours to a few days, and tends to fluctuate during the course of the day, often with worsening in the evening and night when external orienting stimuli decrease (Criterion B). There is evidence from the history, physical examination, or laboratory findings that the disturbance is a physiological consequence of an underlying medical condition, substance intoxication or withdrawal, use of a medication, or a toxin exposure, or a combination of these factors (Criterion E). The etiology should be coded according to the etiologically appropriate subtype (i.e., substance or medication intoxication,

substance withdrawal, another medical condition, or multiple etiologies). Delirium often occurs in the context of an underlying NCD. The impaired brain function of individuals with mild and major NCD renders them more vulnerable to developing a delirium.

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There is an accompanying change in at least one other area that may include memory and learning (particularly recent memory), disorientation (particularly to time and place), alteration in language (particularly semantic comprehension), or perceptual distortion or a perceptual-motor disturbance (Criterion C). The perceptual disturbances accompanying delirium include misinterpretations, illusions, or hallucinations; these disturbances are typically visual, but may occur in other modalities as well, and range from simple and uniform to highly complex.

Normal attention/arousal, delirium, and coma lie on a continuum. *Coma* is defined as a state of unconsciousness with an absence of cognition or sleep-wake cycle, along with the lack of any meaningful response to verbal or physical stimuli. *Delirium* is an impaired state of consciousness in the setting of an aroused cortex. The ability to evaluate cognition to diagnose delirium depends on there being a level of cortical arousal and wakefulness sufficient for response to verbal stimulation; hence, delirium should not be diagnosed in the context of coma (Criterion D). Stuporous individuals also have a reduced level of brain arousal, but not to the extent of the complete unconsciousness of coma. Coma and stupor can be due to neurological conditions or drug-induced as with iatrogenic deep sedation in intensive care unit (ICU) settings or general anesthesia. Those individuals who show only minimal responses to verbal or physical stimulation are incapable of engaging with attempts at standardized testing or even interview. This inability to engage should be classified as a disorder of arousal such as coma or stupor, and not as delirium. However, delirium can be a stage that follows emergence from coma or stupor, especially when coma is the result of a neurological condition. Further, the sleep-wake cycle disturbance characteristic of the circadian rhythm disturbance in delirium can interfere with full assessment of the individual if in a sleep phase, which should be distinguished from a disorder of brain arousal.

Associated Features

Delirium is often associated with a disturbance in the sleep-wake cycle. This disturbance can include daytime sleepiness, nighttime agitation, difficulty falling asleep, excessive sleepiness throughout the day, or wakefulness throughout the night. In some cases, complete reversal of the night-day sleep-wake cycle can occur. Sleep-wake cycle disturbances are very common in delirium and have been proposed as a core criterion for the diagnosis.

The individual with delirium may exhibit emotional disturbances, such as anxiety, fear, depression, irritability, anger, euphoria, and apathy. There may be rapid and unpredictable shifts from one emotional state to another. The disturbed emotional state may also be evident in calling out, screaming, cursing, muttering, moaning, or making other sounds. These behaviors are especially prevalent at night and under conditions in which stimulation and environmental cues are lacking.

Prevalence

The prevalence of delirium is highest among hospitalized older individuals and varies depending on the individuals' characteristics, setting of care, and sensitivity of the detection method. Data from the United States and Finland indicate that the prevalence of delirium in the community overall is low (1%–2%). The prevalence is 8%–17% in older individuals presenting to North American emergency departments, where the delirium often indicates a medical illness.

Based on data from various countries, the prevalence of delirium when individuals are admitted to the hospital ranges from 18% to 35%, and estimates of the occurrence of delirium arising during hospitalization range from 29% to 64% in general hospital populations. Internationally, delirium occurs in 11%–51% of older individuals postoperatively and in up to 81% of those in intensive care. The prevalence of delirium ranges from 20% to 22% in individuals in nursing homes or post–acute care settings and occurs in up to 88% of individuals with terminal illness at the end of life. Despite having higher risk factors for delirium, such as cardiovascular disease, sepsis, and respiratory failure, younger African Americans

tended to have lower rates of the occurrence of delirium compared with White individuals of similar age in a large case series of ICU patients in the United States.

Development and Course

The majority of individuals with delirium have a full recovery with or without treatment, especially those who are not elderly. Delirium may progress to stupor, coma, seizures, or death, particularly if undetected and the underlying cause(s) remains untreated.

There is increasing evidence that delirium may be associated in long-term follow-up with cognitive decline or major NCD in the elderly, particularly in those with preexisting underlying cognitive impairment. Mortality among hospitalized individuals with delirium is high; as many as 38%–41% of individuals with delirium die within 1 year after diagnosis; the risk of death is particularly great among those with malignancies and other significant underlying medical illness.

Risk and Prognostic Factors

Delirium may be increased in the context of functional impairment, preexisting cognitive impairment, sensory impairment (e.g., vision/hearing), increasing age, illness severity or comorbidity, infection, depression, history of stroke, and history of alcohol use. Both major and mild NCDs can increase the risk for delirium and complicate the course. Falls may be an outcome of delirium but are not found to be a risk factor. In a meta-analysis of studies from 1990 through 2016, anticholinergic use was not a validated predictor of delirium.

Older individuals are especially susceptible to delirium compared with younger adults. Among children, susceptibility to delirium in infancy and through childhood may be associated with significant childhood morbidity and mortality, whereas individuals in early adulthood through mid-adulthood may have less susceptibility to delirium and lower mortality risk.

Sex- and Gender-Related Diagnostic Issues

The symptoms associated with delirium may vary in men and women. Men more commonly manifest motor agitation and affective lability, whereas women more commonly manifest hypoactive delirium. Male sex is a risk factor for delirium, and sex- or gender-related factors may interact with other risk factors.

Diagnostic Markers

In addition to laboratory findings characteristic of underlying medical conditions (or intoxication or withdrawal states), there is often generalized irregular theta slowing on electroencephalography, and fast activity is occasionally found (e.g., in some cases of alcohol withdrawal delirium). However, electroencephalography is unable to detect slowing associated with delirium without comparison to premorbid baseline alpha rhythms unless the slowing is in the abnormal theta or delta frequency range.

Functional Consequences of Delirium

Delirium itself is associated with increased functional decline and risk of institutional placement. Hospitalized individuals 65 years or older with delirium are at greater risk for poor outcomes following discharge, including mortality, institutionalization, and dementia.

Differential Diagnosis

Psychotic disorders and bipolar and depressive disorders with psychotic features. Delirium that is characterized by vivid hallucinations, delusions, language disturbances, and agitation must be distinguished from brief psychotic disorder, schizophrenia,

schizophreniform disorder, and other psychotic disorders, as well as from manic or major depressive episodes, with psychotic features.

Acute stress disorder. Delirium associated with fear, anxiety, and dissociative symptoms, such as depersonalization, must be distinguished from acute stress disorder, which is precipitated by exposure to a severely traumatic event.

Malingering and factitious disorder. Delirium can be distinguished from these disorders on the basis of the often atypical symptomatic presentation in malingering and factitious disorder and the absence of another medical condition or substance that is etiologically related to the apparent cognitive disturbance.

Other neurocognitive disorders. The most common differential diagnostic issue when evaluating confusion in older adults is disentangling symptoms of delirium and major NCD. The clinician must determine whether the individual has delirium; a delirium superimposed on a preexisting NCD, such as that due to Alzheimer's disease; or an NCD without delirium. The traditional distinction between delirium and major NCD according to acuteness of onset and temporal course is particularly difficult in those elderly individuals who had a prior NCD that may not have been recognized, or who developed persistent cognitive impairment following an episode of delirium. When delirium and major NCD are comorbid, the management of the delirium should generally be given priority.

Other Specified Delirium

R41.0

This category applies to presentations in which symptoms characteristic of delirium that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for delirium or any of the disorders in the neurocognitive disorders diagnostic class. The other specified delirium category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for delirium or any specific neurocognitive disorder. This is done by recording "other specified delirium" followed by the specific reason (e.g., "subsyndromal delirium").

An example of a presentation that can be specified using the "other specified" designation is the following:

Subsyndromal delirium: A delirium-like presentation involving disturbances in attention, higher-level thought, and circadian rhythm, in which the severity of cognitive impairment falls short of that required for the diagnosis of delirium.

Unspecified Delirium

R41.0

This category applies to presentations in which symptoms characteristic of delirium that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for delirium or any of the disorders in the neurocognitive disorders diagnostic class. The unspecified delirium category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for delirium, and includes presentations for which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Major and Mild Neurocognitive Disorders

Major Neurocognitive Disorder

Diagnostic Criteria

A. Evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains (complex

attention, executive function, learning and memory, language, perceptual-motor, or social cognition) based on:

1. Concern of the individual, a knowledgeable informant, or the clinician that there has been a significant decline in cognitive function; and
2. A substantial impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.

B. The cognitive deficits interfere with independence in everyday activities (i.e., at a minimum, requiring assistance with complex instrumental activities of daily living such as paying bills or managing medications).

C. The cognitive deficits do not occur exclusively in the context of a delirium.

D. The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder, schizophrenia).

Specify whether due to:

Note: Each subtype listed has specific diagnostic criteria and corresponding text, which follow the general discussion of major and mild neurocognitive disorders.

Alzheimer's disease

Frontotemporal degeneration

Lewy body disease

Vascular disease

Traumatic brain injury

Substance/medication use

HIV infection

Prion disease

Parkinson's disease

Huntington's disease

Another medical condition

Multiple etiologies

Unspecified etiology

Coding note: Code based on medical or substance etiology. In most cases of major neurocognitive disorder, there is need for an additional code for the etiological medical condition, which must immediately precede the diagnostic code for major neurocognitive disorder, as noted in the coding table on pp. 682–683.

Specify (see coding table for details):

Without behavioral disturbance: If the cognitive disturbance is not accompanied by any clinically significant behavioral disturbance.

With behavioral disturbance *(specify disturbance):* If the cognitive disturbance is accompanied by a clinically significant behavioral disturbance (e.g., psychotic symptoms, mood disturbance, agitation, apathy, or other behavioral symptoms).

Coding note: Use additional code(s) to indicate clinically significant psychiatric symptoms due to the same medical condition causing the major neurocognitive disorder (e.g., **F06.2** psychotic disorder due to Alzheimer's disease, with delusions; **F06.32** depressive disorder due to Parkinson's disease, with major depressive–like episode). **Note:** Mental disorders due to another medical condition are included with disorders with which they share phenomenology (e.g., for depressive disorders due to another medical condition, see the chapter "Depressive Disorders").

Specify current severity:

Mild: Difficulties with instrumental activities of daily living (e.g., housework, managing money).

Moderate: Difficulties with basic activities of daily living (e.g., feeding, dressing).

Severe: Fully dependent.

Coding and Recording Procedures

The following are examples of coding and recording major neurocognitive disorders due to an etiological subtype *(for more information, see coding table on pp. 682–683 and coding notes in the specific diagnostic criteria for each major and mild neurocognitive disorder subtype)*:

Major neurocognitive disorder due to probable Alzheimer's disease, without behavioral disturbance, mild: G30.9 Alzheimer's disease, F02.80 major neurocognitive disorder due to probable Alzheimer's disease, without behavioral disturbance, mild.

Major neurocognitive disorder due to traumatic brain injury, with behavioral disturbance, moderate: S06.2X9S diffuse traumatic brain injury with loss of consciousness of unspecified duration, sequela; F02.81 major neurocognitive disorder due to traumatic brain injury, with behavioral disturbance, moderate; F06.34 bipolar and related disorder due to traumatic brain injury, with mixed features.

Mild Neurocognitive Disorder

Diagnostic Criteria

A. Evidence of modest cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition) based on:

1. Concern of the individual, a knowledgeable informant, or the clinician that there has been a mild decline in cognitive

function; and

2. A modest impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.

B. The cognitive deficits do not interfere with capacity for independence in everyday activities (i.e., complex instrumental activities of daily living such as paying bills or managing medications are preserved, but greater effort, compensatory strategies, or accommodation may be required).

C. The cognitive deficits do not occur exclusively in the context of a delirium.

D. The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder, schizophrenia).

Specify whether due to:

Note: Each subtype listed has specific diagnostic criteria and corresponding text, which follow the general discussion of major and mild neurocognitive disorders.

Alzheimer's disease

Frontotemporal degeneration

Lewy body disease

Vascular disease

Traumatic brain injury

Substance/medication use

HIV infection

Prion disease

Parkinson's disease

Huntington's disease

Another medical condition

Multiple etiologies

Unspecified etiology

Coding note: For mild neurocognitive disorder due to any of the medical etiologies listed above, code **G31.84.** Do *not* use additional codes for the presumed etiological medical conditions. For substance/medication-induced mild neurocognitive disorder, code based on type of substance; see "Substance/Medication-Induced Major or Mild Neurocognitive Disorder." For unspecified mild neurocognitive disorder, code **R41.9.**

Specify (behavioral disturbance cannot be coded but should still be recorded):

Without behavioral disturbance: If the cognitive disturbance is not accompanied by any clinically significant behavioral disturbance.

With behavioral disturbance *(specify disturbance):* If the cognitive disturbance is accompanied by a clinically significant behavioral disturbance (e.g., psychotic symptoms, mood disturbance, agitation, apathy, or other behavioral symptoms). **Coding note:** Use additional code(s) to indicate clinically significant psychiatric symptoms due to the same medical condition causing the mild neurocognitive disorder (e.g., **F06.2** psychotic disorder due to traumatic brain injury, with delusions; **F06.32** depressive disorder due to HIV disease, with major depressive–like episode). **Note:** Mental disorders due to another medical condition are included with disorders with which they share phenomenology (e.g., for depressive disorders due to another medical condition, see the chapter "Depressive Disorders").

Coding and Recording Procedures

The following are examples of coding and recording mild neurocognitive disorders due to an etiological subtype *(for more information, see coding table on pp. 682–683 and coding notes in*

the specific diagnostic criteria for each major and mild neurocognitive disorder subtype):

G31.84 Mild neurocognitive disorder due to Alzheimer's disease, without behavioral disturbance.

G31.84 Mild neurocognitive disorder due to traumatic brain injury, with behavioral disturbance; F06.34 bipolar and related disorder due to traumatic brain injury, with mixed features.

Etiological subtype	Associated etiological medical code for major neurocognitive disorder[a]	Major neurocognitive disorder code	Mild neurocognitive disorder code
Alzheimer's disease	G30.9	F02.8x[b]	G31.84[c] Do not use additional code for Alzheimer's disease.
Frontotemporal degeneration	G31.09	F02.8x[b]	G31.84[c] Do not use additional code for frontotemporal degeneration.
Lewy body disease	G31.83	F02.8x[b]	G31.84[c] Do not use additional code for Lewy body disease.
Vascular disease	No additional medical code.	F01.5x[b] Do not use additional code for the vascular disease.	G31.84[c] Do not use additional code for the vascular disease.
Traumatic brain injury	S06.2X9S	F02.8x[b]	G31.84[c] Do not use additional code for the traumatic brain injury.
Substance/medication-induced	No additional medical code.	Code based on the type of substance causing the major neurocognitive disorder.[d]	Code based on the type of substance causing the mild neurocognitive disorder.[d]
HIV infection	B20	F02.8x[b]	G31.84[c] Do not use additional code for HIV infection.

Etiological subtype	Associated etiological medical code for major neurocognitive disorder[a]	Major neurocognitive disorder code	Mild neurocognitive disorder code
Prion disease	A81.9	F02.8x[b]	G31.84[c] Do not use additional code for prion disease.
Parkinson's disease	G20	F02.8x[b]	G31.84[c] Do not use additional code for Parkinson's disease.
683 Huntington's disease	G10	F02.8x[b]	G31.84[c] Do not use additional code for Huntington's disease.
Due to another medical condition	Code the other medical condition first (e.g., G35 multiple sclerosis).	F02.8x[b]	G31.84[c] Do not use additional codes for the presumed etiological medical conditions.
Due to multiple etiologies	Code all of the etiological medical conditions first (with the exception of vascular disease).	F02.8x[b] (code once for major neurocognitive disorder due to all etiologies that apply) Code also major vascular NCD (F01.5x), if present. Code also the relevant substance/medication-induced major neurocognitive disorders if substances or medications play a role in the etiology.	G31.84[c] Do not use additional codes for the presumed etiological medical conditions. Code also the relevant substance/medication-induced mild neurocognitive disorders if substances or medications play a role in the etiology.
Unspecified neurocognitive disorder	No additional medical code.	R41.9[c]	R41.9[c]

[a]Code first, before code for major neurocognitive disorder.

[b]Code fifth character based on symptom specifier: .x0 without behavioral disturbance; .x1 with behavioral disturbance (e.g., psychotic symptoms, mood disturbance, agitation, apathy, or other

behavioral symptoms). **Note:** The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

[c]**Note:** "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.

[d]See coding table in "Substance/Medication-Induced Major or Mild Neurocognitive Disorder" for ICD-10-CM code. **Note:** The severity specifiers "mild," "moderate," and "severe" (for substance/medication-induced major neurocognitive disorder) and the accompanying symptom specifiers "with behavioral disturbance" and "without behavioral disturbance" (for substance/medication-induced major or mild neurocognitive disorder) cannot be coded but should still be recorded.

Subtypes

Major and mild neurocognitive disorders (NCDs) are primarily subtyped according to the known or presumed etiological/pathological entity or entities underlying the cognitive decline. These subtypes are distinguished on the basis of a combination of time course, characteristic domains affected, and associated symptoms. For certain etiological subtypes, the diagnosis depends substantially on the presence of a potentially causative entity, such as Parkinson's or Huntington's disease, or a traumatic brain injury or stroke in the appropriate time period. For other etiological subtypes (generally the neurodegenerative diseases like Alzheimer's disease, frontotemporal degeneration, and Lewy body disease), the diagnosis is based primarily on the cognitive, behavioral, and functional symptoms. Typically, the differentiation among these syndromes that lack an independently recognized etiological entity is clearer at the level of major NCD than at the level of mild NCD, but sometimes characteristic symptoms and associated features are present at the mild level as well.

NCDs are frequently managed by clinicians in multiple disciplines. For many subtypes, multidisciplinary international expert groups have developed specialized consensus criteria based on clinicopathological correlation with underlying brain pathology. The subtype criteria here have been harmonized with those expert criteria.

Specifiers

Evidence for distinct behavioral features in NCDs has been recognized, particularly in the areas of psychotic symptoms and depression. Psychotic features are common in many NCDs, particularly at the mild-to-moderate stage of major NCDs due to Alzheimer's disease, Lewy body disease, and frontotemporal degeneration. If the psychotic symptoms are judged to be due to the Alzheimer's disease, Lewy body disease, or frontotemporal degeneration, an additional diagnosis of psychotic disorder due to Alzheimer's disease, psychotic disorder due to Lewy body disease, or psychotic disorder due to frontotemporal degeneration may be given. Paranoia and other delusions are common features, and often a persecutory theme may be a prominent aspect of delusional ideation. In contrast to psychotic disorders with onset in earlier life (e.g., schizophrenia), disorganized speech and disorganized behavior are not characteristic of psychosis in NCDs. Hallucinations may occur in any modality, although visual hallucinations are more common in NCDs than in depressive, bipolar, or psychotic disorders.

Mood disturbances, including depression, anxiety, and elation, may occur. Depression is common early in the course (including at the mild NCD level) of NCD due to Alzheimer's disease and Parkinson's disease, while elation may occur more commonly in frontotemporal degeneration. If the mood disturbance is judged to be due to the Alzheimer's disease, Parkinson's disease, or frontotemporal degeneration, an additional diagnosis of depressive disorder due to Alzheimer's disease, depressive disorder due to Parkinson's disease, or bipolar and related disorder due to frontotemporal degeneration may be given. Mood symptoms are increasingly recognized to be a significant feature in the earliest stages of mild NCDs such that clinical recognition and intervention may be important.

Agitation is common in a wide variety of NCDs, particularly in major NCD of moderate to severe severity, and often occurs in the setting of confusion or frustration. It may arise as combative behaviors, particularly in the context of resisting caregiving duties such as bathing and dressing. Agitation is characterized as disruptive motor or vocal activity and tends to occur with advanced stages of cognitive impairment across all of the NCDs.

Individuals with NCD can present with a wide variety of behavioral symptoms that are the focus of treatment. Sleep disturbance is a common

symptom that can create a need for clinical attention and may include symptoms of insomnia, hypersomnia, and circadian rhythm disturbances.

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Apathy is common in mild and major NCD. It is observed particularly in NCD due to Alzheimer's disease and may be a prominent feature of NCD due to frontotemporal degeneration. Apathy is typically characterized by diminished motivation and reduced goal-directed behavior accompanied by decreased emotional responsiveness. Symptoms of apathy may manifest early in the course of NCDs when a loss of motivation to pursue daily activities or hobbies may be observed.

Other important behavioral symptoms include wandering, disinhibition, hyperphagia, and hoarding. Some of these symptoms are characteristic of specific disorders, as discussed in the relevant sections. When more than one behavioral disturbance is observed, each type should be noted in writing with the specifier "with behavioral disturbance."

Diagnostic Features

Major and mild NCDs exist on a spectrum of cognitive and functional impairment. Major NCD roughly corresponds to the condition labeled in ICD-10 and ICD-11 (as well as in DSM-IV) as *dementia*. The core feature of NCDs is acquired cognitive decline in one or more cognitive domains (Criterion A) based on both 1) a concern about cognition on the part of the individual, a knowledgeable informant, or the clinician, and 2) performance on an objective assessment that falls below the expected level or that has been observed to decline over time. Both a concern and objective evidence are required because they are complementary. When there is an exclusive focus on objective testing, a disorder may go undiagnosed in high-functioning individuals whose currently "normal" performance actually represents a substantial decline in abilities, or an illness may be incorrectly diagnosed in individuals whose currently "low" performance does not represent a change from their own baseline or is a result of extraneous factors like test conditions or a passing illness. Alternatively, excessive focus on subjective symptoms may fail to diagnose illness in individuals

with poor insight, or whose informants deny or fail to notice their symptoms, or it may be overly sensitive in the so-called worried well.

A cognitive concern differs from a complaint in that it may or may not be voiced spontaneously. Rather, it may need to be elicited by careful questioning about specific symptoms that commonly occur in individuals with cognitive deficits (see Table 1 in the introduction to this chapter). For example, memory concerns include difficulty remembering a short grocery list or keeping track of the plot of a television program; executive concerns include difficulty resuming a task when interrupted, organizing tax records, or planning a holiday meal. At the mild NCD level, the individual is likely to describe these tasks as being more difficult or as requiring extra time or effort or compensatory strategies. At the major NCD level, such tasks may only be completed with assistance or may be abandoned altogether. At the mild NCD level, individuals and their families may not notice such symptoms or may view them as normal, particularly in the elderly; thus, careful history taking is of paramount importance. The difficulties must represent changes rather than lifelong patterns: the individual or informant may clarify this issue, or the clinician can infer change from prior experience with the individual or from occupational or other clues. It is also critical to determine that the difficulties are related to cognitive loss rather than to motor or sensory limitations.

Neuropsychological testing, with performance compared with norms appropriate to the individual's age, sex, educational attainment, and cultural background, is part of the standard evaluation of NCDs and is particularly critical in the evaluation of mild NCD. The use of culturally validated assessment instruments is preferred, which are available for many racial/ethnic and linguistic populations. For major NCD, performance is typically 2 or more standard deviations below appropriate norms (3rd percentile or below). For mild NCD, performance typically lies in the 1–2 standard deviation range (between the 3rd and 16th percentiles). However, neuropsychological testing is not available in all settings, and neuropsychological thresholds are sensitive to the specific test(s) and norms employed, as well as to test conditions, sensory limitations, and intercurrent illness. A variety of brief

office-based or “bedside” assessments, as described in Table 1, can also supply objective data in settings where such testing is unavailable or infeasible. In any case, as with cognitive concerns, objective performance must be interpreted in light of the individual’s prior performance. Optimally, this information would be available from a prior administration of the same test, but often it must be inferred based on appropriate norms, along with the individual’s educational history, occupation, and other factors. Norms are more challenging to interpret in individuals with very high or very low levels of education and in individuals being tested outside their own language or cultural background.

Criterion B relates to the individual’s level of independence in everyday functioning. Individuals with major NCD will have impairment of sufficient severity so as to interfere with independence, such that others will have to take over tasks that the individuals were previously able to complete on their own. Individuals with mild NCD will have preserved independence, although there may be subtle interference with function or a report that tasks require more effort or take more time than previously.

The distinction between major and mild NCD is inherently arbitrary, and the disorders exist along a continuum. Precise thresholds are therefore difficult to determine. Careful history taking, observation, and integration with other findings are required, and the implications of making a diagnosis should be considered when an individual’s clinical manifestations lie at a boundary.

Associated Features

Typically the associated features that support a diagnosis of major or mild NCD will be specific to the etiological subtype (e.g., neuroleptic sensitivity and visual hallucinations in NCD due to Lewy body disease). Diagnostic features specific to each of the subtypes are found in the relevant sections.

Prevalence

The prevalence of NCD varies widely by age and by etiological subtype. Overall prevalence estimates are generally only available for older populations. Among individuals older than 60 years, prevalence increases steeply with age, so prevalence estimates are more accurate for narrow age

bands than for broad categories such as “over 65” (where the mean age can vary greatly with the life expectancy of the given population). For those etiological subtypes occurring across the life span, prevalence estimates for NCD are likely to be available, if at all, only as the fraction of individuals who develop NCD among those with the relevant condition (e.g., traumatic brain injury, HIV infection).

Female gender is associated with higher prevalence of dementia overall, and especially Alzheimer’s disease, but this difference is largely, if not wholly, attributable to greater longevity in females.

Overall, international prevalence estimates for dementia (which is largely congruent with major NCD) are approximately 1%–2% at age 65 years and as high as 30% by age 85 years. The prevalence of mild NCD is very sensitive to the definition of the disorder, particularly in community settings, where evaluations are less detailed. In addition, in contrast with clinical settings, where cognitive concern must be high to seek and locate care, there may be a less clear decline from baseline functioning. Estimates of the prevalence of mild cognitive impairment (which is substantially congruent with mild NCD) among older individuals are fairly variable, ranging from 2% to 10% at age 65 and 5% to 25% by age 85.

Prevalence and incidence of dementia vary cross-nationally and among ethnic and racialized populations in the United States, although methodological differences complicate rate comparisons. Some U.S. studies found that incidence is highest in African Americans followed, in decreasing order, by American Indians/Alaska Natives, Latinx, Pacific Islanders, non-Latinx Whites, and Asian Americans. Among four Asian American populations, Filipino Americans had the highest incidence, followed by Japanese Americans, Chinese

Americans, and Asian-Indian Americans. Latinx subpopulations in the United States have been found to vary considerably in prevalence and incidence of dementia; Caribbean Hispanics have much higher rates than Mexican Americans in some U.S. studies.

Development and Course

The course of NCD varies across etiological subtypes, and this variation can be useful in differential diagnosis. Some subtypes (e.g., those related to traumatic brain injury or stroke) typically begin at a specific time and (at least after initial symptoms related to inflammation or swelling subside) remain static. Others may fluctuate over time (although if this occurs, the possibility of delirium superimposed on NCD should be considered). NCDs due to neurodegenerative diseases like Alzheimer's disease or frontotemporal degeneration typically are marked by insidious onset and gradual progression, and the pattern of onset of cognitive deficits and associated features helps to distinguish among them.

NCDs with onset in childhood and adolescence may have broad repercussions for social and intellectual development, and in this setting intellectual developmental disorder (intellectual disability) or other neurodevelopmental disorders may also be diagnosed to capture the full diagnostic picture and ensure the provision of a broad range of services. In older individuals, NCDs often occur in the setting of medical illnesses, frailty, and sensory loss, which complicate the clinical picture for diagnosis and treatment.

When cognitive loss occurs in youth to midlife, individuals and families are likely to seek care. NCDs are typically easiest to identify at younger ages, although in some settings malingering or factitious disorder may be a concern. Very late in life, cognitive symptoms may not cause concern or may go unnoticed. In late life, mild NCD must also be distinguished from the more modest deficits associated with "normal aging," although a substantial fraction of what has been ascribed to normal aging likely represents prodromal phases of various NCDs. In addition, it becomes harder to recognize mild NCD with age because of the increasing prevalence of medical illness and sensory deficits. It becomes harder to differentiate among subtypes with age because there are multiple potential sources of neurocognitive decline.

Risk and Prognostic Factors

Risk factors vary not only by etiological subtype but also by age at onset within etiological subtypes. Some subtypes are distributed throughout the life span, whereas others occur exclusively or primarily in late life. Even

within the NCDs of aging, the relative prevalence varies with age: Alzheimer's disease is uncommon before age 60 years, and the prevalence increases steeply thereafter, while the overall less common frontotemporal degeneration has earlier onset and represents a progressively smaller fraction of NCDs with age. The strongest risk factor for major and mild NCDs is age, primarily because age increases the risk of neurodegenerative and cerebrovascular disease.

Risk of NCDs varies by ethnic and racialized background and is associated with variation in risk of underlying diseases (e.g., hypertension, diabetes), predisposing conditions (e.g., head injury), environment (e.g., access to nutritious food, safe spaces for exercise), and other factors. For example, in the United States, African Americans and Latinx tend to be at higher risk for vascular dementia than Whites. Lower education and literacy are risk factors for NCDs that also can vary by ethnoracial group because of differential exposure to adverse social determinants of health.

Culture-Related Diagnostic Issues

Individuals' and families' level of awareness and concern about neurocognitive symptoms may vary across ethnic, racialized, and occupational groups. Cultural differences regarding whether decreased cognitive ability is seen as a normal part of aging ("normalization") and in dementia-related stigma can delay families' recognition of a problem and

decrease help seeking for individuals in the early stages of cognitive loss. For example, social stigma appears to be associated with underutilization of services for cognitive impairment among some underserved ethnic and racialized groups (e.g., Chinese Americans, Korean Americans).

Neurocognitive symptoms are more likely to be noticed, particularly at the mild level, in individuals who engage in complex occupational, domestic, or recreational activities. In addition, norms for neuropsychological testing tend to be available only for broad populations, and thus they may not be easily applicable to individuals with less than high school education or those being evaluated outside their primary language or

culture. Culturally related diagnostic challenges include accounting for intraethnic variation in the interpretation of assessments; evaluating the effect on neuropsychological testing of a) the test taker's stereotype threat (i.e., anxiety from concerns that he or she will confirm the negative stereotype of the ethnic or racialized group by underperforming) and/or b) the clinician's implicit (unconscious) bias on test interpretation; and selecting the appropriate language when assessing bilingual individuals.

Bilingual individuals with dementia may lose their facility with acquired nonnative languages, which might affect their ability to communicate with caregivers. The caregiving environment may be influenced by cultural norms of family responsibility to care for the elderly, for example, by affecting the decision whether to care for the elder with NCD at home or in a care facility. In some cultures, adult children are expected to provide care for their older parents (e.g., filial piety) so that a functional limitation may not be as obvious to the dependent elder or the family.

Sex- and Gender-Related Diagnostic Issues

Some studies show that men and women experience major and mild NCD differently. Sex- and gender-related factors may influence incidence and prevalence, the etiology (risk and protective factors), and the clinical manifestations of major and mild NCD. More women than men experience major NCD because of their longer life span. Thus, a woman of a given age has a higher cumulative risk of developing major NCD before death than a man of the same age. The difference in incidence rates is less clear and may vary across populations and over time because of gender-related factors (e.g., education, occupation, family role, stress). For example, the incidence of dementia in several higher-income countries has declined in the past 30 years, and the decline was different in men and women across countries. Women tend to express a broader range of symptoms. In particular, women tend to manifest more psychiatric symptoms such as depression, anxiety, and delusions. Men tend to manifest more aggression, apathy, and vegetative symptoms.

Like age, culture, and occupation, sex and gender issues may affect the level of concern and awareness of cognitive symptoms. In addition, for late-

life NCDs, women are likely to be older, to have more medical comorbidity, and to live alone, which can complicate evaluation and treatment. In addition, there are sex and gender differences in the frequency of some of the etiological subtypes.

Diagnostic Markers

In addition to a careful history, neuropsychological assessments are the key measures for diagnosis of NCDs, particularly at the mild level, where functional changes are minimal and symptoms more subtle. Ideally, individuals will be referred for formal neuropsychological testing, which will provide a quantitative assessment of all relevant domains and thus help with diagnosis; provide guidance to the family on areas where the individual may require more support; and serve as a benchmark for further decline or response to therapies. When such testing is unavailable or not feasible, the brief assessments in Table 1 can provide insight into each domain. More global brief mental status tests may be helpful but may be

insensitive, particularly to modest changes in a single domain or in those with high premorbid abilities, and may be overly sensitive in those with low premorbid abilities.

In distinguishing among etiological subtypes, additional diagnostic markers may come into play, particularly neuroimaging studies such as magnetic resonance imaging scans and positron emission tomography scans. In addition, specific markers may be involved in the assessment of specific subtypes and may become more important as additional research findings accumulate over time, as discussed in the relevant sections.

Association With Suicidal Thoughts or Behavior

Large-scale studies indicate elevated rates of suicidal behavior in individuals with NCD due to a variety of etiologies compared with persons without an NCD. A nationwide study in Taiwan reported that attempted suicide in late life is associated with subsequent dementia.

Functional Consequences of Major and Mild Neurocognitive Disorders

By definition, major and mild NCDs affect functioning, given the central role of cognition in human life. Thus, the criteria for the disorders, and the threshold for differentiating mild from major NCD, are based in part on functional assessment. Within major NCD there is a broad range of functional impairment, as implemented in the severity specifiers. In addition, the specific functions that are compromised can help identify the cognitive domains affected, particularly when neuropsychological testing is not available or is difficult to interpret.

Differential Diagnosis

Normal cognition. The differential diagnosis between normal cognition and mild NCD, as between mild and major NCD, is challenging because the boundaries are inherently arbitrary. Careful history taking and objective assessment are critical to these distinctions. A longitudinal evaluation using quantified assessments may be key in detecting mild NCD.

Delirium. Both mild and major NCD may be difficult to distinguish from a persistent delirium, which can co-occur. Careful assessment of attention and arousal will help to make the distinction.

Major depressive disorder. The distinction between mild NCD and major depressive disorder, which may co-occur with NCD, can also be challenging. Specific patterns of cognitive deficits may be helpful. For example, consistent memory and executive function deficits are typical of Alzheimer's disease, whereas nonspecific or more variable performance is seen in major depression. Alternatively, treatment of the depressive disorder with repeated observation over time may be required to make the diagnosis.

Specific learning disorder and other neurodevelopmental disorders. A careful clarification of the individual's baseline status will help distinguish an NCD from a specific learning disorder or other neurodevelopmental disorders. Additional issues may enter the differential for specific etiological subtypes, as described in the relevant sections.

Comorbidity

NCDs are common in older individuals and thus often co-occur with a wide variety of age-related diseases that may complicate diagnosis or treatment. Most notable of these is delirium, for which NCD increases the risk. In older individuals, a delirium during hospitalization is, in many cases, the first time that an NCD is noticed, although a careful history will often reveal evidence of earlier decline. Mixed NCDs are also common in older

individuals, as many etiological entities increase in prevalence with age. In younger individuals, NCD often co-occurs with neurodevelopmental disorders; for example, a head injury in a preschool child may also lead to significant developmental and learning issues. Additional comorbidity of NCD is often related to the etiological subtype, as discussed in the relevant sections.

Major or Mild Neurocognitive Disorder Due to Alzheimer's Disease

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. There is insidious onset and gradual progression of impairment in one or more cognitive domains (for major neurocognitive disorder, at least two domains must be impaired).
C. Criteria are met for either probable or possible Alzheimer's disease as follows:

For major neurocognitive disorder:

Probable Alzheimer's disease is diagnosed if either of the following is present; otherwise, **possible Alzheimer's disease** should be diagnosed.

1. Evidence of a causative Alzheimer's disease genetic mutation from family history or genetic testing.
2. All three of the following are present:
    a. Clear evidence of decline in memory and learning and at least one other cognitive domain (based on detailed history or serial neuropsychological testing).
    b. Steadily progressive, gradual decline in cognition, without extended plateaus.
    c. No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological, mental, or systemic disease or condition likely contributing to cognitive decline).

For mild neurocognitive disorder:

Probable Alzheimer's disease is diagnosed if there is evidence of a causative Alzheimer's disease genetic mutation from either genetic testing or family history.

Possible Alzheimer's disease is diagnosed if there is no evidence of a causative Alzheimer's disease genetic mutation from either genetic testing or family history, and all three of the following are present:

1. Clear evidence of decline in memory and learning.
2. Steadily progressive, gradual decline in cognition, without extended plateaus.
3. No evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease, or another neurological or systemic disease or condition likely contributing to cognitive decline).

D. The disturbance is not better explained by cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder due to probable or possible Alzheimer's disease, with behavioral disturbance, code first **G30.9** Alzheimer's disease, followed by **F02.81.** For major neurocognitive disorder due to probable or possible Alzheimer's disease, without behavioral disturbance, code first **G30.9** Alzheimer's disease, followed by **F02.80.**

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Note: The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild neurocognitive disorder due to Alzheimer's disease, code **G31.84.** (**Note:** Do *not* use the additional code for Alzheimer's disease. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild neurocognitive disorder due to Alzheimer's disease: Use additional code(s) to indicate clinically significant psychiatric symptoms due to Alzheimer's disease (e.g., **F06.2** psychotic disorder due to Alzheimer's disease, with delusions; **F06.32** depressive disorder due to Alzheimer's disease, with major depressive–like episode).

Diagnostic Features

Beyond the neurocognitive disorder (NCD) syndrome (Criterion A), the core features of major or mild NCD due to Alzheimer's disease include an insidious onset and gradual progression of cognitive and behavioral symptoms (Criterion B). The typical presentation is amnestic (i.e., with impairment in memory and learning). Unusual nonamnestic presentations, particularly visuospatial and logopenic aphasic variants, also exist. A significant proportion of individuals, likely more than half, first present with behavioral symptoms before the onset of cognitive symptoms; the presence of behavioral disturbance should be noted using the appropriate specifier codes. At the mild NCD phase, Alzheimer's disease manifests

typically with impairment in memory and learning, sometimes accompanied by deficits in executive function. At the major NCD phase, visuoconstructional/perceptual-motor ability and language (e.g., word retrieval) will also be impaired, particularly when the NCD is moderate to severe. Social cognition tends to be preserved until late in the course of the disease with the exception of individuals who have the less common variants with significant dysexecutive and behavioral disturbance.

A level of diagnostic certainty must be specified denoting Alzheimer's disease as the "probable" or "possible" etiology (Criterion C). *Probable Alzheimer's disease* is diagnosed in both major and mild NCD if there is evidence of a causative Alzheimer's disease gene, either from genetic testing or from an autosomal dominant family history coupled with autopsy confirmation or a genetic test in an affected family member. At present, the designation of "probable" represents the highest level of diagnostic certainty within the current criteria framework. However, current developments in biomarkers continue to increase diagnostic certainty (e.g., when brain positron emission tomography [PET] scans may indicate the presence of Alzheimer's pathology, such as evidence of amyloid and/or tau deposition by imaging or cerebrospinal [CSF] analysis). For major NCD, a typical clinical picture, without extended plateaus or evidence of mixed etiology, can also be diagnosed as due to probable Alzheimer's disease. However, in some individuals there may be prolonged periods of very slow or minimal progression. For mild NCD, given the lesser degree of certainty that the deficits will progress, these features are only sufficient for a *possible* Alzheimer's etiology. As stated above, however, new biomarker methods may affect the use of "probable" and "possible" in mild NCD. If the etiology appears mixed, mild NCD due to multiple etiologies should be diagnosed. In any case, for both mild and major NCD due to Alzheimer's disease, the clinical features must not suggest another primary etiology for the NCD (Criterion D). As biomarker data continue to inform the nature of underlying pathologies, it is likely that the existence of multiple etiologies will be more systematically mapped in the future to better identify diagnostic variations in NCD due to multiple etiologies.

Associated Features

For individuals with NCD due to Alzheimer's disease, symptoms extend beyond cognitive deficits to include neuropsychiatric symptoms such as agitation, apathy, depression, delusions, and sleep disorders. Neuropsychiatric symptoms may also be described as

behavioral and psychological symptoms of dementia and have been observed in neurocognitive disorders of all etiologies. These symptoms are nearly universal in Alzheimer's disease as confirmed in two U.S. population samples, with 5-year follow-up in one reporting that 98% of individuals with NCD due to Alzheimer's disease develop neuropsychiatric symptoms. Neuropsychiatric symptoms lead to disability, worsening quality of life, greater impairment in activities of daily living, faster cognitive and functional decline, greater caregiver burden, earlier institutionalization, and accelerated mortality. Neuropsychiatric symptoms are often more distressing than cognitive manifestations and are frequently the reason that health care assistance is sought. These symptoms are also frequently present at the mild NCD stage, with evidence suggesting that more than half of individuals who develop dementia begin with neuropsychiatric symptoms. At the mild NCD stage or the mildest level of major NCD, depression, irritability, and/or apathy are most often seen. With moderately severe major NCD, delusions, agitation, combativeness, and wandering are common. Late in the illness, gait disturbance, dysphagia, incontinence, myoclonus, and seizures are observed.

Prevalence

The prevalence of overall NCD due to Alzheimer’s disease rises steeply with age. In high-income countries, it ranges from 5% to 10% in individuals ages 60–69 years to at least 25% thereafter. An estimated 5.4 million Americans of all ages had dementia due to Alzheimer’s disease in 2016, including about 200,000 individuals with disease onset before age 65. Dementia due to Alzheimer’s disease is found in 11% of individuals age 65 and older and 32% of those age 85 and older. Estimates applying incidence rates of dementia due to Alzheimer’s disease to U.S. census data indicate

that 81% of those with the disease are age 75 or older. The percentage of dementias attributable to Alzheimer's disease ranges from about 60% to over 90%, depending on the setting and diagnostic criteria. Mild NCD due to Alzheimer's disease is likely to represent a substantial fraction of mild cognitive impairment (MCI) as well.

Studies show that prevalence of dementia due to Alzheimer's disease tends to vary by ethnoracial background; for example, in the United States, prevalence in individuals age 65 years and older ranges from 3.5% to 14.4%, depending on ethnoracial group, age, and assessment methodology. Higher prevalence has been found among African Americans and U.S. Latinx of Caribbean origin, after adjustment for gender and clinical comorbidities.

Development and Course

Major or mild NCD due to Alzheimer's disease progresses gradually, at times with plateaus, through severe dementia to death. The mean duration of survival after diagnosis is approximately 10 years, reflecting the advanced age of the majority of individuals rather than the course of the disease; some individuals can live with the disease for as long as 20 years. Late-stage individuals are eventually mute and bedbound. Death most commonly results from aspiration in those who survive through the full course. In mild NCD due to Alzheimer's disease, impairments increase over time, and functional status gradually declines until symptoms reach the threshold for the diagnosis of major NCD.

The onset of symptoms is usually at ages 70 through 89; early-onset forms seen in individuals ages 40–59 are often, but not always, related to known causative mutations. Symptoms and pathology do not differ much by onset ages. However, younger individuals are more likely to survive the full course of the disease, while older individuals are more likely to have numerous medical comorbidities that affect the course and management of the illness. Diagnostic complexity is higher in older adults because of the increased likelihood of comorbid medical illness and mixed pathology. Age at symptom onset, rate of cognitive decline, and survival rates appear to vary by ethnoracial background. For example, compared

with non-Latinx Whites, U.S. Latinx can develop Alzheimer's disease symptoms up to 4 years earlier, African Americans tend to show slower cognitive decline, and both underserved groups may have longer survival periods.

Risk and Prognostic Factors

A number of risk factors have been identified, including low educational status, midlife hypertension, obesity, and hearing loss, as well as late-life smoking, depression, physical inactivity, social isolation, and diabetes. The co-occurrence of multiple vascular risk factors also increases risk for Alzheimer's disease and may act by increasing cerebrovascular pathology or also through direct effects on Alzheimer's pathology. Traumatic brain injury, especially in men, may increase risk for major or mild NCD due to Alzheimer's disease, although this relationship remains controversial.

Genetic and physiological. Age is definitively the strongest risk factor for Alzheimer's disease, as the prevalence estimates demonstrate. A strong genetic predisposition (60%–80% of attributable risk) has been demonstrated. Rare mutations on chromosomes 1, 14, and 21 follow Mendelian inheritance, leading to autosomal dominant forms. Individuals with Down syndrome (trisomy 21) may develop Alzheimer's disease if they survive to midlife. The most common risk factors are polygenic, with more than 45 risk genes/loci having been identified, typically with small effects on risk. The strongest genetic susceptibility polymorphism, apolipoprotein E4 (*APOE*E4*), increases risk and decreases age at onset, particularly in homozygous individuals, although some homozygous individuals survive to advanced ages without developing symptoms.

Ethnoracial and national origin are related to the genetic susceptibility profile for Alzheimer's disease. While *APOE*E4* is associated with Alzheimer's disease risk, this association has not been consistently found across all ethnic and racialized groups. For example, some studies have identified a unique mutation in the *Gly206Ala* presenilin 1 gene among individuals of Puerto Rican descent with Alzheimer's disease, which is also related to early onset. Moreover, some studies have found a stronger

association with *ABCA7*, a protein transporter gene, among individuals who identify as African American than among U.S. Whites.

Culture-Related Diagnostic Issues

Detection of an NCD may be more difficult in cultural and socioeconomic settings where memory loss is considered normal in old age, where older adults face fewer cognitive demands in everyday life, or where very low educational levels pose greater challenges to objective cognitive assessment.

Sex- and Gender-Related Diagnostic Issues

Women were found to have a higher incidence of Alzheimer's disease than men in several European studies, but the incidence was similar in men and women in most North American studies. Some studies suggested that the symptoms of dementia progress faster in women than in men. However, because women perform better than men of the same age on some verbal memory tests, it is also possible that gender differences reflect the cut-off scores of tests used to support a diagnosis. Different cut-off scores may be useful in men and women when assessing for mild cognitive impairment.

Diagnostic Markers

Amyloid-predominant neuritic plaques, tau-predominant neurofibrillary tangles, and neuronal loss observed microscopically or manifested in regional cortical atrophy (e.g., hippocampal, parietal, frontal) are hallmarks of the pathological diagnosis of Alzheimer's disease and may be confirmed via postmortem histopathological examination. For

early-onset cases with apparent autosomal dominant inheritance, a mutation in one of the known causative Alzheimer's disease genes—amyloid precursor protein (*APP*), presenilin 1 (*PSEN1*), or presenilin 2 (*PSEN2*)—may be involved, and genetic testing for such mutations is commercially available, although usually without clinical utility. While *APOE E*4* cannot serve as a diagnostic marker because it is a risk factor (i.e., neither

necessary nor sufficient for disease occurrence), in rare instances genetic testing at this locus may have utility in clinical settings.

Since amyloid beta-42 deposition in the brain occurs early in the pathophysiological cascade, amyloid-based diagnostic tests such as amyloid imaging on brain PET scans and reduced levels of amyloid beta-42 in the CSF may have diagnostic value. Similarly, tau PET imaging or CSF analyses for elevated total tau or phospho-tau levels are available for clinical use. Signs of neuronal injury, such as hippocampal and temporoparietal cortical atrophy on a magnetic resonance image scan and temporoparietal hypometabolism on a fluorodeoxyglucose PET scan, provide evidence of neuronal damage but are less specific for Alzheimer's disease. Most of these biomarkers have been validated and are widely available in tertiary care settings. Blood-derived biomarkers for Alzheimer's disease are being developed and are likely to become clinically available as diagnostic, prognostic, and theranostic indicators.

Association With Suicidal Thoughts or Behavior

Alzheimer's disease is associated with a moderate risk of suicide even many years after the diagnosis; thus, ongoing assessment of mood and suicidality is appropriate. A large population study in Denmark found that the risk of suicide in individuals with a hospital-determined diagnosis of dementia was three- to eightfold greater compared with persons without dementia. In contrast, several other studies found mixed results regarding suicide risk in individuals with Alzheimer's disease. A review of the neurobiology of suicide in the elderly found preliminary evidence of an association with cognitive deficits and elderly suicidal behavior, especially regarding impaired decision-making and reduced cognitive inhibition.

Functional Consequences of Major or Mild Neurocognitive Disorder Due to Alzheimer's Disease

Because of the effect on cognition, behavior, and functioning, NCD due to Alzheimer's disease has a serious and substantial impact on individuals, their caregivers, and families. Early in the disease course, memory loss, disorientation, and mood symptoms adversely impact independence, and create safety concerns (e.g., around driving). For individuals with onset at

younger ages, NCD due to Alzheimer's disease can lead to early retirement. As the disease advances, individuals become increasingly disabled in instrumental and basic daily living activities, slowly becoming fully dependent on others. Caregivers for individuals with NCD due to Alzheimer's disease often see their social network deteriorate and develop a series of health and mental health problems that can adversely affect outcomes for both the caregiver and the individual with the NCD.

Differential Diagnosis

Other neurocognitive disorders. Major and mild NCDs due to other neurodegenerative processes (e.g., Lewy body disease, frontotemporal degeneration) share the insidious onset and gradual decline caused by Alzheimer's disease but have distinctive core features of their own (which are not always present). For example, NCD with Lewy bodies is typically characterized by frequent fluctuations in cognition early in the disease, parkinsonian features, gait imbalances, and visual hallucinations. Individuals with frontotemporal NCD may present with a distinct behavioral or language variant. The behavioral variant typically

first manifests with prominent changes in social behavior, such as disinhibition, apathy, or perseverative behavior, that may not infrequently lead to a primary psychiatric diagnosis. In contrast, the language variant of frontotemporal NCD may manifest with impairments in expressive language or word comprehension.

In major or mild vascular NCD, there is typically a history of stroke temporally related to the onset of cognitive impairment, and infarcts or hemosiderin deposits observed on brain imaging can be judged sufficient to account for the clinical picture. However, major or mild vascular NCD shares many clinical features with Alzheimer's disease; frequently Alzheimer's pathology is present alone or in combination with vascular pathologies. It should be noted that white matter change alone does not constitute enough evidence of cerebrovascular disease to propose a mixed etiology if the other diagnostic considerations support the diagnosis of NCD

due to Alzheimer's disease. The presence of subcortical ischemic changes on neuroimaging must be interpreted carefully in view of whether concurrent Alzheimer's pathology is present.

Other concurrent, active neurological or systemic illness. Other neurological or systemic illness should be considered if there is an appropriate temporal relationship and severity to account for the clinical picture. At the mild NCD level, it may be difficult to distinguish an Alzheimer's disease etiology from that of another medical condition (e.g., thyroid disorders, vitamin B_{12} deficiency).

Major depressive disorder. Particularly at the mild NCD level, the differential diagnosis also includes major depression. The presence of depression may be associated with reduced daily functioning and poor concentration that may resemble an NCD, but improvement with treatment of depression may be useful in making the distinction. If the symptoms meeting criteria for a major depressive episode are judged to be due to the physiological effects of Alzheimer's disease, a diagnosis of depressive disorder due to Alzheimer's disease, with major depressive–like episode, should be given instead of major depressive disorder.

Comorbidity

Most individuals with Alzheimer's disease are elderly and have multiple medical conditions that can complicate diagnosis and influence the clinical course. Major or mild NCD due to Alzheimer's disease commonly co-occurs with cerebrovascular disease, which contributes to the clinical picture. When a comorbid condition contributes to the NCD in an individual with Alzheimer's disease, then NCD due to multiple etiologies should be diagnosed.

Major or Mild Frontotemporal Neurocognitive Disorder

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. The disturbance has insidious onset and gradual progression.
C. Either (1) or (2):
    1. Behavioral variant:
        a. Three or more of the following behavioral symptoms:
            i. Behavioral disinhibition.
            ii. Apathy or inertia.
            iii. Loss of sympathy or empathy.

            iv. Perseverative, stereotyped or compulsive/ritualistic behavior.
            v. Hyperorality and dietary changes.
        b. Prominent decline in social cognition and/or executive abilities.
    2. Language variant:
        a. Prominent decline in language ability, in the form of speech production, word finding, object naming, grammar, or word comprehension.
D. Relative sparing of learning and memory and perceptual-motor function.
E. The disturbance is not better explained by cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder.

Probable frontotemporal neurocognitive disorder is diagnosed if either of the following is present; otherwise, **possible frontotemporal neurocognitive disorder** should be diagnosed:

1. Evidence of a causative frontotemporal neurocognitive disorder genetic mutation, from either family history or genetic testing.

2. Evidence of disproportionate frontal and/or temporal lobe involvement from neuroimaging.

Possible frontotemporal neurocognitive disorder is diagnosed if there is no evidence of a genetic mutation, and neuroimaging has not been performed.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder due to probable or possible frontotemporal degeneration, with behavioral disturbance, code first **G31.09** frontotemporal degeneration, followed by **F02.81.** For major neurocognitive disorder due to probable or possible frontotemporal degeneration, without behavioral disturbance, code first **G31.09** frontotemporal degeneration, followed by **F02.80.**

Note: The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild neurocognitive disorder due to frontotemporal degeneration, code **G31.84.** (**Note:** Do *not* use the additional code for frontotemporal degeneration. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild frontotemporal neurocognitive disorder: Use additional code(s) to indicate clinically significant psychiatric symptoms due to frontotemporal degeneration (e.g., **F06.33** bipolar and related disorder due to frontotemporal degeneration, with manic features; **F07.0** personality change due to frontotemporal degeneration, disinhibited type).

Diagnostic Features

Major or mild frontotemporal neurocognitive disorder (NCD) comprises a number of syndromic variants characterized by the progressive development of behavioral and personality change and/or language impairment. The behavioral variant and two language variants (semantic and agrammatic/nonfluent) exhibit distinct patterns of brain atrophy and

some distinctive neuropathology. The criteria must be met for either the behavioral or the language variant to make the diagnosis, but many individuals present with features of both.

Individuals with behavioral-variant major or mild frontotemporal NCD present with varying degrees of apathy or disinhibition. They may lose interest in socialization, self-care, and personal responsibilities, or display socially inappropriate behaviors. Insight is usually impaired, and this often delays medical consultation. The first referral is often to a psychiatrist. Individuals may develop changes in social style, and in religious and political

beliefs, with repetitive movements, hoarding, changes in eating behavior, and hyperorality. In later stages, loss of sphincter control may occur. Cognitive decline is less prominent, and formal testing may show relatively few deficits in the early stages. Common neurocognitive symptoms are lack of planning and organization, distractibility, and poor judgment. Deficits in executive function, such as poor performance on tests of mental flexibility, abstract reasoning, and response inhibition, are present, but learning and memory are relatively spared, and perceptual-motor abilities are almost always preserved in the early stages.

Individuals with language-variant major or mild frontotemporal NCD present with primary progressive aphasia with gradual onset, with two subtypes commonly described: semantic variant and agrammatic/nonfluent variant; each variant has distinctive features and corresponding neuropathology. A third form of progressive language decline, called logopenic progressive aphasia, is associated with left temporoparietal dysfunction and is often caused by Alzheimer’s disease pathology.

“Probable” is distinguished from “possible” frontotemporal NCD by the presence of causative genetic factors (e.g., mutations in the gene coding for microtubule-associated protein tau) or by the presence of distinctive atrophy or reduced activity in frontotemporal regions on structural or functional imaging.

Associated Features

Extrapyramidal features may be prominent in some cases, with an overlap with syndromes such as progressive supranuclear palsy and corticobasal degeneration. Features of motor neuron disease may be present in some cases (e.g., muscle atrophy, weakness). A subset of individuals develop visual hallucinations.

Prevalence

Major or mild frontotemporal NCD is a common cause of early-onset NCD in individuals younger than 65 years. In international studies, population prevalence estimates are in the range of 2–31 per 100,000, with overall rates generally noted to be equal in men and women, although variation exists among studies. Approximately 20%–25% of cases of frontotemporal NCD occur in individuals older than 65 years. Frontotemporal NCD accounts for about 5% of all cases of dementia in unselected autopsy series. The behavioral variant is the most common presentation of NCD due to frontotemporal degeneration, occurring in approximately 60% of cases.

Development and Course

Individuals with major or mild frontotemporal NCD commonly present in their 50s, although the age at onset varies from the 20s through the 80s. The disease is gradually progressive, with median survival being 6–11 years after symptom onset and 3–4 years after diagnosis. Survival is shorter and decline is faster in major or mild frontotemporal NCD than in typical Alzheimer's disease.

Risk and Prognostic Factors

Genetic and physiological. Approximately 40% of individuals with major or mild frontotemporal NCD have a family history of early-onset NCD, and approximately 10% show an autosomal dominant inheritance pattern. A number of genetic factors have been identified, such as mutations in the gene encoding the microtubule-associated protein tau (*MAPT*), the granulin gene (*GRN*), and the C9ORF72 gene (*C9orf72*). A number of families with causative mutations have been identified (see the section "Diagnostic Markers" for this disorder), but many individuals with known familial

transmission do not have a known mutation. The presence of motor neuron disease is associated with a more rapid deterioration.

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Diagnostic Markers

Computed tomography (CT) or structural magnetic resonance imaging (MRI) may show distinct patterns of atrophy. In behavioral-variant major or mild frontotemporal NCD, both frontal lobes (especially the medial frontal lobes) and the anterior temporal lobes are atrophic. In semantic language–variant major or mild frontotemporal NCD, the middle, inferior, and anterior temporal lobes are atrophic bilaterally but asymmetrically, with the left side usually being more affected. Nonfluent language–variant major or mild frontotemporal NCD is associated with predominantly left posterior frontal-insular atrophy. Functional imaging demonstrates hypoperfusion and/or cortical hypometabolism in the corresponding brain regions, which may be present in the early stages in the absence of structural abnormality. Emerging biomarkers for Alzheimer's disease (e.g., cerebrospinal fluid amyloid-beta and tau levels, and amyloid imaging) may help in the differential diagnosis, but the distinction from Alzheimer's disease can remain difficult.

In familial cases of frontotemporal NCD, the identification of genetic mutations may help confirm the diagnosis. Mutations associated with frontotemporal NCD include the genes encoding microtubule-associated protein tau (MAPT) and granulin (GRN), C9ORF72, transactive response DNA-binding protein of 43 kDa (TDP-43, or TARDBP), valosin-containing protein (VCP), chromatin modifying protein 2B (CHMP2B), and fused in sarcoma protein (FUS).

Functional Consequences of Major or Mild Frontotemporal Neurocognitive Disorder

Because of the relative early age at onset of the disorder, the disorder often affects workplace and family life. Because of the involvement of language and/or behavior, function is often more severely impaired relatively early in the course. For individuals with the behavioral variant, prior to diagnostic

clarification there may be significant family disruption, legal involvement, and problems in the workplace because of socially inappropriate behaviors. The functional impairment attributable to behavioral change and language dysfunction, which can include hyperorality, impulsive wandering, and other disinhibited behaviors, may far exceed that attributable to the cognitive disturbance and may lead to nursing home placement or institutionalization. These behaviors can be severely disruptive, even in structured care settings, particularly when the individuals are otherwise healthy, nonfrail, and free of other medical comorbidities.

Differential Diagnosis

Other neurocognitive disorders. Other neurodegenerative diseases may be distinguished from major or mild frontotemporal NCD by their characteristic features. In major or mild NCD due to Alzheimer's disease, decline in learning and memory is an early feature. However, 10%–30% of individuals presenting with a syndrome suggestive of major or mild frontotemporal NCD are found at autopsy to have Alzheimer's disease pathology. This occurs more frequently in individuals who present with progressive dysexecutive syndromes in the absence of behavioral changes or movement disorder or in those with the logopenic variant.

In major or mild NCD with Lewy bodies, core and suggestive features of Lewy bodies must be present. In major or mild NCD due to Parkinson's disease, spontaneous parkinsonism emerges well before the cognitive decline. In major or mild vascular NCD, depending on affected brain regions, there may also be loss of executive function and behavioral changes such as apathy, and this disorder should be considered in the differential diagnosis. However, history of a cerebrovascular event is temporally related to the onset of cognitive impairment in major or mild vascular NCD, and neuroimaging reveals infarctions or white matter lesions sufficient to account for the clinical picture.

Other neurological conditions. Major or mild frontotemporal NCD overlaps with progressive supranuclear palsy, corticobasal degeneration, and motor

neuron disease clinically as well as pathologically. Progressive supranuclear palsy is characterized by supranuclear gaze palsies and axial-predominant parkinsonism. Pseudobulbar signs may be present, and retropulsion (losing balance in a backward direction) is often prominent. Neurocognitive assessment shows psychomotor slowing, poor working memory, and executive dysfunction. Corticobasal degeneration presents with asymmetric rigidity, limb apraxia, postural instability, myoclonus, alien limb phenomenon, and cortical sensory loss. Many individuals with behavioral-variant major or mild frontotemporal NCD show features of motor neuron disease, which tend to be mixed upper and predominantly lower motor neuron disease.

Other mental disorders and medical conditions. Behavioral-variant major or mild frontotemporal NCD may be mistaken for a primary mental disorder, such as major depression, bipolar disorders, or schizophrenia, and individuals with this variant often present initially to psychiatry. Over time, the development of progressive neurocognitive difficulties will help to make the distinction. A careful medical evaluation will help to exclude treatable causes of NCDs, such as metabolic disturbances, nutritional deficiencies, and infections. If the symptoms characteristic of a primary mental disorder (e.g., delusions) are judged to be due to the physiological effects of frontotemporal degeneration, a diagnosis of the appropriate mental disorder due to frontotemporal degeneration should be given instead of the primary psychotic disorder (e.g., psychotic disorder due to frontotemporal degeneration, with delusions).

Major or Mild Neurocognitive Disorder With Lewy Bodies

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. The disorder has an insidious onset and gradual progression.

C. The disorder meets a combination of core diagnostic features and suggestive diagnostic features for either probable or possible neurocognitive disorder with Lewy bodies.

For probable major or mild neurocognitive disorder with Lewy bodies, the individual has two core features, or one suggestive feature with one or more core features. **For possible major or mild neurocognitive disorder with Lewy bodies,** the individual has only one core feature, or one or more suggestive features.

1. Core diagnostic features:
    a. Fluctuating cognition with pronounced variations in attention and alertness.
    b. Recurrent visual hallucinations that are well formed and detailed.
    c. Spontaneous features of parkinsonism, with onset subsequent to the development of cognitive decline.
2. Suggestive diagnostic features:
    a. Meets criteria for rapid eye movement sleep behavior disorder.
    b. Severe neuroleptic sensitivity.

D. The disturbance is not better explained by cerebrovascular disease, another neurodegenerative disease, the effects of a substance, or another mental, neurological, or systemic disorder.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder with probable or possible Lewy bodies, with behavioral disturbance, code first **G31.83** Lewy body disease, followed by **F02.81.**

For major neurocognitive disorder with probable or possible Lewy bodies, without behavioral disturbance, code first **G31.83** Lewy

body disease, followed by **F02.80.** *Note:* The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild neurocognitive disorder with Lewy bodies, code **G31.84.** (**Note:** Do *not* use the additional code for Lewy body disease. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild neurocognitive disorder with Lewy bodies: Use additional code(s) to indicate clinically significant psychiatric symptoms due to Lewy body disease (e.g., **F06.0** psychotic disorder due to Lewy body disease, with hallucinations; **F06.31** depressive disorder due to Lewy body disease, with depressive features).

Diagnostic Features

Major neurocognitive disorder with Lewy bodies corresponds to the condition known as dementia with Lewy bodies (DLB). The overall major or mild neurocognitive disorder with Lewy bodies (NCDLB) category includes not only progressive cognitive impairment (with early changes in attention, executive function, and visuoperceptual ability, rather than learning and memory) but also recurrent, complex, visual hallucinations; and concurrent symptoms of rapid eye movement (REM) sleep behavior disorder (which can be a very early manifestation); as well as hallucinations in other sensory modalities, apathy, anxiety, depression, and delusions. The cognitive symptoms may fluctuate in a pattern that can resemble a delirium, for which an adequate precipitating factor may or may not be found. The variable presentation of NCDLB symptoms reduces the likelihood of all symptoms being observed in a brief clinic visit and necessitates a thorough assessment, including caregiver observations. The use of assessment scales specifically designed to assess fluctuation may aid in diagnosis. Another core feature is spontaneous parkinsonism; this can often be relatively mild, and the degree of response to levodopa therapy is variable. Up to 25% of individuals with probable NCDLB may never develop extrapyramidal signs, and they are not essential for diagnosis. The parkinsonism must be

distinguished from neuroleptic-induced extrapyramidal signs. Accurate diagnosis is essential to safe treatment planning, as up to 50% of individuals with NCDLB have severe sensitivity to neuroleptic drugs, and these medications should be used with extreme caution in individuals suspected of having an NCDLB diagnosis.

The diagnosis of mild NCDLB is appropriate for individuals who present with the core clinical features at a stage when cognitive or functional impairments are not of sufficient severity to fulfill criteria for major NCD, particularly if nonamnestic cognitive deficits are prominent. However, as for all mild NCDs, there will often be insufficient evidence to justify any single etiology, and use of the unspecified diagnosis may be more appropriate.

Associated Features

Individuals with NCDLB frequently experience repeated falls, syncope, or other transient episodes of unresponsiveness. Autonomic dysfunction may be observed, including orthostatic hypotension, constipation, and urinary incontinence; hypersomnia and hyposmia may also be observed.

Prevalence

Limited data from several high-income and low- and middle-income countries show that the population-based prevalence estimates for NCDLB range from 0% to 1.2% of the general elderly population, and from 0% to 9.7% of all dementia cases. The mean prevalence of major NCDLB was 4.2% of all dementias in the community, and in clinic-based studies this increased to 7.5% of all dementias. The clinical prevalence of major NCDLB among

individuals with dementia does not appear to be significantly affected by either age or sex. In studies from the United States and United Kingdom, the pathological lesions known as Lewy bodies are present in 20%–35% of cases of dementia. In a population-based study in Minnesota that relied on

medical records, the incidence of NCDLB was approximately three times higher in men than in women age 65 or older.

Development and Course

NCDLB is a gradually progressive disorder with insidious onset. However, there is often a prodromal history of confusional episodes (delirium) of acute onset, which may be precipitated by illness or surgery. The distinction between NCDLB, in which Lewy bodies are primarily limbic in location (with or without neocortical involvement), and major or mild NCD due to Parkinson's disease, which starts in the brain stem, is the order in which the cognitive and motor symptoms emerge. In NCDLB, the cognitive decline is manifested early in the course of illness (see the section "Differential Diagnosis" for this disorder).

Onset of symptoms is typically observed in individuals ages 50–89, with most cases having onset in individuals in the mid-70s. Disease course may be characterized by occasional plateaus but eventually progresses through severe dementia to death. Average duration of survival is 5.5–7.7 years from the onset of cognitive decline.

Risk and Prognostic Factors

Genetic and physiological. Familial aggregation may occur, and several risk genes have been identified; but in most cases of NCDLB, there is no family history. The available studies suggest that genetic risk factors are as important in NCDLB as in Alzheimer's disease or Parkinson's disease.

Diagnostic Markers

Biomarkers indicative of NCDLB may be considered to carry diagnostic weight equivalent to core clinical features; these include low striatal dopamine transporter uptake on single photon emission computed tomography (SPECT) or positron emission tomography (PET) scan, abnormal (low uptake) (MIBG) myocardial scintigraphy suggesting cardiac sympathetic denervation, and polysomnographic confirmation of REM sleep without atonia. The associated condition REM sleep behavior disorder may be diagnosed through a formal sleep study or identified by questioning

the individual or informant about relevant symptoms. The underlying neurodegenerative disease is primarily associated with misfolding and aggregation of α-synuclein, which may be confirmed via postmortem histopathological examination. Neuropsychological testing beyond the use of a brief screening instrument may be necessary to define cognitive deficits clearly. Assessment scales developed to measure fluctuation can be useful.

Biomarkers supportive of NCDLB but with more limited evidence of diagnostic value include the following: preservation of medial temporal volume relative to Alzheimer's disease on magnetic resonance imaging (MRI), generalized low uptake on SPECT/PET perfusion scan with reduced occipital activity with or without the cingulate island sign (sparing of the posterior cingulate cortex relative to the precuneus plus cuneus on fluorodeoxyglucose-PET imaging), and prominent slow-wave activity on electroencephalogram with periodic fluctuations in the pre-alpha/theta range.

Functional Consequences of Major or Mild Neurocognitive Disorder With Lewy Bodies

Individuals with NCDLB are more functionally impaired than would be expected for their cognitive deficits when contrasted to individuals with other neurodegenerative diseases,

such as Alzheimer's disease. This is largely a result of motor and autonomic impairments, which cause problems with toileting, transferring, and eating. Sleep disorders and prominent psychiatric symptoms may also add to functional difficulties. Consequently, the quality of life of individuals with NCDLB is often significantly worse than that of individuals with Alzheimer's disease.

Differential Diagnosis

Major or mild neurocognitive disorder due to Parkinson's disease. The distinction between NCDLB and NCD due to Parkinson's disease is based on the timing and sequence of motor symptoms and cognitive symptoms.

Consensus criteria for DLB separate NCDLB from NCD due to Parkinson's disease by specifying that for dementia to be attributed to Parkinson's disease, the Parkinson's disease diagnosis is present for at least 1 year before cognitive decline has reached the level of major NCD, whereas for NCDLB, the cognitive symptoms may begin before, with, or in the absence of parkinsonism. By contrast, expert consensus criteria for Parkinson's disease propose that if cognitive decline occurs prior to a motor diagnosis, the diagnosis of Parkinson's disease may still be made; therefore, a clinician may attribute the cognitive decline to the Parkinson's disease and diagnose NCD due to Parkinson's disease. Consequently, the clinician may choose to diagnose NCD due to Parkinson's disease or NCDLB for individuals with major NCD that starts either before or within 12 months of Parkinson's disease. In such circumstances, the clinician decides which diagnosis is more appropriate. If Parkinson's disease has been diagnosed for at least 1 year prior to the onset of cognitive symptoms, then both expert criteria agree that NCD due to Parkinson's disease would typically be the appropriate diagnosis. The timing and sequence of parkinsonism and mild NCD may be particularly difficult to determine, and unspecified NCD may need to be diagnosed until the order of clinical progression becomes evident.

Comorbidity

Lewy body pathology frequently coexists with Alzheimer's disease, transactive response DNA-binding protein 43 (TDP-43)–related pathology, and cerebrovascular disease pathology, particularly in the oldest age groups. TDP-43 is a protein that has been identified as a source of the proteinopathies across a range of neurodegenerative disorders, including amyotrophic lateral sclerosis and frontotemporal degeneration. The presence of multiple pathological lesions has implications for disease prognosis and may be associated with a more rapid cognitive decline and shorter survival time.

Major or Mild Vascular Neurocognitive Disorder

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. The clinical features are consistent with a vascular etiology, as suggested by either of the following:
    1. Onset of the cognitive deficits is temporally related to one or more cerebrovascular events.
    2. Evidence for decline is prominent in complex attention (including processing speed) and frontal-executive function.

C. There is evidence of the presence of cerebrovascular disease from history, physical examination, and/or neuroimaging considered sufficient to account for the neurocognitive deficits.
D. The symptoms are not better explained by another brain disease or systemic disorder.

Probable vascular neurocognitive disorder is diagnosed if one of the following is present; otherwise **possible vascular neurocognitive disorder** should be diagnosed:

1. Clinical criteria are supported by neuroimaging evidence of significant parenchymal injury attributed to cerebrovascular disease (neuroimaging-supported).
2. The neurocognitive syndrome is temporally related to one or more documented cerebrovascular events.
3. Both clinical and genetic (e.g., cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) evidence of cerebrovascular disease is present.

Possible vascular neurocognitive disorder is diagnosed if the clinical criteria are met but neuroimaging is not available and the

temporal relationship of the neurocognitive syndrome with one or more cerebrovascular events is not established.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder probably or possibly due to vascular disease, with behavioral disturbance, code **F01.51.**

For major neurocognitive disorder probably or possibly due to vascular disease, without behavioral disturbance, code **F01.50.**

An additional medical code for the vascular disease is not used.

Note: The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild vascular neurocognitive disorder, code **G31.84.** (**Note:** Do *not* use an additional code for the vascular disease. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild vascular neurocognitive disorder: Use additional code(s) to indicate clinically significant psychiatric symptoms due to the cerebrovascular disease (e.g., **F06.31** depressive disorder due to cerebrovascular disease, with depressive features).

Diagnostic Features

The diagnosis of major or mild vascular neurocognitive disorder (NCD) requires the establishment of an NCD (Criterion A) and the determination that cerebrovascular disease is the dominant if not exclusive pathology that accounts for the cognitive deficits (Criteria B and C). Vascular etiology may range from large vessel stroke to microvascular disease; the presentation is therefore very heterogeneous, stemming from the types of vascular lesions and their extent and location. The lesions may be focal, multifocal, or diffuse and occur in various combinations. Pathogenic mechanisms responsible for brain parenchymal injury include hypoperfusion and hypoxia, oxidative stress and inflammation leading to endothelial dysfunction, impairment of autoregulation, and disruption of neurovascular coupling.

Many individuals with major or mild vascular NCD present with multiple infarctions, with an acute stepwise or fluctuating decline in cognition, and intervening periods of stability and even some improvement. Others may have gradual onset with slow progression, a rapid development of deficits followed by relative stability, or another complex presentation. Major or mild vascular NCD with a gradual onset and slow progression is generally attributable to small vessel disease leading to lesions in the white matter, basal ganglia, or thalamus. The gradual progression in these cases is often punctuated by acute

events that leave subtle neurological deficits. The cognitive deficits in these cases can be attributed to disruption of cortical-subcortical circuits; complex attention, particularly speed of information processing, and executive function are likely to be affected. Clinical subtypes of vascular NCD have been described and include 1) poststroke NCD, manifesting immediately after a stroke; 2) subcortical ischemic vascular NCD; 3) multi-infarct (cortical) NCD; and 4) cortical-subcortical vascular NCD.

Assessing for the presence of sufficient cerebrovascular disease relies on history, physical examination, and neuroimaging (Criterion C). Etiological certainty requires the demonstration of abnormalities on neuroimaging. The lack of neuroimaging can result in significant diagnostic inaccuracy by overlooking “silent” brain infarction and white matter lesions. However, if the neurocognitive impairment is temporally associated with one or more well-documented strokes, a probable diagnosis can be made in the absence of neuroimaging. Clinical evidence of cerebrovascular disease includes documented history of stroke, with cognitive decline temporally associated with the event, or physical signs consistent with stroke (e.g., hemiparesis; pseudobulbar syndrome, visual field defect). Neuroimaging (magnetic resonance imaging [MRI] or computed tomography [CT]) evidence of cerebrovascular disease comprises one or more of the following: one or more large vessel infarcts or hemorrhages, a strategically placed single infarct or hemorrhage (e.g., in angular gyrus, thalamus, basal forebrain), two or more lacunes outside the brain stem, or extensive and confluent white matter lesions. The latter is often termed

small vessel disease or *subcortical ischemic changes* on clinical neuroimaging evaluations. MRI is the preferred mode of neuroimaging, and there has been interest in using specialized MRI techniques to detect cerebral microbleeds, cortical microinfarcts, dilated perivascular spaces, and diffusion-based analyses of white matter tracts and network connectivity.

For mild vascular NCD, history of a single stroke or extensive white matter disease is generally sufficient. For major vascular NCD, two or more strokes, a strategically placed stroke, or a combination of white matter disease and one or more lacunes is generally necessary. However, the relationship between identifiable vascular pathology in the brain on neuroimaging and the cognitive symptoms is imperfect, and clinical judgment is generally needed to relate the vascular lesions to the cognitive syndrome.

The neurocognitive symptoms must not be better explained by another medical condition or mental disorder. For example, prominent memory deficit early in the course might suggest NCD due to Alzheimer's disease, early and prominent parkinsonian features would suggest NCD due to Parkinson's disease, and a close association between onset of cognitive and depressive symptoms would suggest cognitive impairment as a result of depression.

A number of expert international groups have similarly defined and categorized the vascular NCDs, with which DSM-5 criteria generally display good correspondence.

Associated Features

A neurological assessment often reveals history of stroke or transient ischemic episodes, and signs indicative of brain infarctions. Also commonly associated are personality and mood changes, abulia, depression, and emotional lability. The development of late-onset depressive symptoms accompanied by psychomotor slowing and executive dysfunction is a common presentation among older adults with progressive small vessel ischemic disease (so-called vascular depression).

Prevalence

Vascular disease is the second most common cause of NCD after Alzheimer's disease. In the United States, population prevalence estimates for vascular dementia are 0.98% for individuals ages 71–79 years, 4.09% for those ages 80–89 years, and 6.19% for those age 90 years or older. Within 3 months following stroke, 20%–30% of individuals are diagnosed with

dementia. In a European autopsy series of decedents ages 60–103 years, the prevalence of pure vascular dementia was 12.3%. Among those ages 60–69 years, the prevalence was higher (15.0%) compared with those older than 90 years (8.7%). Mixed dementia (Alzheimer's plus vascular pathology) was present in 5.5% of the overall cohort, with a higher prevalence in those older than 90 years (10.6%) compared with those ages 60–69 years (5.2%). Higher prevalence of vascular dementia has been found among African Americans, Mexican Americans, and South Asian Americans compared with non-Latinx Whites, possibly because of higher rates of risk factors such as diabetes and cardiovascular disease. In Japan and several other Asian countries, the prevalence of dementia due to Alzheimer's disease has increased over time relative to vascular dementia. Currently, the prevalence of dementia due to Alzheimer's disease among Japanese Americans is 2.6 times higher than that of vascular dementia.

Stroke is more common in men through age 65 years, but more common in women after age 65 years. Overall, the rate of vascular NCD was higher in men in some studies.

Development and Course

Major or mild vascular NCD can occur at any age, although the prevalence increases exponentially after age 65 years. In older individuals, additional pathologies are almost always present and partly account for the neurocognitive deficits. The course may vary from acute onset with partial improvement to stepwise decline to progressive decline, with fluctuations and plateaus of varying durations. Pure subcortical major or mild vascular NCD can have a slowly progressive course that simulates major or mild

NCD due to Alzheimer's disease. The risk of an ischemic stroke progressing to vascular NCD within 5 years was almost twice as high among African Americans as among non-Latinx Whites in the United States and occurred at younger ages. This is possibly a result of the impact of higher rates of hypertension, diabetes, and adverse social determinants of mental health known to worsen dementia risk, such as limited formal education and low socioeconomic status.

Risk and Prognostic Factors

Environmental. The neurocognitive outcomes of vascular brain injury are influenced by neuroplasticity factors such as education, physical exercise, and mental activity.

Genetic and physiological. The major risk factors for major or mild vascular NCD are the same as those for cerebrovascular disease and stroke, including hypertension, diabetes, smoking, obesity, high cholesterol levels, high homocysteine levels, other risk factors for atherosclerosis and arteriolosclerosis, atrial fibrillation, and other conditions increasing the risk of cerebral emboli. Cerebral amyloid angiopathy, leading to cerebral hemorrhage, is an important risk factor in which amyloid deposits occur within arterial vessels. A genetic risk factor is the hereditary condition cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy, or CADASIL. Other rarer forms of genetic disorders linked to vascular NCD exist, but overall the contribution of genetics is small.

Diagnostic Markers

Structural neuroimaging, using MRI or CT, has an important role in the diagnostic process. There are no other established biomarkers of major or mild vascular NCD.

Functional Consequences of Major or Mild Vascular Neurocognitive Disorder

Major or mild vascular NCD is commonly associated with physical deficits that cause additional disability.

Differential Diagnosis

Other neurocognitive disorders. Since incidental brain infarctions and white matter lesions are common in older individuals, it is important to consider other possible etiologies when an NCD is present in an individual with white matter lesions. A history of memory deficit early in the course, and progressive worsening of memory, language, executive function, and perceptual-motor abilities in the absence of corresponding focal lesions on brain imaging, are suggestive of Alzheimer's disease as the primary diagnosis. Potential biomarkers currently being validated for Alzheimer's disease, such as cerebrospinal fluid levels of β-amyloid and phosphorylated tau, and amyloid and tau imaging, may prove to be helpful in the differential diagnosis. NCD with Lewy bodies is distinguished from major or mild vascular NCD by its core features of fluctuating cognition, visual hallucinations, and spontaneous parkinsonism. While deficits in executive function and language occur in major or mild vascular NCD, the insidious onset and gradual progression of behavioral features or language impairment are characteristic of frontotemporal NCD and are not typical of vascular etiology.

Other medical conditions. A diagnosis of major or mild vascular NCD is not made if other diseases (e.g., brain tumor, multiple sclerosis, encephalitis, toxic or metabolic disorders) are present and are of sufficient severity to account for the cognitive impairment.

Other mental disorders. A diagnosis of major or mild vascular NCD is inappropriate if the symptoms can be entirely attributed to delirium, although delirium may sometimes be superimposed on a preexisting major or mild vascular NCD, in which case both diagnoses can be made. If the criteria for major depressive disorder are met and the cognitive impairment is temporally related to the likely onset of the depression, major or mild vascular NCD should not be diagnosed. However, if the NCD preceded the development of the depression, or the severity of the cognitive impairment is out of proportion to the severity of the depression, depressive disorder due to cerebrovascular disease should be diagnosed instead of major depressive disorder.

Comorbidity

Major or mild NCD due to Alzheimer's disease commonly co-occurs with major or mild vascular NCD, in which case both diagnoses should be made. Major or mild vascular NCD and depression frequently co-occur.

Major or Mild Neurocognitive Disorder Due to Traumatic Brain Injury

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.

B. There is evidence of a traumatic brain injury—that is, an impact to the head or other mechanisms of rapid movement or displacement of the brain within the skull, with one or more of the following:

   1. Loss of consciousness.
   2. Posttraumatic amnesia.
   3. Disorientation and confusion.
   4. Neurological signs (e.g., neuroimaging demonstrating injury; visual field cuts; anosmia; hemiparesis; hemisensory loss; cortical blindness; aphasia; apraxia; weakness;

      loss of balance; other sensory loss that cannot be accounted for by peripheral or other causes).

C. The neurocognitive disorder presents immediately after the occurrence of the traumatic brain injury or immediately after recovery of consciousness and persists past the acute post-injury period.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder due to traumatic brain injury, with behavioral disturbance: code first **S06.2X9S** diffuse traumatic brain injury with loss of consciousness of unspecified duration, sequela; followed by **F02.81** major neurocognitive disorder due to traumatic brain injury, with behavioral disturbance.

For major neurocognitive disorder due to traumatic brain injury, without behavioral disturbance: code first **S06.2X9S** diffuse traumatic brain injury with loss of consciousness of unspecified duration, sequela; followed by **F02.80** major neurocognitive disorder due to traumatic brain injury, without behavioral disturbance.

Note: The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild neurocognitive disorder due to traumatic brain injury, code **G31.84.** (**Note:** Do *not* use the additional code for traumatic brain injury. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild neurocognitive disorder due to traumatic brain injury: Use additional code(s) to indicate clinically significant psychiatric symptoms due to the traumatic brain injury (e.g., **F06.34** bipolar and related disorder due to traumatic brain injury, with mixed features; **F07.0** personality change due to traumatic brain injury, apathetic type).

Specifiers

Rate the severity of the neurocognitive disorder (NCD), not the underlying traumatic brain injury (see the section "Development and Course" for this disorder).

Diagnostic Features

Major or mild NCD due to traumatic brain injury (TBI) denotes an acquired and persistent disorder of cognition resulting from a traumatic brain injury.

Traumatic brain injury is defined as disruption of brain structure and/or function resulting from the application of biomechanical forces (including acceleration/deceleration forces and blast-related forces), as manifested immediately by one or more of the following clinical signs: loss of consciousness, loss of memory for events immediately before or after the injury (posttraumatic amnesia), alteration in mental state (e.g., confusion, disorientation, slowed thinking), or focal neurological signs (e.g., hemiparesis, hemisensory loss, cortical blindness, aphasia, apraxia, weakness, loss of balance, other sensory loss that cannot be accounted for by peripheral or other causes) (Criterion B). These manifestations of TBI must not be due to alcohol or other drugs or medications, other injuries or treatment(s) for other injuries (e.g., facial injuries, intubation, or bodily/systemic injuries), or psychological trauma, language barrier, or coexisting medical conditions.

The severity of a TBI is classified as mild, complicated mild, moderate, or severe according to the thresholds in Table 2. An individual whose injury phenomenologically meets criteria for mild TBI but whose computed tomographic or magnetic resonance imaging in the acute period after TBI reveals traumatic intracranial abnormalities (i.e., traumatic epidural or subdural hematoma, subarachnoid or intracerebral hemorrhage, cerebral contusions or laceration) is classified as complicated mild TBI. Outcomes of individuals with complicated mild TBI are more like those with moderate TBI than those with uncomplicated mild TBI.

TABLE 2 Classification of traumatic brain injury (TBI) severity

TBI severity	Mild TBI	Complicated mild TBI	Moderate TBI	Severe TBI
Loss of consciousness duration	≤ 30 minutes	≤ 30 minutes	> 30 minutes to < 24 hours	≥ 24 hours
Posttraumatic amnesia duration (densely impaired new learning)	≤ 1 day	≤ 1 day	> 1 day to < 7 days	≥ 7 days
Alteration of consciousness duration (e.g., confusion, disorientation, slowed thinking)	≤ 1 day	≤ 1 day	> 1 day to < 7 days	≥ 7 days

TBI severity	Mild TBI	Complicated mild TBI	Moderate TBI	Severe TBI
Glasgow Coma Scale score (30 minutes after the event)	13–15	13–15	9–12	3–8
Computed tomography or magnetic resonance imaging of the brain	Normal	Abnormal	Normal or abnormal	Normal or abnormal

To be attributable to TBI, the NCD must manifest either immediately after the brain injury occurs or immediately after the individual recovers consciousness after the injury, and persist past the acute postinjury period (Criterion C).

While the specific cognitive impairments associated with major or mild NCD due to TBI are variable, impairments in complex attention, processing speed, learning and memory, and executive function are common, as are disturbances in social cognition. In more severe TBI in which there is brain contusion, intracranial hemorrhage, or penetrating injury, there may be additional neurocognitive impairments associated with the affected region of the brain and the volume of brain tissue lost (e.g., aphasia, apraxia, disturbances in perceptual-motor function).

Associated Features

The diagnosis may also be supported by subtle neurological signs (e.g., multiple primitive reflexes such as glabellar sign, snout response, palmomental reflex) or deficits in saccades and smooth-pursuit eye movements co-occurring with frontally mediated cognitive impairments such as complex attention problems, slow processing speed, impaired memory retrieval, or executive dysfunction. Particularly in some cases of penetrating TBI, the diagnosis of NCD due to TBI may be supported by posttraumatic epilepsy with focal onset in a location that corresponds to the anatomy of a cognitive domain in which an individual demonstrates impairment (e.g., medial temporal lobe–onset seizures and episodic memory impairment; frontal lobe seizures and executive dysfunction or social cognitive impairment).

Prevalence

The prevalence of major and mild NCD due to TBI varies with injury severity and time since injury, with the highest frequencies among individuals with more severe injury and during the acute/subacute post-injury period. In the United States, more than 2.87 million TBIs occur annually, including more than 837,000 TBIs in children. These TBIs account for 2.5 million emergency department visits, 288,000 hospitalizations, and more than 56,000

deaths annually. Among individuals presenting to an emergency department with TBI, the rates for men are 547.6 per 100,000 and for women are 385.9 per 100,000. The TBI rate is higher for men than women in every age group up to age 75 years, after which the rates of TBI between men and women approach parity. The leading causes of TBI in the United States are falls (178.4 per 100,000), collision with a moving or stationary object (termed “struck by/against” events) (92.7 per 100,000), motor vehicle crashes (74.7 per 100,000), and assaults (50.6 per 100,000 persons). Concussion in sport is increasingly recognized as a cause of mild TBI.

Men are approximately 40% more likely to experience a TBI compared with women in the young and adult populations; however, women may have higher risk of TBI after age 65 years. It has been suggested that men with moderate or severe TBI may have a worse prognosis than women with the same level of severity; however, the findings have been mixed. The cause of TBI also differs by sex and gender. Men are more likely to experience injuries at work, in motor vehicle accidents, and during military activities, whereas women are more likely to experience injuries from assault and domestic violence.

Development and Course

The course of recovery from TBI is variable, depending not only on the specifics of the injury but also on pre-injury and postinjury factors. These factors may favor or impede recovery and include age; prior history of TBI; neurological, psychiatric, and substance use comorbidities and

complications; genetics; the timeliness and effectiveness of medical and rehabilitative interventions; and psychosocial support, among others.

Neurocognitive impairments are most severe in the acute period following the TBI and may be accompanied by disturbances of emotion and behavior. Across the spectrum of TBI severity, substantial improvement in neurocognitive and associated psychiatric and neurological symptoms and signs is expected. The extent of recovery and the variability in neurocognitive outcomes tend to reflect the severity of TBI, with complete recovery being typical after mild TBI and more variable, and often incomplete, recovery following more severe TBI.

Neurocognitive impairments associated with mild TBI typically resolve within days to weeks after the injury, with complete resolution within 3–12 months post-injury. Other symptoms (e.g., depression, irritability, fatigue, headache, photosensitivity, sleep disturbance) that may potentially co-occur with the neurocognitive symptoms also tend to resolve in the weeks following mild TBI. Persistent symptoms after mild TBI or subsequent neurocognitive deterioration should trigger consideration of other potential causes of neurocognitive symptoms and functional limitations, including major depressive disorder, posttraumatic stress disorder (PTSD), anxiety disorders, substance use disorders, sleep disturbances, negative injury perceptions, and poor expectations for recovery. When neurocognitive symptoms and functional limitations persist after mild TBI (including repetitive mild TBI) despite treatment of their other potential causes, diagnosis of an NCD due to TBI may be appropriate.

Neurocognitive impairments and associated functional limitations produced by moderate and severe TBI typically improve over weeks to months after the injury, although long-term neurocognitive recovery is often incomplete among individuals with more severe injuries. Nonetheless, neurocognitive and functional improvement may continue for years after moderate or severe TBI, with more individuals cognitively improving than declining during the first 5 years postinjury. With moderate and severe TBI, in addition to persistence of neurocognitive deficits, there may be associated neurological, medical, emotional, and behavioral complications. These include seizures (particularly in the first year), photosensitivity, hyperacusis, irritability, aggression, depression, sleep disturbance, fatigue, apathy, inability to resume occupational and social functioning at preinjury

level, and deterioration in interpersonal relationships. Moderate and severe TBI have been

associated with increased risk of depression, aggression, and possibly neurodegenerative diseases such as Alzheimer's disease, Lewy body disease, and frontotemporal degeneration.

The features of persisting major or mild NCD due to TBI will vary by age, specifics of the injury, and cofactors. Persisting TBI-related impairment in an infant or child may be reflected in delays in reaching developmental milestones (e.g., language acquisition), worse academic performance, and possibly impaired social development. Among older teenagers and adults, persisting symptoms may include various neurocognitive deficits, irritability, hypersensitivity to light and sound, easy fatigability, and mood changes, including depression, anxiety, hostility, or apathy. In older individuals, mild TBI may produce neurocognitive outcomes like those associated with moderate or severe TBI in younger adults.

Risk and Prognostic Factors

Risk factors for adverse cognitive outcomes after TBI include age older than 40 years, lower pre-injury cognitive abilities (especially as indexed by education or academic competence), preinjury depressive symptoms, possibly pre-injury unemployment, and injury severity. Other risk factors for adverse cognitive outcomes include a longer duration of posttraumatic amnesia, evidence of traumatic intracranial abnormalities on early computed tomography or magnetic resonance imaging (MRI) studies (i.e., traumatic epidural or subdural hematoma, subarachnoid or intracerebral hemorrhage, cerebral contusions or laceration, diffuse axonal injury) and neurogenetic profile (e.g., *APOE*E4* allele carrier status, catechol-O-methyltransferase genotype, *ANKK1* Taq1A allele status). Pre-injury alcohol or substance use disorders increase the risk of sustaining a TBI as well as the risk of adverse cognitive outcomes, including memory impairment and executive dysfunction.

Diagnostic Markers

The diagnosis of major or mild NCD due to TBI may be supported by contemporaneous computed tomographic or MRI findings (e.g., focal atrophy, encephalomalacia, gliosis, white matter abormalities) in brain areas or networks subserving specific cognitive domains in which an individual demonstrates impairment. The diagnosis may also be supported by subtle neurological signs (e.g., multiple primitive reflexes such as glabellar sign, snout response, palmomental reflex) or deficits in saccades and smooth-pursuit eye movements co-occurring with frontally mediated cognitive impairments such as complex attention problems, slow processing speed, impaired memory retrieval, or executive dysfunction. Particularly in some cases of penetrating TBI, the diagnosis of NCD due to TBI may be supported by posttraumatic epilepsy with focal onset in a location that corresponds to the anatomy of a cognitive domain in which an individual demonstrates impairment (e.g., medial temporal lobe–onset seizures and episodic memory impairment; frontal lobe seizures and executive dysfunction or social cognitive impairment).

Performance on commonly used general cognitive screening measures, particularly when interpreted using large-scale, population-based normative data, may usefully identify individuals in need of further neurodiagnostic assessment. However, the diagnosis of major or mild NCD due to TBI rests on performance on domain-specific cognitive assessment interpreted in light of the individual's prior performance (e.g., neuropsychological estimates of pre-injury cognitive ability or appropriate norms) and assessment of functional status.

While neuroimaging and other clinical assessments (e.g., subtle neurological signs) may provide supportive information, they cannot independently diagnose NCD due to TBI. At present, there are no other established biomarkers of major or mild NCD due to TBI.

Association With Suicidal Thoughts or Behavior

Individuals with TBI, including moderate or severe TBI, are at increased long-term risk for suicide. While depression is a substantial contributor to this risk, it does not fully account for it. Rates of suicidal ideation are as high as 10%, and rates of suicide attempt are 0.8%–1.7%

over the first 20 years after TBI. The development of depression and/or suicidal behavior at 1-year post-injury is associated with consistently elevated rates of depression and suicidal behavior 5 years after TBI. While the relationship between cognitive impairments and suicide risk after TBI is complex, assessing suicide risk is an important element in the evaluation of individuals with major or mild NCD due to TBI.

Youth who have had concussions may be at higher risk for suicidal behavior. There is an increased risk of suicide among both veteran and civilian cohorts with TBI, and individuals seeking mental health care may have a history of TBI. Individuals seeking rehabilitative services for TBI are also at heightened risk for suicidal thoughts and behavior.

Functional Consequences of Major or Mild Neurocognitive Disorder Due to Traumatic Brain Injury

Approximately 3.17 million individuals in the United States (approximately 1.1% of the population) live with a TBI-related disability, including neurocognitive impairments that compromise the ability to work or perform daily activities and that are associated with the need for ongoing medical care, rehabilitation, support, and services. Cognitive impairments interfere with functional independence, productive employment, and community participation and may reduce satisfaction with life. The influence of cognitive impairments on functional status varies with the type and severity of those impairments; with the presence and severity of co-occurring psychiatric, substance use, neurological, and medical conditions; and with family, other psychosocial, and medical support.

With mild NCD due to TBI, individuals may report reduced cognitive efficiency, difficulty concentrating, and lessened ability to perform usual activities. With major NCD due to TBI, an individual may have difficulty in independent living and self-care. Prominent neuromotor features, such as severe incoordination, ataxia, and motor slowing, may be present in major NCD due to TBI and may add to functional difficulties.

Individuals with TBI histories report more depressive and anxious symptoms, and these can amplify cognitive complaints and worsen

functional outcome. Additionally, loss of emotional control, including aggressive or inappropriate affect and apathy, may be present after more severe TBI with greater neurocognitive impairment. These features may compound difficulties with functional independence and self-care.

Differential Diagnosis

Other mental disorders and medical conditions. Mental disorders (e.g., major depressive disorder, anxiety disorders, PTSD, alcohol and other substance use disorders, sleep disturbances), prescribed medications (e.g., typical antipsychotics, benzodiazepines, drugs with anticholinergic properties, antiepileptic drugs), and other medical conditions may contribute to or account for cognitive impairments among individuals with TBI, and need to be considered in the differential diagnosis of major or mild NCD due to TBI.

Factitious disorder and malingering. Alternative explanations for neurocognitive symptoms should be considered when the severity of neurocognitive symptoms and functional limitations are inconsistent with the cognitive outcomes expected after TBI—and particularly mild TBI—and when neuropsychological assessment reveals poor effort or is otherwise not valid for interpretation. In such circumstances, the possibility of factitious disorder or malingering (especially in situations in which there might be external incentives such as obtaining financial compensation) should be considered.

Comorbidity

Major or mild NCD due to TBI may be accompanied by other specified or unspecified depressive or anxiety disorders characterized by disturbances in emotional function (e.g., irritability, easy frustration, tension and anxiety, affective lability). Other specified or

unspecified personality disorders may also occur as a result of symptoms such as disinhibition, apathy, suspiciousness, or aggression. Medical comorbidities may occur with neurological and physical disturbances

characterized by headache, fatigue, sleep disorders, vertigo or dizziness, tinnitus or hyperacusis, photosensitivity, anosmia, reduced tolerance to psychotropic medications, and, particularly in more severe TBI, neurological symptoms and signs (e.g., seizures, hemiparesis, visual disturbances, cranial nerve deficits) and evidence of orthopedic injuries. The most common medical and psychiatric comorbidities associated with moderate-to-severe TBI are (in order of frequency) back pain, depression, hypertension, anxiety, fractures, high blood cholesterol, sleep disorders, panic attacks, osteoarthritis, and diabetes.

Among individuals with substance use disorders, the neurocognitive effects of the substance contribute to or compound the TBI-associated cognitive disturbances, particularly among individuals with two or more TBIs.

PTSD can co-occur with TBI in civilian, military, and veterans populations. TBI and PTSD produce similar neurocognitive symptoms (e.g., disturbances of complex attention, processing speed, learning and memory, and executive function), and either or both conditions, as well as co-occurring depression and sleep disturbances, may explain neurocognitive symptoms in individuals with such comorbidities.

Substance/Medication-Induced Major or Mild Neurocognitive Disorder

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. The neurocognitive impairments do not occur exclusively during the course of a delirium and persist beyond the usual duration of intoxication and acute withdrawal.
C. The involved substance or medication and duration and extent of use are capable of producing the neurocognitive impairment.

D. The temporal course of the neurocognitive deficits is consistent with the timing of substance or medication use and abstinence (e.g., the deficits remain stable or improve after a period of abstinence).

E. The neurocognitive disorder is not attributable to another medical condition or is not better explained by another mental disorder.

Coding note (see also coding table on pp. 682–683): The ICD-10-CM codes for the [specific substance/medication]-induced neurocognitive disorders are indicated in the table below. Note that the ICD-10-CM code depends on whether or not there is a comorbid substance use disorder present for the same class of substance. In any case, an additional separate diagnosis of a substance use disorder is not given.

Substance-induced major neurocognitive disorder: If a mild substance use disorder is comorbid with the substance-induced major neurocognitive disorder, the 4th position character is "1," and the clinician should record "mild [substance] use disorder" before the substance-induced major neurocognitive disorder (e.g., "mild inhalant use disorder with inhalant-induced major neurocognitive disorder"). For alcohol and sedative, hypnotic, or anxiolytic substances, a mild substance use disorder is insufficient to cause a substance-induced major neurocognitive disorder; thus, there are no available ICD-10-CM codes for this combination. If a moderate or severe substance use disorder is comorbid with the substance-induced major neurocognitive disorder, the 4th position character is "2," and the clinician should record "moderate [substance] use disorder" or "severe [substance] use disorder," depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder, then the 4th position character is "9," and the clinician should record only the substance-induced major neurocognitive disorder.

Substance-induced mild neurocognitive disorder: If a mild substance use disorder is comorbid with the substance-induced mild neurocognitive disorder, the 4th position character is “1,” and the clinician should record “mild [substance] use disorder” before the substance-induced mild neurocognitive disorder (e.g., “mild cocaine use disorder with cocaine-induced mild neurocognitive disorder”). If a moderate or severe substance use disorder is comorbid with the substance-induced mild neurocognitive disorder, the 4th position character is “2,” and the clinician should record “moderate [substance] use disorder” or “severe [substance] use disorder,” depending on the severity of the comorbid substance use disorder. If there is no comorbid substance use disorder, then the 4th position character is “9,” and the clinician should record only the substance-induced mild neurocognitive disorder.

The severity specifiers “mild,” “moderate,” and “severe” (for major neurocognitive disorder) and the accompanying symptom specifiers “with behavioral disturbance” and “without behavioral disturbance” (for major or mild neurocognitive disorder) cannot be coded but should still be recorded.

	ICD-10-CM		
	With mild use disorder	**With moderate or severe use disorder**	**Without use disorder**
Substance-induced major neurocognitive disorder (NCD)			
Alcohol (major NCD), nonamnestic-confabulatory type	NA	F10.27	F10.97
Alcohol (major NCD), amnestic-confabulatory type	NA	F10.26	F10.96
Inhalant (major NCD)	F18.17	F18.27	F18.97
Sedative, hypnotic, or anxiolytic (major NCD)	NA	F13.27	F13.97
Other (or unknown) substance (major NCD)	F19.17	F19.27	F19.97
Substance-induced mild neurocognitive disorder (NCD)			
Alcohol (mild NCD)	F10.188	F10.288	F10.988

	ICD-10-CM		
Inhalant (mild NCD)	F18.188	F18.288	F18.988
Sedative, hypnotic, or anxiolytic (mild NCD)	F13.188	F13.288	F13.988
Amphetamine-type substance (or other stimulant) (mild NCD)	F15.188	F15.288	F15.988
Cocaine (mild NCD)	F14.188	F14.288	F14.988
Other (or unknown) substance (mild NCD)	F19.188	F19.288	F19.988

Specify if:

Persistent: Neurocognitive impairment continues to be significant after an extended period of abstinence.

Recording Procedures

The name of the substance/medication-induced neurocognitive disorder (NCD) begins with the specific substance (e.g., alcohol) that is presumed to be causing the neurocognitive symptoms. The ICD-10-CM code that corresponds to the applicable drug class is selected from the table included in the criteria set. For substances that do not fit into any of the classes (e.g., intrathecal methotrexate), the ICD-10-CM code for the other (or unknown) substance class should be used and the name of the specific substance recorded (e.g., F19.988 intrathecal methotrexate-induced mild neurocognitive disorder). In cases in which a substance is judged to be an etiological factor but the specific substance is unknown, the ICD-10-CM code for the other (or unknown) substance class is used, and the fact that the substance is unknown is recorded (e.g., F19.97 unknown substance-induced major neurocognitive disorder).

When recording the name of the disorder, the comorbid substance use disorder (if any) is listed first, followed by the word “with,” followed by the name of the disorder (i.e., [specific substance]–induced major neurocognitive disorder or [specific substance]–induced mild neurocognitive disorder), followed by the type in the case of alcohol (i.e., nonamnestic-confabulatory type, amnestic-confabulatory type), followed by

specification of duration (i.e., persistent). For example, in the case of persistent amnestic-confabulatory symptoms in a man with a severe alcohol use disorder, the diagnosis is F10.26 severe alcohol use disorder with alcohol-induced major neurocognitive disorder, amnestic-confabulatory type, persistent. A separate diagnosis of the comorbid severe alcohol use disorder is not given. If the substance-induced neurocognitive disorder occurs without a comorbid substance use disorder (e.g., after a sporadic heavy use of inhalants), no accompanying substance use disorder is noted (e.g., F18.988 [specific inhalant]–induced mild neurocognitive disorder).

Diagnostic Features

Substance/medication-induced major or mild NCD is characterized by neurocognitive impairments that persist beyond the usual duration of intoxication and acute withdrawal (Criterion B). Initially, these manifestations can reflect slow recovery of brain functions from a period of prolonged substance use, and improvements in neurocognitive as well as brain imaging indicators may be seen over many months. If the disorder continues for an extended period, *persistent* should be specified. The given substance and its use must be known to be capable of causing the observed impairments (Criterion C). While nonspecific decrements in a range of cognitive abilities can occur with nearly any substance of abuse and a variety of medications, some patterns occur more frequently with selected drug classes. For example, NCD due to sedative, hypnotic, or anxiolytic drugs (e.g., benzodiazepines, barbiturates) may show greater disturbances in memory than in other cognitive functions. NCD induced by alcohol frequently manifests with a combination of impairments in executive-function and memory and learning domains. The temporal course of the substance-induced NCD must be consistent with that of use of the given substance (Criterion D). Alcohol-induced, amnestic-confabulatory type (Korsakoff's) NCD is characterized by an impairment in recent memory that is out of proportion to additional NCD symptoms. Features include prominent amnesia (severe difficulty learning new information with rapid forgetting) and a tendency to confabulate, although confabulation can be seen with any severe diminution of recent memory. These manifestations may co-occur with signs of thiamine encephalopathy (Wernicke's

encephalopathy) with associated features such as nystagmus and ataxia. Ophthalmoplegia of Wernicke's encephalopathy is typically characterized by a lateral gaze paralysis. The neurocognitive deficits associated with inhalant misuse include diminished executive functioning, slower cognitive speed, and additional impaired performance on aspects of the Wisconsin Card Sorting and the Stroop tests. Neurocognitive symptoms associated with stimulant use include difficulties with learning and memory and executive function. Methamphetamine use can also be associated with

evidence of vascular injury (e.g., focal weakness, unilateral incoordination, asymmetrical reflexes). The most common neurocognitive profile approximates that seen in vascular NCD. Substances that cause NCD included in the other (or unknown) substance category include intrathecal methotrexate and organophosphate insecticides, as well as compounds that are misused and known to induce adverse cognitive effects but are less well characterized (e.g., kratom/*Mitragyna speciosa*).

When one is determining the relationship between NCD conditions and any group of drugs, it is important to consider whether the deficit was present before the use of the substance and consequently would not be attributable to the substance—and may have even contributed to poor judgment that resulted in the substance use. For example, evidence of decreased impulse control and related impairment of executive functions have been reported as associated with the onset of the use of stimulants and other drugs. In studies in which neurocognitive function is carefully assessed prior to substance use, and then subjects are followed up over several months or more, the ability of drugs other than alcohol, other depressants, and inhalants to cause clinically significant persistent NCDs is not clear.

Associated Features

Inhalant-induced NCD conditions may be associated with the smell of the inhalant on an individual's breath or a rash around the individual's nose or mouth from "huffing" the drug from a container. These are most often seen

in individuals with limited access to other drugs who have histories of inhalant use as well as the early onset of use of multiple substances, especially if their symptoms fulfill criteria for conduct or antisocial personality disorders. A high risk is also seen in workers exposed to solvents in the workplace. Mild NCD induced by drugs with central nervous system depressant effects may manifest with added symptoms of increased irritability, anxiety, sleep disturbance, and dysphoria. NCD induced by stimulant drugs may manifest with rebound depression, hypersomnia, and apathy. In severe forms of substance/medication-induced major NCD (e.g., associated with long-term alcohol use), there may be prominent neuromotor features, such as incoordination, ataxia related to cerebellar damage, and motor slowing, as well as medical complications such as hypokalemia and cardiac arrhythmias. There may also be loss of emotional control, including aggressive or inappropriate affect, or apathy.

Prevalence

The prevalence of these conditions is not well known. Prevalence figures are more available for the use of these substances and for associated substance use disorders rather than for the neurocognitive conditions. Substance/medication-induced major or mild NCDs are more likely in those who are older, have longer duration of use, and have other risk factors such as nutritional deficits.

For alcohol use disorder, the rate of mild NCD is approximately 30%–40% in the first 2 months of abstinence. Mild NCD may persist, particularly in those who do not achieve stable abstinence until after age 50 years. Major NCD is rare and may result from concomitant nutritional deficits, as in alcohol-induced amnestic-confabulatory NCD. Alcohol-induced major NCD may be more common in men.

Few studies are available regarding the prevalence of NCD from other brain depressant drugs (i.e., sedatives, hypnotics, or anxiolytics), likely reflecting the relative rarity of studies of substance use disorders on these drugs and the relatively low level of heavy and persistent “recreational” use of sedative, hypnotic, or anxiolytic drugs compared with alcohol, cannabis, and many other drugs.

More data are available on the prevalence of inhalant use. Such exposure has been linked to both major and mild NCD of varied duration in both higher- and lower-income populations. However, persistent use to the point of developing an NCD is estimated to be less than 1% of the U.S. population.

In the case of stimulants (methamphetamines and cocaine), cerebrovascular disease can also occur, resulting in diffuse or focal brain injury that can be of mild or major neurocognitive levels.

Development and Course

The onset of substance use disorders tends to occur during late adolescence and peak in the 20s and 30s. Although longer history of severe substance use disorder is associated with greater likelihood of NCD, the relationships are not straightforward, with substantial and even complete recovery of neurocognitive functions being common among persons who achieve stable abstinence prior to age 50 years. Substance/medication-induced major or mild NCD is most likely to become persistent in individuals who continue to have substance use disorders past the age of 50 years, presumably because of a combination of lessened neural plasticity and the onset of other age-related brain changes.

NCD conditions may involve a fairly rapid onset of neurocognitive impairment in individuals whose history includes the use of multiple types of drugs of abuse, especially with an early onset of substance use. Earlier commencement of heavy use, particularly of alcohol, may lead to defects in later neural development (e.g., later stages of maturation of frontal circuitries), which may have effects on social cognition as well as other neurocognitive abilities. For alcohol-induced NCD, there may be an additive effect of aging and alcohol-induced brain injury.

Risk and Prognostic Factors

Risk factors for substance/medication-induced NCDs include older age, longer duration of use, and persistent use past age 50 years.

For alcohol-induced NCD, long-term nutritional deficiencies, liver disease, vascular risk factors, and cardiovascular and cerebrovascular disease may contribute to risk. An increased risk for alcohol-induced, amnestic confabulatory–type NCD occurs in the context of a genetic transketolase deficiency as well as in the context of poor nutrition.

Sedative-, hypnotic-, or anxiolytic-induced NCDs have not been well studied, but these problems may be increased in individuals with long-term anxiety disorders or sleep impairment who have been taking benzodiazepines or other hypnotic medications in increasing amounts for months or years.

Diagnostic Markers

Magnetic resonance imaging (MRI) of individuals with chronic alcohol use disorder frequently reveals cortical thinning, white matter loss, and enlargement of sulci and ventricles. While neuroimaging abnormalities are more common in those with NCDs, it is possible to observe NCDs without neuroimaging abnormalities, and vice versa. Specialized techniques (e.g., diffusion tensor imaging) may reveal damage to specific white matter tracts. Magnetic resonance spectroscopy may reveal reduction in *N*-acetylaspartate, and increase in markers of inflammation (e.g., myoinositol) or white matter injury (e.g., choline). Many of these brain imaging changes and neurocognitive manifestations reverse following successful abstinence. In individuals with methamphetamine use disorder, MRI may also reveal hyperintensities suggestive of microhemorrhages or larger areas of infarction.

Functional Consequences of Substance/Medication-Induced Major or Mild Neurocognitive Disorder

The functional consequences of substance/medication-induced mild NCD are sometimes augmented by reduced cognitive efficiency and difficulty concentrating beyond that seen in many other NCDs. In addition, at both major and mild levels, substance/medication-induced NCDs may have associated motor syndromes that increase the level of functional impairment.

Differential Diagnosis

Individuals with substance use disorders, substance intoxication, and substance withdrawal are at increased risk for other conditions that may independently, or through a compounding effect, result in neurocognitive disturbance. These include history of traumatic brain injury and infections that can accompany substance use disorder (e.g., HIV, hepatitis C virus, syphilis). Therefore, presence of substance/medication-induced major or mild NCD should be differentiated from NCDs arising outside the context of substance use, intoxication, and withdrawal, including these accompanying conditions (e.g., traumatic brain injury).

Comorbidity

Substance use disorders, substance intoxication, and substance withdrawal are highly comorbid with other mental disorders. In general, the higher the exposure to drugs of abuse, the greater the risk for a substance- or medication-induced NCD. Comorbid posttraumatic stress disorder, psychotic disorders, depressive and bipolar disorders, and neurodevelopmental disorders can contribute to neurocognitive impairment in substance users. Traumatic brain injury occurs more frequently with substance use, complicating efforts to determine the etiology of NCD in such cases. Severe, long-term alcohol use disorder can be associated with major organ system disease, including cerebrovascular disease and cirrhosis; inhalant use disorder is associated with higher rates of kidney and liver damage; and amphetamine- and cocaine-induced NCD may be accompanied by major or mild vascular NCD secondary to stimulant use.

Major or Mild Neurocognitive Disorder Due to HIV Infection

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. There is documented infection with human immunodeficiency virus (HIV).
C. The neurocognitive disorder is not better explained by non-HIV conditions, including secondary brain diseases such as progressive multifocal leukoencephalopathy or cryptococcal meningitis.
D. The neurocognitive disorder is not attributable to another medical condition and is not better explained by a mental disorder.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder due to HIV infection, with behavioral disturbance, code first **B20** HIV infection, followed by **F02.81** major neurocognitive disorder due to HIV infection, with behavioral disturbance.

For major neurocognitive disorder due to HIV infection, without behavioral disturbance, code first **B20** HIV infection, followed by **F02.80** major neurocognitive disorder due to HIV infection, without behavioral disturbance.

Note: The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild neurocognitive disorder due to HIV infection, code **G31.84.** (**Note:** Do *not* use the additional code for HIV infection. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild neurocognitive disorder due to HIV infection: Use additional code(s) to indicate clinically significant psychiatric symptoms due to HIV infection (e.g., **F06.34** bipolar and related disorder due to HIV infection, with mixed features; **F07.0** personality change due to traumatic brain injury, apathetic type).

Diagnostic Features

HIV disease is caused by infection with human immunodeficiency virus type-1 (HIV-1), which is acquired through exposure to bodily fluids of an infected individual through injection substance use, unprotected sexual contact, or accidental or iatrogenic exposure (e.g., needle puncture injury to medical personnel). HIV infects several types of cells, most particularly "T-helper" (CD4) lymphocytes and monocytes. Over time, the infection can cause severe decreases in the CD4 count, resulting in severe immunocompromise, often leading to opportunistic infections and neoplasms. Infected monocytes can enter the central nervous system, leading to infection of macrophages and microglia. A small percentage of astrocytes may harbor productive HIV infection. The advanced form of HIV infection is termed *acquired immune deficiency syndrome* (AIDS). Diagnosis of HIV is confirmed by established laboratory methods, such as the reverse-transcription polymerase chain reaction (RT-PCR) assay for HIV RNA and the antibody/antigen combination test. Of note, in-home self-testing for HIV is available.

Some individuals with HIV infection develop a neurocognitive disorder (NCD), which generally shows a "subcortical pattern" with prominently impaired executive function, slowing of processing speed, problems with more demanding attentional tasks, and difficulty in learning new information, but fewer problems with recall of learned information. In major NCD, slowing may be prominent. Language difficulties, such as aphasia, are uncommon, although reductions in fluency may be observed. HIV pathogenic processes can affect any part of the brain; therefore, other patterns are possible.

Associated Features

Major or mild NCD due to HIV infection is more prevalent in individuals of increasing age, lower educational level, or female sex, and among those with major depressive disorder, alcohol or other substance use disorders, and medical comorbidities (particularly diabetes and hypertension). The NCD risk due to HIV infection is also increased with any of the following: prior episodes of immunosuppression, high viral loads in the cerebrospinal fluid, and increased levels of tumor necrosis factor–alpha (TNF-α),

interleukin-6 (IL-6), C-reactive protein, D-dimer, sCD14, sCD163, and neurofilament light chain in the peripheral blood or clinical laboratory indicators of advanced HIV disease, such as a low CD4 cell nadir, anemia, and hypoalbuminemia. Individuals with major NCD may show more prominent neuromotor features, such as severe incoordination, ataxia, and motor slowing. These features may become more prominent with NCD disease progression.

Prevalence

Depending on the clinical stage of HIV disease, approximately one-third to over one-half of HIV-infected individuals have at least some evidence of a neurocognitive disturbance, but most of these disturbances would not meet criteria for mild NCD and would instead represent individuals with asymptomatic neurocognitive impairment (ANI), who may have substandard performance on one or more tests of neurocognitive abilities but do not have any impairment in functional status. Rates in North America and Western Europe have largely shown that ANI accounts for the majority of neurocognitive disturbances, whereas mild NCD due to HIV accounts for approximately one-quarter of individuals, and major NCD criteria are met for typically less than 5% of individuals with HIV-related neurocognitive disturbances. In Germany, the overall prevalence of HIV-associated NCDs among HIV

clinic participants was 43%, 90% of whom were in treatment: 20% had ANI, 17% had mild NCD, and 6% had HIV-associated dementia. In low- and middle-income countries, prevalence of HIV-associated NCDs is higher among untreated individuals with HIV. In other parts of the world, and in cohorts composed mostly of individuals infected with HIV on effective antiretroviral treatment tested with comprehensive cognitive test batteries, the overall rates of cognitive impairment were found to be around 25%–35%.

In the United States, the incidence of HIV infection is higher in men than in women in every ethnic group. However, evidence supports a sex

difference in NCD due to HIV infection, with more frequent neurocognitive impairment in women, including when sex is maintained as a risk factor in a multivariate analysis. The higher rate of impairment in women may be associated with differences in educational quality.

Development and Course

In the development and course of NCD due to HIV, individuals may have neurocognitive impairment when the HIV infection is asymptomatic; the Centers for Disease Control and Prevention classifies the underlying HIV infection in three stages: asymptomatic, early symptomatic, and late symptomatic/AIDS. The course of NCD due to HIV infection can resolve, improve, remain stable, slowly worsen, rapidly worsen, or have a fluctuating course. Rapid progression of neurocognitive impairment is uncommon in the context of currently available combination antiretroviral treatment, although it may still occur in the context of a subgroup associated with older age, as well as in association with specific comorbidities promoting cognitive impairment. Nevertheless, for the predominant proportion of individuals with HIV, an abrupt change in mental status warrants an evaluation of other medical sources for the cognitive change, including secondary infections. Because HIV infection preferentially affects subcortical regions over the course of illness, including deep white matter, the progression of the disorder follows a subcortical pattern. The subcortical pattern of cognitive impairment is characterized by mental slowing associated with motor dysfunction, procedural learning deficits, and free recall deficits, with relative sparing of recognition memory, verbal abstraction, and naming.

Because HIV infection can affect a variety of brain regions and the illness can take on many different trajectories depending on associated comorbidities and the consequences of HIV infection, the overall course of an NCD due to HIV infection has considerable heterogeneity. A subcortical neurocognitive profile may interact with age over the life course, such that an interaction occurs between age and clinical stage of HIV disease in the domains of episodic memory and motor impairment (e.g., slowed gait). This interaction increases the overall prevalence of neurocognitive impairment and the likelihood that it will be more pronounced in later life.

Acquisition of HIV infection typically occurs in adults in high-income countries, via high-risk behaviors (e.g., unprotected sex; injection substance use) beginning in late adolescence and peaking during young and middle adulthood, with a significant contribution persisting into older age. In lower-income regions, where HIV testing and antiretroviral treatments for pregnant women are not readily available, perinatal transmission is common. The NCD in such infants and children may manifest primarily as neurodevelopmental delay. As individuals treated for HIV survive into older age, additive and interactive neurocognitive effects of HIV and aging, including other NCDs (e.g., due to Alzheimer's disease, due to Parkinson's disease), are possible. More than 50% of individuals with HIV in the United States are older than 50 years. Long-term antiretroviral therapy is indicated for the ongoing control of HIV infection. However, some antiretroviral therapy may be associated with inflammation, neurotoxic effects, and metabolic changes that can lead to vascular compromise and indirectly increase neurocognitive impairment in conjunction with aging and medical comorbidities that may worsen cognition.

Risk and Prognostic Factors

Paradoxically, NCD due to HIV infection has not declined significantly with the advent of effective antiretroviral therapy, although the most severe presentations (consistent with the diagnosis of major NCD) have decreased sharply. Contributory factors may include inadequate control of HIV in the central nervous system (CNS), the evolution of antiretroviral drug-resistant viral strains, the effects of chronic long-term systemic and brain inflammation, and the effects of comorbid factors such as aging, substance use disorder, hypertension, diabetes, past history of CNS trauma, and co-infections, such as with the hepatitis C virus. Chronic exposure to antiretroviral drugs has also been associated with neurotoxicity in its own right.

Diagnostic Markers

An HIV diagnosis may be made from a test conducted on the blood, oral fluids, or urine. In addition, HIV characterization of the cerebrospinal fluid may be helpful if it reveals a disproportionately high viral load in the cerebrospinal fluid versus in the plasma or if there are indicators of a high level of neuroinflammation. Neuroimaging (e.g., magnetic resonance imaging [MRI]) may reveal reduction in total brain volume, cortical thinning, reduction in white matter volume, and patchy areas of abnormal white matter (hyperintensities). MRI of the brain or lumbar puncture may be helpful to exclude a specific medical condition (e.g., cryptococcal meningitis, meningoencephalitis, herpes simplex virus type 1 or type 2 encephalitis, progressive multifocal leukoencephalopathy) that might contribute to CNS changes in the context of AIDS. Specialized techniques such as diffusion tensor imaging may reveal damage to specific white matter tracts. Arterial spin-labeling (ASL) developed as a new type of MRI (ASL-MRI) may reveal regional changes in brain perfusion in 3–5 minutes without infusion of extrinsic tracers, and translocator protein 18-kDa (TSPO) positron emission tomography scanning may reveal neuroinflammation.

Functional Consequences of Major or Mild Neurocognitive Disorder Due to HIV Infection

Functional consequences of major or mild NCD due to HIV infection are variable across individuals. Thus, impaired executive functions and slowed information processing may substantially interfere with adherence to the effective antiretroviral therapy regimens, although these regimens have been greatly simplified since their inception. Thus, functional status must be assessed and mapped directly to neurocognitive impairment in order to determine the severity of the NCD. Functional status related to neurocognitive impairment due to HIV should be separated from dysfunction attributable to other concomitant disorders that can affect neurocognitive function.

Differential Diagnosis

In the presence of comorbidities, such as other infections (e.g., hepatitis C virus, syphilis), substance use disorder (e.g., methamphetamine use

disorder), prior traumatic brain injury, or neurodevelopmental conditions, major or mild NCD due to HIV infection can be diagnosed provided there is evidence that infection with HIV has worsened any NCDs because of such preexisting or comorbid conditions. Among older adults, onset of neurocognitive decline related to cerebrovascular disease or primary neurodegeneration (e.g., major or mild NCD due to Alzheimer's disease) may need to be differentiated; these conditions may be suggested by a relatively more progressive course of decline than is seen in NCD due to HIV. HIV infection itself has been shown to increase the risk of cerebrovascular disease. Because more severe immunodeficiency can result in opportunistic infections of the brain (e.g., toxoplasmosis; cryptococcosis) and neoplasia (e.g., CNS lymphoma), sudden

onset of an NCD or sudden worsening of an NCD demands active investigation of non-HIV etiologies. Delirium is important to consider because it occurs frequently over the disease course of individuals with HIV and may be due to multiple etiologies (including SARS-CoV-2 co-infection).

Comorbidity

HIV disease is accompanied by chronic systemic and CNS inflammation and diseases that can be associated with an NCD. These complications can be part of the pathogenesis of major or mild NCD as well as ANI due to HIV infection. HIV frequently co-occurs with conditions such as substance use disorders and other sexually transmitted infections. Both medical and psychiatric comorbidities have been identified that increase the likelihood of a diagnosis of NCD due to HIV infection. Women and members of underserved ethnic and racialized groups may show variation in the rates of the comorbidities associated with NCD due to HIV infection.

Major or Mild Neurocognitive Disorder Due to Prion Disease

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. There is insidious onset, and rapid progression of impairment is common.
C. There are motor features of prion disease, such as myoclonus or ataxia, or biomarker evidence.
D. The neurocognitive disorder is not attributable to another medical condition and is not better explained by another mental disorder.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder due to prion disease, with behavioral disturbance, code first **A81.9** prion disease, followed by **F02.81** major neurocognitive disorder due to prion disease, with behavioral disturbance.

For major neurocognitive disorder due to prion disease, without behavioral disturbance, code first **A81.9** prion disease, followed by **F02.80** major neurocognitive disorder due to prion disease, without behavioral disturbance.

Note: The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild neurocognitive disorder due to prion disease, code **G31.84.** (**Note:** Do *not* use the additional code for prion disease. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild neurocognitive disorder due to prion disease: Use additional code(s) to indicate clinically significant psychiatric symptoms due to prion disease (e.g., **F06.2** psychotic disorder due to prion disease, with delusions; **F06.32** depressive disorder due to prion disease with major depressive–like episode).

Diagnostic Features

The classification of major or mild neurocognitive disorder (NCD) due to prion disease includes NCDs due to a group of subacute spongiform encephalopathies (including sporadic Creutzfeldt-Jakob disease, genetic Creutzfeldt-Jakob disease, iatrogenic Creutzfeldt-Jakob disease, variant Creutzfeldt-Jakob disease, variably protease-sensitive prionopathy,

kuru [found among the Fore people in Papua New Guinea], Gerstmann-Sträussler-Scheinker syndrome, and fatal insomnia) caused by transmissible agents known as *prions*. Given that the most common type is sporadic Creutzfeldt-Jakob disease, it is typically referred to as simply Creutzfeldt-Jakob disease (CJD). Variant CJD is much rarer and is associated with transmission of bovine spongiform encephalopathy, also called “mad cow disease.” Typically, individuals with CJD present with neurocognitive deficits, ataxia, and abnormal movements such as myoclonus, chorea, or dystonia; a startle reflex is also common. The history often reveals rapid progression to major NCD over as little as 6 months, and thus the disorder is typically seen only at the major level. However, many individuals with the disorder may have atypical presentations, and the disease can be confirmed only by biopsy or at autopsy. For example, individuals with variant CJD may present with a greater preponderance of psychiatric symptoms than do individuals with other types of prion disease, characterized by low mood, withdrawal, and anxiety. Although biomarker evidence is not necessarily required for the diagnosis if the motor features of prion disease (e.g., myoclonus, ataxia) are present, confidence that the NCD is due to prion disease is greatly increased if characteristic biomarkers are present.

Prevalence

Prevalence is unknown but very low given the short survival. Based on data from nine high-income countries, the annual incidence of sporadic CJD is approximately one or two cases per million people. Incidence varies by age and is highest in those age 65 years or older (4.8/1,000,000 individuals) and is higher in Whites compared with Blacks. Incidence among ethnic Chinese

in Taiwan is lower than general population rates in the United States and other reporting countries.

Development and Course

Prion disease may develop at any age in adults—the peak age for sporadic CJD is approximately 67 years—although it has been reported to occur in individuals spanning the teenage years to late life. Non-Latinx Whites were found to have an older mean age at onset compared with other ethnic and racialized populations in the United States. Prodromal symptoms of prion disease may include fatigue, anxiety, problems with appetite or sleeping, or difficulties with concentration. After several weeks, these symptoms may be followed by incoordination, altered vision, or abnormal gait or other movements that may be myoclonic, choreoathetoid, or ballistic, along with a rapidly progressive dementia. The disease typically progresses very rapidly to the major level of impairment over several months. More rarely, it can progress over 2 years and appear similar in its course to other NCDs.

Risk and Prognostic Factors

Environmental. Cross-species transmission of prion infections, with agents that are closely related to the human form, has been demonstrated (e.g., the outbreak of bovine spongiform encephalopathy inducing variant CJD in the United Kingdom during the mid-1990s). Transmission by corneal transplantation, cadaveric dura mater grafts, contaminated neurosurgical instruments, cadaver-derived human growth hormone and pituitary gonadotropin injections, and blood transfusion (only in the case of variant CJD) has been documented. Studies have not demonstrated an increased risk of sporadic CJD in health care professionals.

Genetic and physiological. In up to 15% of prion disease cases, there are autosomal dominant genetic mutations in the prion protein gene (*PRNP*), which encodes for a normal neuronal membrane–bound protein. The codon 129 polymorphism of *PRNP* mediates the risk

of sporadic and acquired prion diseases as well as modifies clinical manifestation, age at disease onset, and disease duration.

Diagnostic Markers

Prion disease can be definitively confirmed only by brain biopsy or at autopsy. There are several cerebrospinal fluid (CSF) proteins that are markers of neuronal injury and are frequently elevated in prion disease; the ones most commonly used for diagnostic purposes are 14-3-3 and tau, which have high sensitivity but variable specificity. Real-time quaking induced conversion (RT-QuIC) is another CSF diagnostic test that is able to amplify minute amounts of disease-causing prion proteins and has extremely high specificity. Magnetic resonance brain imaging is currently considered the most sensitive diagnostic test when DWI (diffusion-weighted imaging) is performed, with the most common finding being multifocal gray matter hyperintensities in subcortical and/or cortical regions. In some individuals, the electroencephalogram reveals periodic sharp, often triphasic and synchronous discharges at a rate of 0.5–2 Hz at some point during the course of the disorder. It is important to note that the above diagnostic markers vary across prion disease type (e.g., sporadic CJD, genetic CJD, variant CJD).

Differential Diagnosis

Other major neurocognitive disorders. Major NCD due to prion disease may appear similar in its course to other NCDs, but prion diseases are typically distinguished by their rapid progression and prominent cerebellar and motor symptoms.

Major or Mild Neurocognitive Disorder Due to Parkinson's Disease

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.

B. The disturbance occurs in the setting of established Parkinson's disease.
C. There is insidious onset and gradual progression of impairment.
D. The neurocognitive disorder is not attributable to another medical condition and is not better explained by another mental disorder.

Major or mild neurocognitive disorder probably due to Parkinson's disease should be diagnosed if 1 and 2 are both met. **Major or mild neurocognitive disorder possibly due to Parkinson's disease** should be diagnosed if 1 or 2 is met:

1. There is no evidence of mixed etiology (i.e., absence of other neurodegenerative or cerebrovascular disease or another neurological, mental, or systemic disease or condition likely contributing to cognitive decline).
2. The Parkinson's disease clearly precedes the onset of the neurocognitive disorder.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder probably or possibly due to Parkinson's disease, with behavioral disturbance, code first **G20** Parkinson's disease, followed by **F02.81.**

For major neurocognitive disorder probably or possibly due to Parkinson's disease, without behavioral disturbance, code first **G20** Parkinson's disease, followed by **F02.80.**

Note: The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild neurocognitive disorder due to Parkinson's disease, code **G31.84.** (**Note:** Do *not* use the additional code for Parkinson's disease. "With behavioral disturbance" and "without

behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild neurocognitive disorder due to Parkinson's disease: Use additional code(s) to indicate clinically significant psychiatric symptoms due to Parkinson's disease (e.g., **F06.0** psychotic disorder due to Parkinson's disease, with hallucinations; **F06.31** depressive disorder due to Parkinson's disease, with depressive features; **F07.0** personality change due to traumatic brain injury, apathetic type).

Diagnostic Features

The essential feature of major or mild neurocognitive disorder (NCD) due to Parkinson's disease is cognitive decline observed at the time of or following the onset of idiopathic Parkinson's disease. The disturbance must occur in the setting of established Parkinson's disease (Criterion B), and deficits must have developed gradually (Criterion C). The rate of progression of cognitive deficits may vary; for some individuals with mild deficits there may be very minimal change over time.

The NCD is viewed as *probably* due to Parkinson's disease when there is no evidence of another disorder that might be responsible for the cognitive decline *and* when the Parkinson's disease precedes onset of the NCD. The NCD is considered *possibly* due to Parkinson's disease when only one of these conditions is met but not both conditions. A diagnosis of Parkinson's disease prior to the onset of the cognitive change increases the diagnostic confidence that the NCD is attributable to Parkinson's disease, as denoted by the *probable* designation.

Associated Features

Frequently present features include apathy, depressed mood, anxious mood, hallucinations, delusions, personality changes, rapid eye movement (REM) sleep behavior disorder, excessive daytime sleepiness, freezing of gait, falls, bilateral involvement early in disease, postural instability and gait disturbance (PIGD) subtype, and hyposmia. The combination of postural and gait instability may occur early in the disease and may be described by

the term *PIGD subtype* to distinguish from tremor-predominant Parkinson's disease.

Prevalence

The prevalence of Parkinson's disease in the United States steadily increases with age from approximately 0.4% between ages 60 and 69 years to 1.4% between ages 80 and 89 years. Parkinson's disease is more common in men than in women. Similarly, the prevalence of NCD due to Parkinson's disease is higher in men than in women. However, it is not clear if the incidence of NCD due to Parkinson's disease is higher in men than in women. Among individuals with Parkinson's disease, as many as 80% will eventually develop a major NCD. Among those without a major NCD, the prevalence of mild NCD in Parkinson's disease has been estimated at 25%–27%. For individuals with incident-untreated Parkinson's disease, a range of 9%–19% have mild NCD, whereas other studies have reported major NCD occurring in 24% of newly diagnosed untreated Parkinson's disease. Among African Americans, the risk of Parkinson's disease tends to be lower than among non-Latinx Whites, but the risk of dementia among those with the disease tends to be higher.

Development and Course

Onset of Parkinson's disease is typically between ages 50 and 89 years, with most expression in the early 60s. Mild NCD often develops relatively early in the course of Parkinson's disease, whereas major impairment typically does not occur until individuals are older.

Risk and Prognostic Factors

Environmental. Risk factors for Parkinson's disease include exposure to pesticides, solvents, and possibly traumatic brain injury.

Genetic and physiological. Potential risk factors for NCD among individuals with Parkinson's disease include older age at disease onset, increasing severity of disease, prominent gait symptoms, severe autonomic disturbance

(particularly orthostatic hypotension), REM sleep behavior disorder, and possibly being a man and having fewer years of formal education. Individuals with Parkinson's disease with glucocerebrosidase gene (*GBA*) mutations and *APOE*E4* genotype have been demonstrated to have worse cognition in cross-sectional and longitudinal research.

Culture-Related Diagnostic Issues

Guam dementia is a late-onset NCD observed among 8.8% of Chamorros (the indigenous population of Guam) age 65 years and older. Characterized by neurofibrillary tangles but without the amyloid plaques found in Alzheimer's disease, it is thought to be possibly related to a unique parkinsonism-dementia complex and amyotrophic lateral sclerosis. An association has been found with processing and eating fadang made with cycad seeds.

Diagnostic Markers

Neuropsychological testing, with a focus on tests that are not affected by motor slowing (i.e., not timed or requiring use of hands), is critical in detecting the core cognitive deficits, particularly at the mild NCD phase. Characteristic features observed in neuropsychological testing early in the disorder may include reduced attention, executive dysfunction, slowed information processing, and deficits in memory and visuospatial function, whereas many language skills may remain intact.

Dopamine transporter scans, such as DaT scans, may differentiate Lewy body–related dementias (i.e., NCD due to Parkinson's disease, NCD with Lewy bodies) from non–Lewy body–related dementias (e.g., NCD due to Alzheimer's disease).

Differential Diagnosis

Major or mild neurocognitive disorder with Lewy bodies (NCDLB). The distinction between NCDLB and NCD due to Parkinson's disease is based on the timing and sequence of motor symptoms and cognitive symptoms.

Consensus criteria for dementia with Lewy bodies separate NCDLB from NCD due to Parkinson's disease by specifying that for dementia to be attributed to Parkinson's disease, the Parkinson's disease diagnosis must be present for at least 1 year before cognitive decline has reached the level of major NCD, whereas for NCDLB, the cognitive symptoms may begin before, with, or in the absence of parkinsonism. By contrast, expert consensus criteria for Parkinson's disease propose that if cognitive decline occurs prior to a motor diagnosis, the diagnosis of Parkinson's disease may still be made; therefore, a clinician may attribute the cognitive decline to the Parkinson's disease and diagnose NCD due to Parkinson's disease. Consequently, the clinician may choose to diagnose NCD due to Parkinson's disease or NCDLB for individuals with major NCD that starts either before or within 12 months of Parkinson's disease. In such circumstances, the clinician decides which diagnosis is more appropriate. If Parkinson's disease has been diagnosed for at least 1 year prior to the onset of cognitive symptoms, then both expert criteria agree that NCD due to Parkinson's disease would typically be the appropriate diagnosis. The timing and sequence of parkinsonism and mild NCD may be particularly difficult to determine, and unspecified NCD may need to be diagnosed until the order of clinical progression becomes evident.

Major or mild neurocognitive disorder due to Alzheimer's disease. The motor features are the key to distinguishing major or mild NCD due to Parkinson's disease from major or mild NCD due to Alzheimer's disease. However, the two disorders can co-occur, and individuals with well-established Alzheimer's disease can develop mild parkinsonism.

Major or mild vascular neurocognitive disorder. Major or mild vascular NCD may manifest with parkinsonian features that may occur as a consequence of diffuse cortical or subcortical small vessel disease. However, the parkinsonian features typically are not sufficient for a diagnosis of Parkinson's disease, and the course of the NCD usually has a clear association with cerebrovascular changes.

Neurocognitive disorder due to another medical condition (e.g., neurodegenerative disorders).
When a diagnosis of major or mild NCD due to Parkinson's disease is being considered, the distinction must also be made from other brain disorders,

such as progressive supranuclear palsy, corticobasal degeneration, multiple system atrophy, tumors, and hydrocephalus.

Antipsychotic (or other dopamine receptor–blocking drug)–induced parkinsonism. Antipsychotic (or other dopamine receptor–blocking drug)–induced parkinsonism can occur in individuals with other NCDs, particularly when antipsychotic medications are prescribed for the behavioral manifestations of such disorders.

Comorbidity

Parkinson's disease may coexist with Alzheimer's disease and cerebrovascular disease, especially in older individuals. Individuals with NCD due to Parkinson's disease may display clinical or biomarker features that suggest the presence of both Parkinson's disease and other pathologies. Evidence for mixed etiology does not preclude the contribution of Parkinson's disease to an NCD. The compounding of multiple pathological features may diminish the functional abilities of individuals with Parkinson's disease. Motor symptoms and frequent co-occurrence of depression, psychosis, REM sleep behavior disorder, or apathy can make functional impairment worse.

Major or Mild Neurocognitive Disorder Due to Huntington's Disease

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. There is insidious onset and gradual progression.
C. There is clinically established Huntington's disease, or risk for Huntington's disease based on family history or genetic testing.
D. The neurocognitive disorder is not attributable to another medical condition and is not better explained by another mental disorder.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder due to Huntington's disease, with behavioral disturbance, code first **G10** Huntington's disease, followed by **F02.81** major neurocognitive disorder due to Huntington's disease, with behavioral disturbance.

For major neurocognitive disorder due to Huntington's disease, without behavioral disturbance, code first **G10** Huntington's disease, followed by **F02.80** major neurocognitive disorder due to Huntington's disease, without behavioral disturbance.

Note: The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild neurocognitive disorder due to Huntington's disease, code **G31.84.** (**Note:** Do *not* use the additional code for Huntington's disease. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild neurocognitive disorder due to Huntington's disease: Use additional code(s) to indicate clinically significant psychiatric symptoms due to Huntington's disease (e.g., **F06.31** depressive disorder due to Huntington's disease with depressive features; **F06.4** anxiety disorder due to Huntington's disease).

Diagnostic Features

Progressive cognitive impairment is a core feature of Huntington's disease, with early changes in executive function (i.e., processing speed, organization, and planning) typically being more prominent than decline in learning and memory. Cognitive and associated behavioral changes often precede the emergence of the typical motor abnormalities of bradykinesia (i.e., slowing of voluntary movement) and chorea (i.e., involuntary jerking movements). A diagnosis of definite Huntington's disease is given in the

presence of unequivocal, extrapyramidal motor abnormalities in an individual with either a family history of Huntington's disease or genetic testing showing a CAG trinucleotide repeat expansion in the *HTT* gene, located on chromosome 4.

Associated Features

Irritability, apathy, anxiety, obsessive-compulsive symptoms, depression, and, more rarely, psychosis can all be associated with Huntington's disease and often precede the onset of motor symptoms.

Prevalence

Neurocognitive deficits are an eventual outcome of Huntington's disease; the worldwide prevalence is estimated to be 2.7 per 100,000. The prevalence of Huntington's disease in North America, Europe, and Australia is 5.7 per 100,000, with a much lower prevalence of 0.40 per 100,000 in Asia.

Development and Course

The age at diagnosis of Huntington's disease varies widely, but symptoms are most often observed between ages 35 and 45 years. Age at onset is inversely correlated with CAG expansion length. Juvenile Huntington's disease (onset before age 20) may present more commonly with bradykinesia, dystonia, and rigidity than with the choreic movements characteristic of the adult-onset disorder. The disease is gradually progressive, with the average length of survival after clinical diagnosis estimated to be approximately 10–20 years, although affected individuals may demonstrate significant variability in disease progression.

Phenotypic expression of Huntington's disease varies by presence of motor, cognitive, and psychiatric symptoms. Psychiatric and cognitive abnormalities can predate the motor abnormality by a decade or more. Initial symptoms requiring care often include irritabity, anxiety, or depressed mood. Other behavioral disturbances may include pronounced apathy, disinhibition, impulsivity, and impaired insight, with apathy often becoming more progressive over time. Early movement symptoms may

involve the appearance of fidgetiness of the extremities as well as mild *apraxia* (i.e., difficulty with purposeful movements), particularly with fine motor tasks. As the disorder progresses, other motor problems include impaired gait (*ataxia*) and postural instability. Motor impairment eventually affects speech production (*dysarthria*) such that the speech becomes very difficult to understand, which may result in significant distress resulting from the communication barrier in the context of comparatively intact cognition. Advanced motor disease severely affects gait with

progressive ataxia. Eventually individuals become nonambulatory. End-stage motor disease impairs motor control of eating and swallowing, typically a major contributor to the death of the individual from aspiration pneumonia.

Risk and Prognostic Factors

Genetic and physiological. The genetic basis of Huntington's disease is a fully penetrant autosomal dominant expansion of the CAG trinucleotide, often called a *CAG repeat* in the huntingtin gene. A repeat length of 40 or more is invariably associated with Huntington's disease, with longer repeat lengths associated with early age at onset. A CAG repeat length in the 36–39 range is considered to be partially penetrant, which means that this length could or could not lead to Huntington's disease. If Huntington's disease does occur with repeat lengths in this range, it is more often associated with onset late in life (diagnosis after age 70).

Diagnostic Markers

Genetic testing is the primary laboratory test for the determination of Huntington's disease, which is an autosomal dominant disorder with complete penetrance. The trinucleotide CAG is observed to have a repeat expansion in the gene that encodes huntingtin protein on chromosome 4. A diagnosis of Huntington's disease is not made in the presence of the gene expansion alone, but the diagnosis is made only after motor symptoms become manifest. Some individuals with a positive family history request

genetic testing in a presymptomatic stage. Associated features may also include neuroimaging changes; volume loss in the basal ganglia, particularly the caudate nucleus and putamen, is well known to occur and progresses over the course of illness. Other structural and functional changes have been observed in brain imaging but remain research measures.

Association With Suicidal Thoughts or Behavior

In Huntington's disease, an elevated suicide risk compared with the general population has been well documented. A literature review and report of data from a large observational study found that suicide is among the leading causes of death in Huntington's disease. The elevated risk of suicidal thoughts in Huntington's disease has been shown in diagnosed individuals both prior to and after manifesting the motor symptoms of Huntington's disease. Risk factors for suicidal thoughts include depressive symptoms, anxiety, irritability, psychosis, and apathy—emphasizing the importance of treating depressive symptoms and assessing suicidal thoughts during clinical monitoring. A large European cohort study of Huntington's disease similarly found that the most frequent causes of death were pneumonia (19.5%), other infections (6.9%), and suicide (6.6%).

Functional Consequences of Major or Mild Neurocognitive Disorder Due to Huntington's Disease

In the prodromal phase of illness and at early diagnosis, occupational decline is most common, with most individuals reporting some loss of ability to engage in their typical work. The emotional, behavioral, and cognitive aspects of Huntington's disease, such as disinhibition and personality changes, are highly associated with functional decline. Cognitive deficits that contribute most to functional decline may include speed of processing, initiation, and attention rather than memory impairment. Given that Huntington's disease onset occurs in productive years of life, it may have a very disruptive effect on performance in the work setting as well as social life, family life, and important aspects of daily functioning such as driving. As the disease progresses, disability from problems such as

impaired gait, dysarthria, and impulsive or irritable behaviors may substantially add to the level of impairment and daily care needs, over and above the care needs attributable to cognitive decline. Severe choreic movements may substantially interfere with provision of care such as bathing, dressing, and toileting.

Differential Diagnosis

Other mental disorders. Early symptoms of Huntington's disease may include instability of mood, irritability, or compulsive behaviors that may suggest another mental disorder. However, genetic testing or the development of motor symptoms will distinguish the presence of Huntington's disease. In such cases, if the mood symptoms are a focus of clinical attention, they may be indicated by an additional diagnosis of depressive disorder due to Huntington's disease, with depressive features.

Other neurocognitive disorders. The early symptoms of Huntington's disease, particularly symptoms of executive dysfunction and impaired psychomotor speed, may resemble other neurocognitive disorders (NCDs), such as major or mild vascular NCD.

Other movement disorders. Huntington's disease must also be differentiated from other disorders or conditions associated with chorea, such as Wilson's disease, drug-induced tardive dyskinesia, Sydenham's chorea, systemic lupus erythematosus, or senile chorea. Rarely, individuals may present with a course similar to that of Huntington's disease but without positive genetic testing; this is considered to be a Huntington's disease phenocopy that results from a variety of potential genetic factors.

Major or Mild Neurocognitive Disorder Due to Another Medical Condition

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. There is evidence from the history, physical examination, or laboratory findings that the neurocognitive disorder is the pathophysiological consequence of another medical condition (e.g., multiple sclerosis).
C. The cognitive deficits are not better explained by another mental disorder (e.g., major depressive disorder) or another specific neurocognitive disorder (e.g., major neurocognitive disorder due to Alzheimer's disease).

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder due to another medical condition, with behavioral disturbance, code first the other medical condition, followed by the major neurocognitive disorder due to another medical condition, with behavioral disturbance (e.g., **G35** multiple sclerosis, **F02.81** major neurocognitive disorder due to multiple sclerosis, with behavioral disturbance).

For major neurocognitive disorder due to another medical condition, without behavioral disturbance, code first the other medical condition, followed by the major neurocognitive disorder due to another medical condition, without behavioral disturbance (e.g., **G35** multiple sclerosis, **F02.80** major neurocognitive disorder due to multiple sclerosis, without behavioral disturbance).

Note: The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For mild neurocognitive disorder due to another medical condition, code **G31.84.** (**Note:** Do *not* use the additional code for the other medical condition. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild neurocognitive disorder due to another medical condition: Use additional code(s) to indicate clinically significant psychiatric symptoms due to another medical condition (e.g., **F06.32** depressive disorder due to multiple sclerosis, with major depressive–like episode).

Diagnostic Features

A number of medical conditions can cause neurocognitive disorders (NCDs) other than those specific etiologies (e.g., Alzheimer's disease) already included in prior NCD criteria sets in this chapter. These conditions include structural lesions (e.g., primary or secondary brain tumors, subdural hematoma, slowly progressive or normal-pressure hydrocephalus), hypoxia related to hypoperfusion from heart failure, endocrine conditions (e.g., hypothyroidism, hypercalcemia, hypoglycemia), nutritional conditions (e.g., deficiencies of thiamine or niacin), other infectious conditions (e.g., neurosyphilis, cryptococcosis), immune disorders (e.g., temporal arteritis, systemic lupus erythematosus), hepatic or renal failure, metabolic conditions (e.g., Kufs' disease, adrenoleukodystrophy, metachromatic leukodystrophy, other storage diseases of adulthood and childhood), and other neurological conditions (e.g., epilepsy, multiple sclerosis). Unusual causes of central nervous system injury, such as electrical shock or intracranial radiation, are generally evident from the history. The temporal association between the onset or exacerbation of the medical condition and the development of the cognitive deficit offers the greatest support that the NCD is a pathophysiological consequence of the medical condition. Diagnostic certainty regarding this relationship may be increased if the neurocognitive deficits ameliorate partially or stabilize in the context of treatment of the medical condition.

Development and Course

Typically the course of the NCD progresses in a manner that is commensurate with progression of the underlying medical condition. In circumstances where the medical condition is treatable (e.g., hypothyroidism), the neurocognitive deficit may improve or at least not

progress. When the medical condition has a deteriorative course (e.g., secondary progressive multiple sclerosis), the neurocognitive deficits will progress along with the temporal course of illness.

Diagnostic Markers

Associated physical examination and laboratory findings and other clinical features depend on the nature and severity of the medical condition.

Differential Diagnosis

Other major or mild neurocognitive disorder. The presence of an attributable medical condition does not entirely exclude the possibility of another etiological type of major or mild NCD. If cognitive deficits persist following successful treatment of an associated medical condition, then another etiology may be responsible for the cognitive decline.

Major or Mild Neurocognitive Disorder Due to Multiple Etiologies

Diagnostic Criteria

A. The criteria are met for major or mild neurocognitive disorder.
B. There is evidence from the history, physical examination, or laboratory findings that the neurocognitive disorder is the pathophysiological consequence of more than one etiological process, excluding substances (e.g., neurocognitive disorder due to Alzheimer's disease with subsequent development of vascular neurocognitive disorder).

   Note: Refer to the diagnostic criteria for the various neurocognitive disorders due to specific medical conditions for guidance on establishing the particular etiologies.

C. The cognitive deficits are not better explained by another mental disorder and do not occur exclusively during the course of a delirium.

Coding note (see coding table on pp. 682–683):

For major neurocognitive disorder due to multiple etiologies, code first all of the etiological medical conditions (with the exception of vascular disease, which is not coded), followed by either **F02.81** for major neurocognitive disorder due to multiple etiologies, with behavioral disturbance; or **F02.80** for major neurocognitive disorder due to multiple etiologies, without behavioral disturbance.

If vascular disease is among the multiple etiological medical conditions, code next either **F01.51** for major vascular neurocognitive disorder, with behavioral disturbance; or **F01.50** for major vascular neurocognitive disorder, without behavioral disturbance. *Note:* The severity specifiers "mild," "moderate," and "severe" cannot be coded for major neurocognitive disorder but should still be recorded.

For example, for a presentation of major neurocognitive disorder, moderate, with a behavioral disturbance, that is judged to be due to Alzheimer's disease, vascular disease, and HIV infection, and in which heavy chronic alcohol use is judged to be a contributing factor, code the following: **G30.9** Alzheimer's disease, **B20** HIV infection; **F02.81** major neurocognitive disorder due to Alzheimer's disease and HIV infection, moderate, with behavioral disturbance; **F01.51** major vascular neurocognitive disorder, moderate, with behavioral disturbance; and **F10.27** severe alcohol use disorder with alcohol-induced major neurocognitive disorder, moderate, nonamnestic-confabulatory type.

For mild neurocognitive disorder due to multiple etiologies, code **G31.84.** (**Note:** Do *not* use the additional codes for the etiologies. "With behavioral disturbance" and "without behavioral disturbance" cannot be coded but should still be recorded.)

For major or mild neurocognitive disorder due to multiple etiologies: Use additional code(s) to indicate clinically significant psychiatric symptoms due to the various etiologies (e.g., **F06.2** psychotic disorder due to Alzheimer's disease, with delusions; **F06.31** depressive disorder due to cerebrovascular disease, with depressive features).

This category is included to cover the clinical presentation of a neurocognitive disorder (NCD) for which there is evidence that multiple medical conditions have played a probable role in the development of the NCD. In addition to evidence indicative of the presence of multiple medical conditions that are known to cause NCD (i.e., findings from the history and physical examination, and laboratory findings), it may be helpful to refer to the diagnostic criteria and text for the various medical etiologies (e.g., NCD due to Parkinson's disease) for more information on establishing the etiological connection for that particular medical condition.

Unspecified Neurocognitive Disorder

R41.9

This category applies to presentations in which symptoms characteristic of a neurocognitive disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the neurocognitive disorders diagnostic class. The unspecified neurocognitive disorder category is used in situations in which the precise etiology cannot be determined with sufficient certainty to make an etiological attribution.

Coding note: For unspecified major or mild neurocognitive disorder, code **R41.9**. (**Note:** Do *not* use additional codes for any

presumed etiological medical conditions. “With behavioral disturbance” and “without behavioral disturbance” cannot be coded but should still be recorded.)

Personality Disorders

This chapter begins with a general definition of personality disorder that applies to each of the 10 specific personality disorders. A *personality disorder* is an enduring pattern of inner experience and behavior that deviates markedly from the norms and expectations of the individual's culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment.

With any ongoing review process, especially one of this complexity, different viewpoints emerge, and an effort was made to accommodate them. Thus, personality disorders are included in both Sections II and III. The material in Section II represents an update of text associated with the same criteria found in DSM-5 (which were carried over from DSM-IV-TR), whereas Section III includes the proposed model for personality disorder diagnosis and conceptualization developed by the DSM-5 Personality and Personality Disorders Work Group. As this field evolves, it is hoped that both versions will serve clinical practice and research initiatives, respectively.

The following personality disorders are included in this chapter.

- **Paranoid personality disorder** is a pattern of distrust and suspiciousness such that others' motives are interpreted as malevolent.
- **Schizoid personality disorder** is a pattern of detachment from social relationships and a restricted range of emotional expression.
- **Schizotypal personality disorder** is a pattern of acute discomfort in close relationships, cognitive or perceptual distortions, and eccentricities of behavior.
- **Antisocial personality disorder** is a pattern of disregard for, and violation of, the rights of others, criminality, impulsivity, and a failure to learn from experience.

- **Borderline personality disorder** is a pattern of instability in interpersonal relationships, self-image, and affects, and marked impulsivity.
- **Histrionic personality disorder** is a pattern of excessive emotionality and attention seeking.
- **Narcissistic personality disorder** is a pattern of grandiosity, need for admiration, and lack of empathy.
- **Avoidant personality disorder** is a pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation.
- **Dependent personality disorder** is a pattern of submissive and clinging behavior related to an excessive need to be taken care of.
- **Obsessive-compulsive personality disorder** is a pattern of preoccupation with orderliness, perfectionism, and control.
- **Personality change due to another medical condition** is a persistent personality disturbance that is judged to be the direct pathophysiological consequence of another medical condition (e.g., frontal lobe lesion).
- **Other specified personality disorder** is a category provided for two situations: 1) the individual's personality pattern meets

the general criteria for a personality disorder, and traits of several different personality disorders are present, but the criteria for any specific personality disorder are not met; or 2) the individual's personality pattern meets the general criteria for a personality disorder, but the individual is considered to have a personality disorder that is not included in the DSM-5 classification (e.g., passive-aggressive personality disorder). **Unspecified personality disorder** is for presentations in which symptoms characteristic of a personality disorder are present but there is insufficient information to make a more specific diagnosis.

The personality disorders are grouped into three clusters based on descriptive similarities. Cluster A includes paranoid, schizoid, and schizotypal personality disorders. Individuals with these disorders often appear odd or eccentric. Cluster B includes antisocial, borderline, histrionic, and narcissistic personality disorders. Individuals with these disorders often appear dramatic, emotional, or erratic. Cluster C includes avoidant, dependent, and obsessive-compulsive personality disorders. Individuals with these disorders often appear anxious or fearful. It should be noted that

this clustering system, although useful in some research and educational situations, has serious limitations and has not been consistently validated. For instance, two or more disorders from different clusters, or traits from several of them, can often co-occur and vary in intensity and pervasiveness.

A review of epidemiological studies from several countries found a median prevalence of 3.6% for disorders in Cluster A, 4.5% for Cluster B, 2.8% for Cluster C, and 10.5% for any personality disorder. Prevalence appears to vary across countries and by ethnicity, raising questions about true cross-cultural variation and about the impact of diverse definitions and diagnostic instruments on prevalence assessments.

Dimensional Models for Personality Disorders

The diagnostic approach used in this manual represents the categorical perspective that personality disorders are qualitatively distinct clinical syndromes. An alternative to the categorical approach is the dimensional perspective that personality disorders represent maladaptive variants of personality traits that merge imperceptibly into normality and into one another. See Section III for a full description of a dimensional model for personality disorders. The DSM-5 personality disorder clusters (i.e., odd-eccentric, dramatic-emotional, and anxious-fearful) may also be viewed as dimensions representing spectra of personality dysfunction on a continuum with other mental disorders. The alternative dimensional models have much in common and together appear to cover the important areas of personality dysfunction. Their integration, clinical utility, and relationship with the personality disorder diagnostic categories and various aspects of personality dysfunction continue to be under active investigation. This includes research on whether the dimensional model can clarify the cross-cultural prevalence variations seen with the categorical model.

General Personality Disorder

Criteria

A. An enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual's culture. This pattern is manifested in two (or more) of the following areas:
    1. Cognition (i.e., ways of perceiving and interpreting self, other people, and events).
    2. Affectivity (i.e., the range, intensity, lability, and appropriateness of emotional response).
    3. Interpersonal functioning.
    4. Impulse control.

B. The enduring pattern is inflexible and pervasive across a broad range of personal and social situations.
C. The enduring pattern leads to clinically significant distress or impairment in social, occupational, or other important areas of functioning.
D. The pattern is stable and of long duration, and its onset can be traced back at least to adolescence or early adulthood.
E. The enduring pattern is not better explained as a manifestation or consequence of another mental disorder.
F. The enduring pattern is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., head trauma).

Diagnostic Features

Personality traits are enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personal contexts. Only when personality traits are inflexible and maladaptive and cause significant functional impairment or subjective distress do they constitute personality disorders. The essential feature of a personality disorder is an enduring pattern of inner experience

and behavior that deviates markedly from the norms and expectations of the individual's culture and is manifested in at least two of the following areas: cognition, affectivity, interpersonal functioning, or impulse control (Criterion A). This enduring pattern is inflexible and pervasive across a broad range of personal and social situations (Criterion B) and leads to clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion C). The pattern is stable and of long duration, and its onset can be traced back at least to adolescence or early adulthood (Criterion D). The pattern is not better explained as a manifestation or consequence of another mental disorder (Criterion E) and is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication, exposure to a toxin) or another medical condition (e.g., head trauma) (Criterion F). Specific diagnostic criteria are also provided for each of the personality disorders included in this chapter.

The diagnosis of personality disorders requires an evaluation of the individual's long-term patterns of functioning, and the particular personality features must be evident by early adulthood. The personality traits that define these disorders must also be distinguished from characteristics that emerge in response to specific situational stressors or more transient mental states (e.g., bipolar, depressive, or anxiety disorders; substance intoxication). The clinician should assess the stability of personality traits over time and across different situations. Although a single interview with the individual is sometimes sufficient for making the diagnosis, it is often necessary to conduct more than one interview and to space these over time. Assessment can also be complicated by the fact that the characteristics that define a personality disorder may not be considered problematic by the individual (i.e., the traits are often ego-syntonic). To help overcome this difficulty, supplementary information from other informants may be helpful.

Development and Course

The features of a personality disorder usually become recognizable during adolescence or early adult life. By definition, a personality disorder is an enduring pattern of thinking, feeling, and behaving that is relatively stable over time. Some types of personality disorder (notably, antisocial and

borderline personality disorders) tend to become less evident or to remit with age, whereas this appears to be less true for some other types (e.g., obsessive-compulsive and schizotypal personality disorders).

Personality disorder categories may be applied with children or adolescents in those relatively unusual instances in which the individual's particular maladaptive personality traits appear to be pervasive, persistent, and unlikely to be limited to a particular

developmental stage or attributable to another mental disorder. It should be recognized that the traits of a personality disorder that appear in childhood will often not persist unchanged into adult life. For a personality disorder to be diagnosed in an individual younger than 18 years, the features must have been present for at least 1 year. The one exception to this is antisocial personality disorder, which cannot be diagnosed in individuals younger than 18 years. Although, by definition, a personality disorder requires an onset no later than early adulthood, individuals may not come to clinical attention until relatively late in life. A personality disorder may be exacerbated following the loss of significant supporting persons (e.g., a spouse) or previously stabilizing social situations (e.g., a job). However, the development of a change in personality in middle adulthood or later life warrants a thorough evaluation to determine the possible presence of a personality change due to another medical condition or an unrecognized substance use disorder.

Culture-Related Diagnostic Issues

Core aspects of personality like emotion regulation and interpersonal functioning are influenced by culture, which also provides means of protection and assimilation and norms for acceptance and denunciation of specific behaviors and personality traits. Judgments about personality functioning must take into account the individual's ethnic, cultural, and social background. Personality disorders should not be confused with problems associated with acculturation following migration or with the expression of habits, customs, or religious and political values based on the

individual's cultural background or context. Behavioral patterns that appear to be rigid and dysfunctional aspects of personality disorder may reflect instead adaptive responses to cultural constraints. For example, reliance on an abusive relationship in a small community where divorce is proscribed may not reflect pathological dependence; conscientious political protest that puts friends and family members at risk with authorities or in conflict with legal norms does not necessarily reflect pathological callousness. There are marked variations in the recognition and diagnosis of personality disorders across cultural, ethnic, and racialized groups. Accuracy of diagnosis can be enhanced by attention to culturally patterned conceptions of self and attachment, assessment biases resulting from clinicians' own cultural backgrounds or use of diagnostic instruments that are not normed to the population being assessed, and the impact of social determinants such as poverty, acculturative stress, racism, and discrimination on feelings, cognitions, and behaviors. It is useful for the clinician, especially when evaluating someone from a different background, to obtain additional information from informants who are familiar with the person's cultural background.

Sex- and Gender-Related Diagnostic Issues

Certain personality disorders (e.g., antisocial personality disorder) are diagnosed more frequently in men. Others (e.g., borderline, histrionic, and dependent personality disorders) are diagnosed more frequently in women; however, in the case of borderline personality disorder, this may be due to higher help-seeking among women. Nonetheless, clinicians must be cautious not to overdiagnose or underdiagnose certain personality disorders in women or in men because of social stereotypes about typical gender roles and behaviors. There is currently insufficient evidence on differences between cis- and transgender individuals with respect to the epidemiology or clinical presentations of personality disorders to draw meaningful conclusions.

Differential Diagnosis

Other mental disorders and personality traits. Many of the specific criteria for the personality disorders describe features (e.g., suspiciousness,

dependency, insensitivity) that are also characteristic of episodes of other mental disorders. A personality disorder should

be diagnosed only when the defining characteristics appeared before early adulthood, are typical of the individual's long-term functioning, and do not occur exclusively during an episode of another mental disorder. It may be particularly difficult (and not particularly useful) to distinguish personality disorders from persistent mental disorders such as persistent depressive disorder that have an early onset and an enduring, relatively stable course. Some personality disorders may have a "spectrum" relationship to other mental disorders (e.g., schizotypal personality disorder with schizophrenia; avoidant personality disorder with social anxiety disorder) based on phenomenological or biological similarities or familial aggregation.

Personality disorders must be distinguished from personality traits that do not reach the threshold for a personality disorder. Personality traits are diagnosed as a personality disorder only when they are inflexible, maladaptive, and persisting and cause significant functional impairment or subjective distress.

Psychotic disorders. For the three personality disorders that may be related to the psychotic disorders (i.e., paranoid, schizoid, and schizotypal), there is an exclusion criterion stating that the pattern of behavior must not have occurred exclusively during the course of schizophrenia, a bipolar or depressive disorder with psychotic features, or another psychotic disorder. When an individual has a persistent mental disorder (e.g., schizophrenia) that was preceded by a preexisting personality disorder, the personality disorder should also be recorded, followed by "premorbid" in parentheses.

Anxiety and depressive disorders. The clinician must be cautious in diagnosing personality disorders during an episode of a depressive disorder or an anxiety disorder, because these conditions may have cross-sectional symptom features that mimic personality traits and may make it more difficult to evaluate retrospectively the individual's long-term patterns of functioning.

Posttraumatic stress disorder.

When personality changes emerge and persist after an individual has been exposed to extreme stress, a diagnosis of posttraumatic stress disorder should be considered.

Substance use disorders. When an individual has a substance use disorder, it is important not to make a personality disorder diagnosis based solely on behaviors that are consequences of substance intoxication or withdrawal or that are associated with activities in the service of sustaining substance use (e.g., antisocial behavior).

Personality change due to another medical condition. When enduring changes in personality arise as a result of the physiological effects of another medical condition (e.g., brain tumor), a diagnosis of personality change due to another medical condition should be considered.

Cluster A Personality Disorders

Paranoid Personality Disorder

Diagnostic Criteria F60.0

A. A pervasive distrust and suspiciousness of others such that their motives are interpreted as malevolent, beginning by early adulthood and present in a variety of contexts, as indicated by four (or more) of the following:

1. Suspects, without sufficient basis, that others are exploiting, harming, or deceiving him or her.
2. Is preoccupied with unjustified doubts about the loyalty or trustworthiness of friends or associates.

3. Is reluctant to confide in others because of unwarranted fear that the information will be used maliciously against him or her.
4. Reads hidden demeaning or threatening meanings into benign remarks or events.
5. Persistently bears grudges (i.e., is unforgiving of insults, injuries, or slights).
6. Perceives attacks on his or her character or reputation that are not apparent to others and is quick to react angrily or to counterattack.
7. Has recurrent suspicions, without justification, regarding fidelity of spouse or sexual partner.

B. Does not occur exclusively during the course of schizophrenia, a bipolar disorder or depressive disorder with psychotic features, or another psychotic disorder and is not attributable to the physiological effects of another medical condition.

Note: If criteria are met prior to the onset of schizophrenia, add "premorbid," i.e., "paranoid personality disorder (premorbid)."

Diagnostic Features

The essential feature of paranoid personality disorder is a pattern of pervasive distrust and suspiciousness of others such that their motives are interpreted as malevolent. This pattern begins by early adulthood and is present in a variety of contexts.

Individuals with this disorder assume that other people will exploit, harm, or deceive them, even if no evidence exists to support this expectation (Criterion A1). They suspect on the basis of little or no evidence that others are plotting against them and may attack them suddenly, at any time and without reason. They often feel that they have been deeply and irreversibly injured by another person or persons even when there is no objective evidence for this. They are preoccupied with unjustified doubts about the loyalty or trustworthiness of their friends and

associates, whose actions are minutely scrutinized for evidence of hostile intentions (Criterion A2). Any perceived deviation from trustworthiness or loyalty serves to support their underlying assumptions. They are so amazed when a friend or associate shows loyalty that they cannot trust or believe it. If they get into trouble, they expect that friends and associates will either attack or ignore them.

Individuals with paranoid personality disorder are reluctant to confide in or become close to others because they fear that the information they share will be used against them (Criterion A3). They may refuse to answer personal questions, saying that the information is “nobody’s business.” They read hidden meanings that are demeaning and threatening into benign remarks or events (Criterion A4). For example, an individual with this disorder may misinterpret an honest mistake by a store clerk as a deliberate attempt to shortchange, or view a casual humorous remark by a coworker as a serious character attack. Compliments are often misinterpreted (e.g., a compliment on a new acquisition is misinterpreted as a criticism for selfishness; a compliment on an accomplishment is misinterpreted as an attempt to coerce more and better performance). They may view an offer of help as a criticism that they are not doing well enough on their own.

Individuals with this disorder persistently bear grudges and are unwilling to forgive the insults, injuries, or slights that they think they have received (Criterion A5). Minor slights arouse major hostility, and the hostile feelings persist for a long time. Because they are constantly vigilant to the harmful intentions of others, they very often feel that their character or reputation has been attacked or that they have been slighted in some other way. They are quick to counterattack and react with anger to perceived insults (Criterion A6). Individuals with this disorder may be pathologically jealous, often suspecting that their spouse

or sexual partner is unfaithful without any adequate justification (Criterion A7). They may gather trivial and circumstantial “evidence” to support their jealous beliefs. They want to maintain complete control of intimate relationships to avoid being betrayed and may constantly question and

challenge the whereabouts, actions, intentions, and fidelity of their spouse or partner.

Paranoid personality disorder should not be diagnosed if the pattern of behavior occurs exclusively during the course of schizophrenia, a bipolar disorder or depressive disorder with psychotic features, or another psychotic disorder, or if it is attributable to the physiological effects of a neurological (e.g., temporal lobe epilepsy) or another medical condition (Criterion B).

Associated Features

Individuals with paranoid personality disorder are generally difficult to get along with and often have problems with close relationships. Their excessive suspiciousness and hostility may be expressed in overt argumentativeness, in recurrent complaining, or by hostile aloofness. They display a labile range of affect, with hostile, stubborn, and sarcastic expressions predominating. Their combative and suspicious nature may elicit a hostile response in others, which then serves to confirm their original expectations.

Because individuals with paranoid personality disorder lack trust in others, they need to have a high degree of control over those around them. They are often rigid, critical of others, and unable to collaborate, although they have great difficulty accepting criticism themselves. They may blame others for their own shortcomings. Because of their quickness to counterattack in response to the threats they perceive around them, they may be litigious and frequently become involved in legal disputes. Individuals with this disorder seek to confirm their preconceived negative notions regarding people or situations they encounter, attributing malevolent motivations to others that are projections of their own fears. They may exhibit thinly hidden, unrealistic grandiose fantasies, are often attuned to issues of power and rank, and tend to develop negative stereotypes of others, particularly those from population groups distinct from their own. Attracted by simplistic formulations of the world, they are often wary of ambiguous situations. They may be perceived as “fanatics” and form tightly knit “cults” or groups with others who share their paranoid belief systems.

Prevalence

The estimated prevalence of paranoid personality based on a probability subsample from Part II of the National Comorbidity Survey Replication was 2.3%. The prevalence of paranoid personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions was 4.4%. A review of six epidemiological studies (four in the United States) found a median prevalence of 3.2%. In forensic settings, the estimated prevalence may be as high as 23%.

Development and Course

Paranoid personality disorder may be first apparent in childhood and adolescence with solitariness, poor peer relationships, social anxiety, underachievement in school, and interpersonal hypersensitivity. Adolescent onset of paranoid personality disorder is associated with a prior history of childhood maltreatment, externalizing symptoms, bullying of peers, and adult appearance of interpersonal aggression.

Risk and Prognostic Factors

Environmental. Exposure to social stressors such as socioeconomic inequality, marginalization, and racism is associated with decreased trust, which in some cases is adaptive.

The combination of social stress and childhood maltreatment accounts for the increased prevalence of paranoid symptoms in social groups facing racial discrimination. Both longitudinal and cross-sectional studies confirm that childhood trauma is a risk factor for paranoid personality disorder.

Genetic and physiological. There is some evidence for an increased prevalence of paranoid personality disorder in relatives of probands with schizophrenia and for a more specific familial relationship with delusional disorder, persecutory type.

Culture-Related Diagnostic Issues

Some behaviors that are influenced by sociocultural contexts or specific life circumstances may be erroneously labeled paranoid and may even be reinforced by the process of clinical evaluation. Migrants, members of socially oppressed ethnic and racialized populations, and other groups facing social adversity, racism, and discrimination may display guarded or defensive behaviors because of unfamiliarity (e.g., language barriers or lack of knowledge of rules and regulations) or in response to the neglect, hostility, or indifference of the majority society. Some cultural groups develop low generalized trust, especially of outgroup members, which may lead to behaviors that can be misjudged as paranoid. These include guardedness, limited outward emotionality, cognitive rigidity, social distance, and hostility or defensiveness in situations experienced as unfair or discriminatory. These behaviors can, in turn, generate anger and frustration in others, including clinicians, thus setting up a vicious cycle of mutual mistrust, which should not be confused with paranoid traits or paranoid personality disorder.

Sex- and Gender-Related Diagnostic Issues

While paranoid personality disorder was found to be more common in men than in women in a meta-analysis relying on clinical and community samples, the National Epidemiologic Survey on Alcohol and Related Conditions found it to be more common in women.

Differential Diagnosis

Other mental disorders with psychotic symptoms. Paranoid personality disorder can be distinguished from delusional disorder, persecutory type; schizophrenia; and a bipolar or depressive disorder with psychotic features because these disorders are all characterized by a period of persistent psychotic symptoms (e.g., delusions and hallucinations). For an additional diagnosis of paranoid personality disorder to be given, the personality disorder must have been present before the onset of psychotic symptoms and must persist when the psychotic symptoms are in remission. When an individual has another persistent mental disorder (e.g., schizophrenia) that was preceded by paranoid personality disorder, paranoid personality disorder should also be recorded, followed by “premorbid” in parentheses.

Personality change due to another medical condition. Paranoid personality disorder must be distinguished from personality change due to another medical condition, in which the traits that emerge are a direct physiological consequence of another medical condition.

Substance use disorders. Paranoid personality disorder must be distinguished from symptoms that may develop in association with persistent substance use.

Paranoid traits associated with physical handicaps. The disorder must also be distinguished from paranoid traits associated with the development of physical handicaps (e.g., a hearing impairment).

Other personality disorders and personality traits. Other personality disorders may be confused with paranoid personality disorder because they have certain features in common. It is therefore important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality features that meet criteria for one or more personality disorders in addition to paranoid personality disorder, all can be diagnosed. Paranoid personality disorder and schizotypal personality disorder share the traits of suspiciousness, interpersonal aloofness, and paranoid ideation, but schizotypal personality disorder also includes symptoms such as magical thinking, unusual perceptual experiences, and odd thinking and speech. Individuals with behaviors that meet criteria for schizoid personality disorder are often perceived as strange, eccentric, cold, and aloof, but they do not usually have prominent paranoid ideation. The tendency of individuals with paranoid personality disorder to react to minor stimuli with anger is also seen in borderline and histrionic personality disorders. However, these disorders are not necessarily associated with pervasive suspiciousness, and borderline personality disorder exhibits higher levels of impulsivity and self-destructive behavior. People with avoidant personality disorder may also be reluctant to confide in others, but more from fear of being embarrassed or found inadequate than from fear of others' malicious intent. Although antisocial behavior may be present in some individuals with paranoid

personality disorder, it is not usually motivated by a desire for personal gain or to exploit others as in antisocial personality disorder, but rather is more often attributable to a desire for revenge. Individuals with narcissistic personality disorder may occasionally display suspiciousness, social withdrawal, or alienation, but this derives primarily from fears of having their imperfections or flaws revealed.

Paranoid traits may be adaptive, particularly in threatening environments. Paranoid personality disorder should be diagnosed only when these traits are inflexible, maladaptive, and persisting and cause significant functional impairment or subjective distress.

Comorbidity

Particularly in response to stress, individuals with this disorder may experience very brief psychotic episodes (lasting minutes to hours). In some instances, paranoid personality disorder may appear as the premorbid antecedent of delusional disorder or schizophrenia. Individuals with paranoid personality disorder may develop major depressive disorder and may be at increased risk for agoraphobia and obsessive-compulsive disorder. Alcohol and other substance use disorders frequently occur. The most common co-occurring personality disorders appear to be schizotypal, schizoid, narcissistic, avoidant, and borderline.

Schizoid Personality Disorder

Diagnostic Criteria F60.1

A. A pervasive pattern of detachment from social relationships and a restricted range of expression of emotions in interpersonal settings, beginning by early adulthood and present in a variety of contexts, as indicated by four (or more) of the following:

   1. Neither desires nor enjoys close relationships, including being part of a family.

2. Almost always chooses solitary activities.
3. Has little, if any, interest in having sexual experiences with another person.
4. Takes pleasure in few, if any, activities.
5. Lacks close friends or confidants other than first-degree relatives.

6. Appears indifferent to the praise or criticism of others.
7. Shows emotional coldness, detachment, or flattened affectivity.

B. Does not occur exclusively during the course of schizophrenia, a bipolar disorder or depressive disorder with psychotic features, another psychotic disorder, or autism spectrum disorder and is not attributable to the physiological effects of another medical condition.

Note: If criteria are met prior to the onset of schizophrenia, add "premorbid," i.e., "schizoid personality disorder (premorbid)."

Diagnostic Features

The essential feature of schizoid personality disorder is a pervasive pattern of detachment from social relationships and a restricted range of expression of emotions in interpersonal settings. This pattern begins by early adulthood and is present in a variety of contexts.

Individuals with schizoid personality disorder appear to lack a desire for intimacy, seem indifferent to opportunities to develop close relationships, and do not seem to derive much satisfaction from being part of a family or other social group (Criterion A1). They prefer spending time by themselves, rather than being with other people. They often appear to be socially isolated or "loners" and almost always choose solitary activities or hobbies that do not include interaction with others (Criterion A2). They prefer mechanical or abstract tasks, such as computer or mathematical games. They may have very little interest in having sexual experiences with another

person (Criterion A3) and take pleasure in few, if any, activities (Criterion A4). There is usually a reduced experience of pleasure from sensory, bodily, or interpersonal experiences, such as walking on a beach at sunset or having sex. These individuals have no close friends or confidants, except possibly a first-degree relative (Criterion A5).

Individuals with schizoid personality disorder often seem indifferent to the approval or criticism of others and do not appear to be bothered by what others may think of them (Criterion A6). They may be oblivious to the normal subtleties of social interaction and often do not respond appropriately to social cues so that they seem socially inept or superficial and self-absorbed. They usually display a “bland” exterior without visible emotional reactivity and rarely reciprocate gestures or facial expressions, such as smiles or nods (Criterion A7). They claim that they rarely experience strong emotions such as anger and joy. They often display a constricted affect and appear cold and aloof. However, in those very unusual circumstances in which these individuals become at least temporarily comfortable in revealing themselves, they may acknowledge having painful feelings, particularly related to social interactions.

Schizoid personality disorder should not be diagnosed if the pattern of behavior occurs exclusively during the course of schizophrenia, a bipolar or depressive disorder with psychotic features, another psychotic disorder, or autism spectrum disorder, or if it is attributable to the physiological effects of a neurological (e.g., temporal lobe epilepsy) or another medical condition (Criterion B).

Associated Features

Individuals with schizoid personality disorder may have particular difficulty expressing anger, even in response to direct provocation, which contributes to the impression that they lack emotion. Their lives sometimes seem directionless, and they may appear to “drift” in their goals. Such individuals often react passively to adverse circumstances and have difficulty responding appropriately to important life events. Because of their lack of social skills and lack of desire for sexual experiences, individuals with this disorder have few friendships, date infrequently, and often do not marry. Occupational functioning may be impaired, particularly if interpersonal

involvement is required, but individuals with this disorder may do well when they work under conditions of social isolation.

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Prevalence

Schizoid personality disorder is uncommon in clinical settings. The estimated prevalence of schizoid personality disorder based on a probability subsample from Part II of the National Comorbidity Survey Replication was 4.9%. The prevalence of schizoid personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions was 3.1%. A review of six epidemiological studies (four in the United States) found a median prevalence of 1.3%.

Development and Course

Schizoid personality disorder may be first apparent in childhood and adolescence with solitariness, poor peer relationships, and underachievement in school, which mark these children or adolescents as different and make them subject to teasing.

Risk and Prognostic Factors

Genetic and physiological. Schizoid personality disorder may have increased prevalence in the relatives of individuals with schizophrenia or schizotypal personality disorder.

Culture-Related Diagnostic Issues

Individuals from a variety of cultural backgrounds sometimes exhibit defensive behaviors and interpersonal styles that may be erroneously labeled as “schizoid.” For example, those who have moved from rural to metropolitan environments may react with “emotional freezing” that may last for several months and manifest as solitary activities, constricted affect, and other deficits in communication. Immigrants from other countries are sometimes mistakenly perceived as cold, hostile, or indifferent, which may be a response to social ostracism from the host society.

Sex- and Gender-Related Diagnostic Issues

While some research suggests that schizoid personality disorder may be more common in men, other research suggests that there is no gender difference in prevalence.

Differential Diagnosis

Other mental disorders with psychotic symptoms. Schizoid personality disorder can be distinguished from delusional disorder, schizophrenia, and a bipolar or depressive disorder with psychotic features because these disorders are all characterized by a period of persistent psychotic symptoms (e.g., delusions and hallucinations). To give an additional diagnosis of schizoid personality disorder, the personality disorder must have been present before the onset of psychotic symptoms and must persist when the psychotic symptoms are in remission. When an individual has a persistent psychotic disorder (e.g., schizophrenia) that was preceded by schizoid personality disorder, schizoid personality disorder should also be recorded, followed by "premorbid" in parentheses.

Autism spectrum disorder. There may be great difficulty differentiating individuals with schizoid personality disorder from individuals with autism spectrum disorder, particularly with milder forms of either disorder, as both include a seeming indifference to companionship with others. However, autism spectrum disorder may be differentiated by stereotyped behaviors and interests.

Personality change due to another medical condition. Schizoid personality disorder must be distinguished from personality change due to another medical condition, in which the traits that emerge are a direct physiological consequence of another medical condition.

Substance use disorders. Schizoid personality disorder must also be distinguished from symptoms that may develop in association with persistent substance use.

Other personality disorders and personality traits.

Other personality disorders may be confused with schizoid personality disorder because they have certain features in common. It is, therefore, important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality features that meet criteria for one or more personality disorders in addition to schizoid personality disorder, all can be diagnosed. Although characteristics of social isolation and restricted affectivity are common to schizoid, schizotypal, and paranoid personality disorders, schizoid personality disorder can be distinguished from schizotypal personality disorder by the lack of cognitive and perceptual distortions and from paranoid personality disorder by the lack of suspiciousness and paranoid ideation. The social isolation of schizoid personality disorder can be distinguished from that of avoidant personality disorder, which is attributable to fear of being embarrassed or found inadequate and excessive anticipation of rejection. In contrast, people with schizoid personality disorder have a more pervasive detachment and limited desire for social intimacy. Individuals with obsessive-compulsive personality disorder may also show an apparent social detachment stemming from devotion to work and discomfort with emotions, but they do have an underlying capacity for intimacy.

Individuals who are “loners” or quite introverted may display personality traits that might be considered schizoid, consistent with the broader conceptualization of schizoid personality disorder as a disorder defined by pathological introversion/detachment. Only when these traits are inflexible and maladaptive and cause significant functional impairment or subjective distress do they constitute schizoid personality disorder.

Comorbidity

Particularly in response to stress, individuals with this disorder may experience very brief psychotic episodes (lasting minutes to hours). In some instances, schizoid personality disorder may appear as the premorbid antecedent of delusional disorder or schizophrenia. Individuals with this disorder may sometimes develop major depressive disorder. Schizoid personality disorder most often co-occurs with schizotypal, paranoid, and avoidant personality disorders.

Schizotypal Personality Disorder

Diagnostic Criteria F21

A. A pervasive pattern of social and interpersonal deficits marked by acute discomfort with, and reduced capacity for, close relationships as well as by cognitive or perceptual distortions and eccentricities of behavior, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Ideas of reference (excluding delusions of reference).
2. Odd beliefs or magical thinking that influences behavior and is inconsistent with subcultural norms (e.g., superstitiousness, belief in clairvoyance, telepathy, or "sixth sense"; in children and adolescents, bizarre fantasies or preoccupations).
3. Unusual perceptual experiences, including bodily illusions.
4. Odd thinking and speech (e.g., vague, circumstantial, metaphorical, overelaborate, or stereotyped).
5. Suspiciousness or paranoid ideation.
6. Inappropriate or constricted affect.

7. Behavior or appearance that is odd, eccentric, or peculiar.
8. Lack of close friends or confidants other than first-degree relatives.
9. Excessive social anxiety that does not diminish with familiarity and tends to be associated with paranoid fears rather than negative judgments about self.

B. Does not occur exclusively during the course of schizophrenia, a bipolar disorder or depressive disorder with

psychotic features, another psychotic disorder, or autism spectrum disorder.

Note: If criteria are met prior to the onset of schizophrenia, add "premorbid," e.g., "schizotypal personality disorder (premorbid)."

Diagnostic Features

The essential feature of schizotypal personality disorder is a pervasive pattern of social and interpersonal deficits marked by acute discomfort with, and reduced capacity for, close relationships as well as by cognitive or perceptual distortions and eccentricities of behavior. This pattern begins by early adulthood and is present in a variety of contexts.

Individuals with schizotypal personality disorder often have ideas of reference (i.e., incorrect interpretations of casual incidents and external events as having a particular and unusual meaning specifically for the person) (Criterion A1). These should be distinguished from delusions of reference, in which the beliefs are held with delusional conviction. These individuals may be superstitious or preoccupied with paranormal phenomena that are outside the norms of their subculture (Criterion A2). They may feel that they have special powers to sense events before they happen or to read others' thoughts. They may believe that they have magical control over others, which can be implemented directly (e.g., believing that their spouse's taking the dog out for a walk is the direct result of thinking an hour earlier it should be done) or indirectly through compliance with magical rituals (e.g., walking past a specific object three times to avoid a certain harmful outcome). Perceptual alterations may be present (e.g., sensing that another person is present or hearing a voice murmuring their name) (Criterion A3). Their speech may include unusual or idiosyncratic phrasing and construction. It is often loose, digressive, or vague, but without actual derailment or incoherence (Criterion A4). Responses can be either overly concrete or overly abstract, and words or concepts are sometimes applied in unusual ways (e.g., the individual may state that he or she was not "talkable" at work).

Individuals with this disorder are often suspicious and may have paranoid ideation (e.g., believing their colleagues at work are intent on undermining their reputation with the boss) (Criterion A5). They are usually

not able to negotiate the full range of affects and interpersonal cuing required for successful relationships and thus often appear to interact with others in an inappropriate, stiff, or constricted fashion (Criterion A6). These individuals are often considered to be odd or eccentric because of unusual mannerisms, an often unkempt manner of dress that does not quite "fit together," and inattention to the usual social conventions (e.g., the individual may avoid eye contact, wear clothes that are ink stained and ill-fitting, and be unable to join in the give-and-take banter of co-workers) (Criterion A7).

Individuals with schizotypal personality disorder experience interpersonal relatedness as problematic and are uncomfortable relating to other people. Although they may express unhappiness about their lack of relationships, their behavior suggests a decreased desire for intimate contacts. As a result, they usually have no or few close friends or confidants other than a first-degree relative (Criterion A8). They are anxious in social situations, particularly those involving unfamiliar people (Criterion A9). They will interact with other individuals when they have to but prefer to keep to themselves because they feel that they are different and just do not "fit in." Their social anxiety does not easily abate, even when they spend more time in the setting or become more familiar with the other people, because their anxiety tends to be associated with suspiciousness regarding others' motivations. For example, when attending a dinner party, the individual with schizotypal

personality disorder will not become more relaxed as time goes on, but rather may become increasingly tense and suspicious.

Schizotypal personality disorder should not be diagnosed if the pattern of behavior occurs exclusively during the course of schizophrenia, a bipolar or depressive disorder with psychotic features, another psychotic disorder, or autism spectrum disorder (Criterion B).

Associated Features

Individuals with schizotypal personality disorder often seek treatment for the associated symptoms of anxiety or depression rather than for the personality disorder features per se.

Prevalence

The estimated prevalence of schizotypal personality disorder based on a probability subsample from Part II of the National Comorbidity Survey Replication was 3.3%.The prevalence of schizotypal personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions data was 3.9%. A review of five epidemiological studies (three in the United States) found a median prevalence of 0.6%.

Development and Course

Schizotypal personality disorder has a relatively stable course, with only a small proportion of individuals going on to develop schizophrenia or another psychotic disorder. Schizotypal personality disorder may be first apparent in childhood and adolescence with solitariness, poor peer relationships, social anxiety, underachievement in school, hypersensitivity, peculiar thoughts and language, and bizarre fantasies. These children may appear “odd” or “eccentric” and attract teasing.

Risk and Prognostic Factors

Genetic and physiological. Schizotypal personality disorder appears to aggregate familially and is more prevalent among the first-degree biological relatives of individuals with schizophrenia than among the general population. There may also be a modest increase in schizophrenia and other psychotic disorders in the relatives of probands with schizotypal personality disorder. Twin studies indicate highly stable genetic factors and rather transient environmental factors for an increased risk for the schizotypal syndrome, and genetic risk variants for schizophrenia may be linked to schizotypal personality disorder. Neuroimaging studies detect group-level differences in the size and function of specific brain regions in individuals with schizotypal personality disorder in comparison with healthy persons,

individuals with schizophrenia, and individuals with other personality disorders.

Culture-Related Diagnostic Issues

Cognitive and perceptual distortions must be evaluated in the context of the individual's cultural milieu. Pervasive culturally determined characteristics, particularly those regarding supernatural and religious beliefs and practices (life beyond death, speaking in tongues, voodoo, shamanism, mind reading, sixth sense, evil eye, magical beliefs related to health and illness), can appear to be schizotypal to the uninformed clinician. Thus, observed cross-national and cross-ethnic variations in the prevalence and expression of schizotypal traits may be a true epidemiological finding or one due to differences in the cultural acceptance of these experiences.

Sex- and Gender-Related Diagnostic Issues

Schizotypal personality disorder appears to be slightly more common in men than in women.

Differential Diagnosis

Other mental disorders with psychotic symptoms. Schizotypal personality disorder can be distinguished from delusional disorder, schizophrenia, and a bipolar or depressive disorder with psychotic features because these disorders are all characterized by a period of persistent psychotic symptoms (e.g., delusions and hallucinations). To give an additional diagnosis of schizotypal personality disorder, the personality disorder must have been present before the onset of psychotic symptoms and persist when the psychotic symptoms are in remission. When an individual has a persistent psychotic disorder (e.g., schizophrenia) that was preceded by schizotypal personality disorder, schizotypal personality disorder should also be recorded, followed by "premorbid" in parentheses.

Neurodevelopmental disorders. There may be great difficulty differentiating children with schizotypal personality disorder from the heterogeneous

group of solitary, odd children whose behavior is characterized by marked social isolation, eccentricity, or peculiarities of language and whose diagnoses would probably include milder forms of autism spectrum disorder or language communication disorders. Communication disorders may be differentiated by the primacy and severity of the disorder in language and by the characteristic features of impaired language found in a specialized language assessment. Milder forms of autism spectrum disorder are differentiated by the even greater lack of social awareness and emotional reciprocity and stereotyped behaviors and interests.

Personality change due to another medical condition. Schizotypal personality disorder must be distinguished from personality change due to another medical condition, in which the traits that emerge are a direct physiological consequence of another medical condition.

Substance use disorders. Schizotypal personality disorder must also be distinguished from symptoms that may develop in association with persistent substance use.

Other personality disorders and personality traits. Other personality disorders may be confused with schizotypal personality disorder because they have certain features in common. It is, therefore, important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality features that meet criteria for one or more personality disorders in addition to schizotypal personality disorder, all can be diagnosed. Although paranoid and schizoid personality disorders may also be characterized by social detachment and restricted affect, schizotypal personality disorder can be distinguished from these two diagnoses by the presence of cognitive or perceptual distortions and marked eccentricity or oddness. Close relationships are limited in both schizotypal personality disorder and avoidant personality disorder; however, in avoidant personality disorder an active desire for relationships is constrained by a fear of rejection, whereas in schizotypal personality disorder there is a lack of desire for relationships and persistent detachment. Individuals with narcissistic personality disorder may also display suspiciousness, social withdrawal, or alienation, but in narcissistic personality disorder these qualities derive primarily from fears of having imperfections or flaws revealed. Individuals with borderline personality disorder may also have

transient, psychotic-like symptoms, but these are usually more closely related to affective shifts in response to stress (e.g., intense anger, anxiety, disappointment) and are usually more dissociative (e.g., derealization, depersonalization). In contrast, individuals with schizotypal personality disorder are more likely to have enduring psychotic-like symptoms that may worsen under stress but are less likely to be invariably associated with pronounced affective symptoms. Although social isolation may occur in borderline personality disorder, it is usually secondary to repeated interpersonal failures due to angry outbursts and frequent mood shifts, rather than a result of a persistent lack of social contacts and desire for intimacy. Furthermore, individuals with schizotypal personality disorder do not usually demonstrate the impulsive or manipulative behaviors of the individual with

borderline personality disorder. However, there is a high rate of co-occurrence between the two disorders, so that making such distinctions is not always feasible. Schizotypal features during adolescence may be reflective of transient emotional turmoil rather than an enduring personality disorder.

Comorbidity

Particularly in response to stress, individuals with this disorder may experience transient psychotic episodes (lasting minutes to hours), although they usually are insufficient in duration to warrant an additional diagnosis such as brief psychotic disorder or schizophreniform disorder. In some cases, clinically significant psychotic symptoms may develop that meet criteria for brief psychotic disorder, schizophreniform disorder, delusional disorder, or schizophrenia. There is considerable co-occurrence with schizoid, paranoid, avoidant, and borderline personality disorders.

Cluster B Personality Disorders

Antisocial Personality Disorder

Diagnostic Criteria F60.2

A. A pervasive pattern of disregard for and violation of the rights of others, occurring since age 15 years, as indicated by three (or more) of the following:

   1. Failure to conform to social norms with respect to lawful behaviors, as indicated by repeatedly performing acts that are grounds for arrest.
   2. Deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure.
   3. Impulsivity or failure to plan ahead.
   4. Irritability and aggressiveness, as indicated by repeated physical fights or assaults.
   5. Reckless disregard for safety of self or others.
   6. Consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations.
   7. Lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another.

B. The individual is at least age 18 years.

C. There is evidence of conduct disorder with onset before age 15 years.

D. The occurrence of antisocial behavior is not exclusively during the course of schizophrenia or bipolar disorder.

Diagnostic Features

The essential feature of antisocial personality disorder is a pervasive pattern of disregard for, and violation of, the rights of others that begins in

childhood or early adolescence and continues into adulthood. This pattern has also been referred to as *psychopathy, sociopathy,* or *dyssocial personality disorder.* Because deceit and manipulation are central features of antisocial personality disorder, it may be especially helpful to integrate information acquired from systematic clinical assessment with information collected from collateral sources.

For this diagnosis to be given, the individual must be at least age 18 years (Criterion B) and must have had evidence of conduct disorder with onset before age 15 years (Criterion C). Conduct disorder involves a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated. The specific behaviors characteristic of conduct disorder fall into one of four categories: aggression to people and animals, destruction of property, deceitfulness or theft, or serious violation of rules.

The pattern of antisocial behavior continues into adulthood. Individuals with antisocial personality disorder fail to conform to social norms with respect to lawful behavior (Criterion A1). They may repeatedly perform acts that are grounds for arrest (whether they are arrested or not), such as destroying property, harassing others, stealing, or pursuing illegal occupations. Persons with this disorder disregard the wishes, rights, or feelings of others. They are frequently deceitful and manipulative in order to gain personal profit or pleasure (e.g., to obtain money, sex, or power) (Criterion A2). They may repeatedly lie, use an alias, con others, or malinger. A pattern of impulsivity may be manifested by a failure to plan ahead (Criterion A3). Decisions are made on the spur of the moment, without forethought and without consideration for the consequences to self or others; this may lead to sudden changes of jobs, residences, or relationships. Individuals with antisocial personality disorder tend to be irritable and aggressive and may repeatedly get into physical fights or commit acts of physical assault (including spouse beating or child beating) (Criterion A4). (Aggressive acts that are required to defend oneself or someone else are not considered to be evidence for this item.) These individuals also display a reckless disregard for the safety of themselves or

others (Criterion A5). This may be evidenced in their driving behavior (i.e., recurrent speeding, driving while intoxicated, multiple accidents). They may engage in sexual behavior or substance use that has a high risk for harmful consequences. They may neglect or fail to care for a child in a way that puts the child in danger.

Individuals with antisocial personality disorder also tend to be consistently and extremely irresponsible (Criterion A6). Irresponsible work behavior may be indicated by significant periods of unemployment despite available job opportunities, or by abandonment of several jobs without a realistic plan for getting another job. There may also be a pattern of repeated absences from work that are not explained by illness either in themselves or in their family. Financial irresponsibility is indicated by acts such as defaulting on debts, failing to provide child support, or failing to support other dependents on a regular basis. Individuals with antisocial personality disorder show little remorse for the consequences of their acts (Criterion A7). They may be indifferent to, or provide a superficial rationalization for, having hurt, mistreated, or stolen from someone (e.g., “life’s unfair,” “losers deserve to lose”). These individuals may blame the victims for being foolish, helpless, or deserving their fate (e.g., “he had it coming anyway”); they may minimize the harmful consequences of their actions; or they may simply indicate complete indifference. They generally fail to compensate or make amends for their behavior. They may believe that everyone is out to “help number one” and that one should stop at nothing to avoid being pushed around.

The antisocial behavior must not occur exclusively during the course of schizophrenia or bipolar disorder (Criterion D).

Associated Features

Individuals with antisocial personality disorder frequently lack empathy and tend to be callous, cynical, and contemptuous of the feelings, rights, and sufferings of others. They may have an inflated and arrogant self-appraisal (e.g., feel that ordinary work is beneath them or lack a realistic concern about their current problems or their future) and may be excessively opinionated, self-assured, or cocky. Some antisocial individuals may display a glib, superficial charm and can be quite voluble and verbally

facile (e.g., using technical terms or jargon that might impress someone who is unfamiliar with the topic). Lack of

empathy, inflated self-appraisal, and superficial charm are features that have been commonly included in traditional conceptions of psychopathy that may be particularly distinguishing of the disorder and more predictive of recidivism in prison or forensic settings, where criminal, delinquent, or aggressive acts are likely to be nonspecific. These individuals may also be irresponsible and exploitative in their sexual relationships. They may have a history of many sexual partners and may never have sustained a monogamous relationship. They may be irresponsible as parents, as evidenced by malnutrition of a child, an illness in the child resulting from a lack of minimal hygiene, a child's dependence on neighbors or nonresident relatives for food or shelter, a failure to arrange for a caretaker for a young child when the individual is away from home, or repeated squandering of money required for household necessities. These individuals may receive dishonorable discharges from the armed services, may fail to be self-supporting, may become impoverished or even homeless, or may spend many years in penal institutions. Individuals with antisocial personality disorder are more likely than individuals in the general population to die prematurely from natural causes and suicide.

Prevalence

The estimated prevalence of antisocial personality disorder based on a probability subsample from Part II of the National Comorbidity Survey Replication was 0.6%.The prevalence of antisocial personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions data was 3.6%. A review of seven epidemiological studies (six in the United States) found a median prevalence of 3.6%. The highest prevalence of antisocial personality disorder (greater than 70%) is among samples of men with the most severe alcohol use disorders and from substance abuse clinics, prisons, or other forensic settings. Lifetime prevalence appears to be similar across non-Latinx White and Black individuals and lower in Latinx

and Asian Americans. Prevalence may be higher in samples affected by adverse socioeconomic (i.e., poverty) or sociocultural (i.e., migration) factors.

Development and Course

Antisocial personality disorder has a chronic course but may become less evident or remit as the individual grows older, often by age 40. Although this remission tends to be particularly evident with respect to engaging in criminal behavior, there is likely to be a decrease in the full spectrum of antisocial behaviors and substance use. By definition, antisocial personality cannot be diagnosed before age 18 years.

Risk and Prognostic Factors

Environmental. Child abuse or neglect, unstable or erratic parenting, or inconsistent parental discipline may increase the likelihood that conduct disorder will evolve into antisocial personality disorder.

Genetic and physiological. Antisocial personality disorder is more common among the first-degree biological relatives of those with the disorder than in the general population. Biological relatives of individuals with this disorder are also at increased risk for somatization disorder (a diagnosis that was replaced in DSM-5 with somatic symptom disorder) and substance use disorders. Within a family that has a member with antisocial personality disorder, males more often have antisocial personality disorder and substance use disorders, whereas females more often have somatization disorder.

Culture-Related Diagnostic Issues

Antisocial personality disorder has been associated with low socioeconomic status and urban settings. The diagnosis may at times be misapplied to individuals in settings in which

seemingly antisocial behavior may be part of a protective survival strategy (e.g., formation of youth gangs in urban areas with high rates of violence

and discrimination). Sociocultural contexts with high rates of child maltreatment or exposure to violence also tend to have elevated prevalence of antisocial behaviors, suggesting either a potential risk factor for the development of antisocial personality disorder or an adverse environment that evokes reactive and contextual antisocial behaviors that do not represent pervasive and enduring traits consistent with a personality disorder. In assessing antisocial traits, it is helpful for the clinician to consider the social and economic context in which the behaviors occur. In the National Epidemiologic Survey on Alcohol and Related Conditions, prevalence appears to vary across U.S. ethnic and racialized groups, possibly because of a combination of true prevalence differences, measurement artifacts, and the impact of adverse environments that generate behaviors that resemble those of antisocial personality disorder but are instead reactive and contextual. Individuals from some socially oppressed groups may be at higher risk for misdiagnosis or overdiagnosis of antisocial personality disorder because they are more likely to be misdiagnosed with conduct disorder in adolescence, which is a requirement for a diagnosis of antisocial personality disorder.

Sex- and Gender-Related Diagnostic Issues

Antisocial personality disorder is three times as common in men than in women. Women with antisocial personality disorder are more likely to have experienced childhood and adult adverse experiences such as sexual abuse compared with men. Clinical presentation may vary, with men more often presenting with irritability/aggression and reckless disregard for the safety of others compared with women. Comorbid substance use disorders are more common in men, while comorbid mood and anxiety disorders are more common in women. There has been some concern that antisocial personality disorder may be underdiagnosed in females, particularly because of the emphasis on aggressive items in the definition of conduct disorder.

Differential Diagnosis

The diagnosis of antisocial personality disorder is not given to individuals younger than 18 years and is given only if there is evidence of conduct

disorder before age 15 years. For individuals older than 18 years, a diagnosis of conduct disorder is given only if the criteria for antisocial personality disorder are not met.

Substance use disorders. When antisocial behavior in an adult is associated with a substance use disorder, the diagnosis of antisocial personality disorder is not made unless the signs of antisocial personality disorder were also present in childhood and have continued into adulthood. When substance use and antisocial behavior both began in childhood and continued into adulthood, both a substance use disorder and antisocial personality disorder should be diagnosed if the criteria for both are met, even though some antisocial acts may be a consequence of the substance use disorder (e.g., illegal selling of drugs, thefts to obtain money for drugs).

Schizophrenia and bipolar disorders. Antisocial behavior that occurs exclusively during the course of schizophrenia or a bipolar disorder should not be diagnosed as antisocial personality disorder.

Other personality disorders. Other personality disorders may be confused with antisocial personality disorder because they have certain features in common. It is therefore important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality features that meet criteria for one or more personality disorders in addition to antisocial personality disorder, all can be diagnosed. Individuals with antisocial personality disorder and narcissistic personality disorder share a tendency to be tough-minded, glib, superficial, exploitative, and lack empathy. However,

narcissistic personality disorder does not include characteristics of impulsivity, aggression, and deceit. In addition, individuals with antisocial personality disorder may not be as needy of the admiration and envy of others, and persons with narcissistic personality disorder usually lack the history of conduct disorder in childhood or criminal behavior in adulthood. Individuals with antisocial personality disorder and histrionic personality disorder share a tendency to be impulsive, superficial, excitement seeking, reckless, seductive, and manipulative, but persons with histrionic

personality disorder tend to be more exaggerated in their emotions and do not characteristically engage in antisocial behaviors. Individuals with histrionic and borderline personality disorders are manipulative to gain nurturance, whereas those with antisocial personality disorder are manipulative to gain profit, power, or some other material gratification. Individuals with antisocial personality disorder tend to be less emotionally unstable and more aggressive than those with borderline personality disorder. Although antisocial behavior may be present in some individuals with paranoid personality disorder, it is not usually motivated by a desire for personal gain or to exploit others as in antisocial personality disorder, but rather is more often attributable to a desire for revenge.

Criminal behavior not associated with a mental disorder. Antisocial personality disorder must be distinguished from antisocial behavior not due to a mental disorder, for example, criminal behavior undertaken for gain that is not accompanied by the personality features characteristic of this disorder. In these cases, the condition adult antisocial behavior may be coded (see "Other Conditions That May Be a Focus of Clinical Attention").

Comorbidity

Individuals with antisocial personality disorder may also experience dysphoria, including complaints of tension, inability to tolerate boredom, and depressed mood. They may have associated anxiety disorders, mood disorders, substance use disorders, somatic symptom disorder, and gambling disorder. Individuals with antisocial personality disorder also often have personality features that meet criteria for other personality disorders, particularly borderline, histrionic, and narcissistic personality disorders. The likelihood of developing antisocial personality disorder in adult life is increased if the individual experienced childhood onset of conduct disorder (before age 10 years) and accompanying attention-deficit/hyperactivity disorder.

Borderline Personality Disorder

Diagnostic Criteria F60.3

A pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Frantic efforts to avoid real or imagined abandonment. (**Note:** Do not include suicidal or self-mutilating behavior covered in Criterion 5.)
2. A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation.
3. Identity disturbance: markedly and persistently unstable self-image or sense of self.
4. Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating). (**Note:** Do not include suicidal or self-mutilating behavior covered in Criterion 5.)
5. Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior.

6. Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days).
7. Chronic feelings of emptiness.
8. Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights).
9. Transient, stress-related paranoid ideation or severe dissociative symptoms.

Diagnostic Features

The essential feature of borderline personality disorder is a pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity that begins by early adulthood and is present in a variety of contexts.

Individuals with borderline personality disorder make frantic efforts to avoid real or imagined abandonment (Criterion 1). The perception of impending separation or rejection, or the loss of external structure, can lead to profound changes in self-image, affect, cognition, and behavior. These individuals are very sensitive to environmental circumstances. They experience intense abandonment fears and inappropriate anger even when faced with a realistic time-limited separation or when there are unavoidable changes in plans (e.g., sudden despair in reaction to a clinician's announcing the end of the hour; panic or fury when someone important to them is just a few minutes late or must cancel an appointment). They may believe that this "abandonment" implies they are "bad." These abandonment fears are related to an intolerance of being alone and a need to have other people with them. Their frantic efforts to avoid abandonment may include impulsive actions such as self-mutilating or suicidal behaviors, which are described separately in Criterion 5 (see also "Association With Suicidal Thoughts or Behavior").

Individuals with borderline personality disorder have a pattern of unstable and intense relationships (Criterion 2). They may idealize potential caregivers or lovers at the first or second meeting, demand to spend a lot of time together, and share the most intimate details early in a relationship. However, they may switch quickly from idealizing other people to devaluing them, feeling that the other person does not care enough, does not give enough, or is not "there" enough. These individuals can empathize with and nurture other people, but only with the expectation that the other person will "be there" in return to meet their own needs on demand. These individuals are prone to sudden and dramatic shifts in their view of others, who may alternatively be seen as beneficent supports or as cruelly punitive. Such shifts often reflect disillusionment with a caregiver whose nurturing qualities had been idealized or whose rejection or abandonment is expected.

There may be an identity disturbance characterized by markedly and persistently unstable self-image or sense of self (Criterion 3). There are sudden and dramatic shifts in self-image (e.g., suddenly changing from the

role of a needy supplicant for help to that of a righteous avenger of past mistreatment). Although they usually have a self-image that is based on the feeling of being bad or evil, individuals with this disorder may at times have feelings that they do not exist at all. This can be both painful and frightening to those with this disorder. Such experiences usually occur in situations in which the individual feels a lack of a meaningful relationship, nurturing, and support. These individuals may show worse performance in unstructured work or school situations. This lack of a full and enduring identity makes it difficult for the individual with borderline personality disorder to identify maladaptive patterns of behavior and can lead to repetitive patterns of troubled relationships.

Individuals with borderline personality disorder display impulsivity in at least two areas that are potentially self-damaging (Criterion 4). They may gamble, spend money irresponsibly, binge eat, abuse substances, engage in unsafe sex, or drive recklessly.

Individuals with this disorder display recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior (Criterion 5). Recurrent suicidal thoughts or behavior are often the reason that these individuals present for help. These self-destructive acts are usually precipitated by threats of separation or rejection or by expectations that the individual assume increased responsibility. Self-mutilative acts (e.g., cutting or burning) are very common and may occur during periods in which the individual is experiencing dissociative symptoms. These acts often bring relief by reaffirming the individual’s ability to feel or by expiating the individual’s sense of being evil.

Individuals with borderline personality disorder may display affective instability that is due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days) (Criterion 6). The basic dysphoric mood of those with borderline personality disorder is often disrupted by periods of anger, panic, or despair and is rarely relieved by periods of well-being or satisfaction. These episodes may reflect the individual’s extreme reactivity to interpersonal stresses.

Individuals with borderline personality disorder may be troubled by chronic feelings of emptiness, which can co-occur with painful feelings of aloneness (Criterion 7). Easily bored, they may frequently seek excitement to avoid their feelings of emptiness.

Individuals with this disorder frequently express inappropriate, intense anger or have difficulty controlling their anger (Criterion 8). They may display extreme sarcasm, enduring bitterness, or verbal outbursts. The anger is often elicited when a caregiver or lover is seen as neglectful, withholding, uncaring, or abandoning. Such expressions of anger are often followed by shame and guilt and contribute to the feeling they have of being evil.

During periods of extreme stress, transient paranoid ideation or dissociative symptoms (e.g., depersonalization) may occur (Criterion 9), but these are generally of insufficient severity or duration to warrant an additional diagnosis. These episodes occur most frequently in response to a real or imagined abandonment. Symptoms tend to be transient, lasting minutes or hours. The real or perceived return of the caregiver's nurturance may result in a remission of symptoms.

Associated Features

Individuals with borderline personality disorder may have a pattern of undermining themselves at the moment a goal is about to be realized (e.g., dropping out of school just before graduation; regressing severely after a discussion of how well therapy is going; destroying a good relationship just when it is clear that the relationship could last). Some individuals develop psychotic-like symptoms (e.g., hallucinations, body-image distortions, ideas of reference, hypnagogic phenomena) during times of stress. Individuals with this disorder may feel more secure with transitional objects (i.e., a pet or inanimate possession) than in interpersonal relationships. Premature death from suicide may occur in individuals with borderline personality disorder, especially in those with co-occurring depressive disorders or substance use disorders. However, deaths from other causes. such as accidents or illness, are more than twice as common as deaths by suicide in individuals with borderline personality disorder. Physical handicaps may result from self-inflicted abuse behaviors or failed suicide attempts. Recurrent job losses, interrupted education, and separation or divorce are

common. Physical and sexual abuse, neglect, hostile conflict, and early parental loss are more common in the childhood histories of those with borderline personality disorder.

Prevalence

The estimated prevalence of borderline personality disorder based on a probability subsample from Part II of the National Comorbidity Survey Replication was 1.4%. The prevalence of borderline personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions data was 5.9%. A review of seven epidemiological studies (six in

the United States) found a median prevalence of 2.7%. The prevalence of borderline personality disorder is about 6% in primary care settings, about 10% among individuals seen in outpatient mental health clinics, and about 20% among psychiatric inpatients.

Development and Course

Borderline personality disorder has typically been thought of as an adult-onset disorder. However, it has been found in treatment settings that symptoms in adolescents as young as age 12 or 13 years can meet full criteria for the disorder. It is not yet known what percentage of adults first entering treatment actually have such an early onset of borderline personality disorder.

Borderline personality disorder has long been thought of as a disorder with a poor symptomatic course, which tended to lessen in severity as those with borderline personality disorder entered their 30s and 40s. However, prospective follow-up studies have found that stable remissions of 1–8 years are very common. Impulsive symptoms of borderline personality disorder remit the most rapidly, while affective symptoms remit at a substantially slower rate. In contrast, recovery from borderline personality disorder (i.e., concurrent symptomatic remission and good psychosocial functioning) is more difficult to achieve and less stable over time. Lack of

recovery is associated with supporting oneself on disability benefits and suffering from poor physical health.

Risk and Prognostic Factors

Environmental. Borderline personality disorder has also been found to be associated with high rates of various forms of reported childhood abuse and emotional neglect. However, reported rates of sexual abuse are higher in inpatients than in outpatients with this disorder, suggesting that a history of sexual abuse is as much a risk factor for severity of borderline psychopathology as it is for the disorder itself. In addition, an empirically based consensus has arisen that suggests that a childhood history of reported sexual abuse is neither necessary nor sufficient for the development of borderline personality disorder.

Genetic and physiological. Borderline personality disorder is about five times more common among first-degree biological relatives of those with the disorder than in the general population. There is also an increased familial risk for substance use disorders, anxiety disorders, antisocial personality disorder, and depressive or bipolar disorders.

Culture-Related Diagnostic Issues

The pattern of behavior seen in borderline personality disorder has been identified in many settings around the world. Sociocultural contexts characterized by social demands that evoke attempts at self-affirmation and acceptance by others, ambiguous or conflictual relationships with authority figures, or marked uncertainties in adaptation can foster impulsivity, emotional instability, explosive or aggressive behaviors, and dissociative experiences that are associated with borderline personality disorder or with transient and contextual reactions to those environments that can be confused with borderline personality disorder. Given that psychodynamic, cognitive, behavioral, and mindfulness aspects of models of mind and self vary cross-culturally, symptoms or traits that suggest the presence of borderline personality disorder (e.g., number of sexual partners, shifting between relationships, substance use) must be evaluated in light of cultural norms to make a valid diagnosis.

Sex- and Gender-Related Diagnostic Issues

While borderline personality disorder is more common among women than men in clinical samples, community samples demonstrate no difference in prevalence between men and women. This discrepancy may reflect a higher degree of help-seeking among women,

leading them to clinical settings. Clinical characteristics of men and women with borderline personality disorder appear to be similar, with potentially a higher degree of externalizing behaviors in boys and men and internalizing behaviors in girls and women.

Association With Suicidal Thoughts or Behavior

ln a longitudinal study, impulsive and antisocial behaviors of individuals with borderline personality disorder were associated with increased suicide risk. In a sample of hospitalized patients with borderline personality disorder followed prospectively for 24 years, around 6% died by suicide, compared with 1.4% in a comparison sample of individuals with personality disorders other than borderline personality disorder. A study of individuals with borderline personality disorder followed for 10 years found that recurrent suicidal behavior was a defining characteristic of borderline personality disorder, associated with declining rates of suicide attempts from 79% to 13% over time.

Differential Diagnosis

Depressive and bipolar disorders. Borderline personality disorder often co-occurs with depressive or bipolar disorders, and when criteria for both are met, both should be diagnosed. Because the cross-sectional presentation of borderline personality disorder can be mimicked by an episode of depressive or bipolar disorder, the clinician should avoid giving an additional diagnosis of borderline personality disorder based only on cross-sectional presentation without having documented that the pattern of behavior had an early onset and a long-standing course.

Separation anxiety disorder in adults.

Separation anxiety disorder and borderline personality disorder are characterized by fear of abandonment by loved ones, but problems in identity, self-direction, interpersonal functioning, and impulsivity are additionally central to borderline personality disorder.

Other personality disorders. Other personality disorders may be confused with borderline personality disorder because they have certain features in common. It is therefore important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality features that meet criteria for one or more personality disorders in addition to borderline personality disorder, all can be diagnosed. Although histrionic personality disorder can also be characterized by attention seeking, manipulative behavior, and rapidly shifting emotions, borderline personality disorder is distinguished by self-destructiveness, angry disruptions in close relationships, and chronic feelings of deep emptiness and loneliness. Paranoid ideas or illusions may be present in both borderline personality disorder and schizotypal personality disorder, but these symptoms are more transient, interpersonally reactive, and responsive to external structuring in borderline personality disorder. Although paranoid personality disorder and narcissistic personality disorder may also be characterized by an angry reaction to minor stimuli, the relative stability of self-image, as well as the relative lack of physical self-destructiveness, repetitive impulsivity, and profound abandonment concerns, distinguishes these disorders from borderline personality disorder. Although antisocial personality disorder and borderline personality disorder are both characterized by manipulative behavior, individuals with antisocial personality disorder are manipulative to gain profit, power, or some other material gratification, whereas the goal in borderline personality disorder is directed more toward gaining the concern of caretakers. Both dependent personality disorder and borderline personality disorder are characterized by fear of abandonment; however, the individual with borderline personality disorder reacts to abandonment with feelings of emotional emptiness, rage, and demands, whereas the individual with dependent personality disorder reacts with increasing appeasement and submissiveness and urgently seeks a replacement relationship to provide caregiving and support. Borderline personality disorder can

further be distinguished from dependent personality disorder by the typical pattern of unstable and intense relationships.

Personality change due to another medical condition. Borderline personality disorder must be distinguished from personality change due to another medical condition, in which the traits that emerge are a direct physiological consequence of another medical condition.

Substance use disorders. Borderline personality disorder must also be distinguished from symptoms that may develop in association with persistent substance use.

Identity problems. Borderline personality disorder should be distinguished from an identity problem, which is reserved for identity concerns related to a developmental phase (e.g., adolescence) and does not qualify as a mental disorder. Adolescents and young adults with identity problems (especially when accompanied by substance use) may transiently display behaviors that misleadingly give the impression of borderline personality disorder. Such situations are characterized by emotional instability, existential dilemmas, uncertainty, anxiety-provoking choices, conflicts about sexual orientation, and competing social pressures to decide on careers.

Comorbidity

Common co-occurring disorders include depressive and bipolar disorders, substance use disorders, anxiety disorders (particularly panic disorder and social anxiety disorder), eating disorders (notably bulimia nervosa and binge-eating disorder), posttraumatic stress disorder, and attention-deficit/hyperactivity disorder. Borderline personality disorder also frequently co-occurs with the other personality disorders.

Histrionic Personality Disorder

Diagnostic Criteria	**F60.4**

A pervasive pattern of excessive emotionality and attention seeking, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Is uncomfortable in situations in which he or she is not the center of attention.
2. Interaction with others is often characterized by inappropriate sexually seductive or provocative behavior.
3. Displays rapidly shifting and shallow expression of emotions.
4. Consistently uses physical appearance to draw attention to self.
5. Has a style of speech that is excessively impressionistic and lacking in detail.
6. Shows self-dramatization, theatricality, and exaggerated expression of emotion.
7. Is suggestible (i.e., easily influenced by others or circumstances).
8. Considers relationships to be more intimate than they actually are.

Diagnostic Features

The essential feature of histrionic personality disorder is pervasive and excessive emotionality and attention-seeking behavior. This pattern begins by early adulthood and is present in a variety of contexts.

Individuals with histrionic personality disorder are uncomfortable or feel unappreciated when they are not the center of attention (Criterion 1). Often lively and dramatic, they tend to draw attention to themselves and may initially charm new acquaintances by their enthusiasm, apparent openness, or flirtatiousness. These qualities wear thin, however, as these individuals continually demand to be the center of attention. They commandeer the

role of “the life of the party.” If they are not the center of attention, they may do something dramatic (e.g., make up stories, create a scene) to draw the focus of attention to themselves. This need is often apparent in their behavior with a clinician (e.g., being flattering, bringing gifts, providing dramatic descriptions of physical and psychological symptoms that are replaced by new symptoms each visit).

The appearance and behavior of individuals with this disorder are often inappropriately sexually provocative or seductive (Criterion 2). This behavior not only is directed toward persons in whom the individual has a sexual or romantic interest but also occurs in a wide variety of social, occupational, and professional relationships beyond what is appropriate for the social context. Emotional expression may be shallow and rapidly shifting (Criterion 3). Individuals with this disorder consistently use physical appearance to draw attention to themselves (Criterion 4). They are overly concerned with impressing others by their appearance and expend an excessive amount of time, energy, and money on clothes and grooming. They may “fish for compliments” regarding appearance and may be easily and excessively upset by a critical comment about how they look or by a photograph that they regard as unflattering.

These individuals have a style of speech that is excessively impressionistic and lacking in detail (Criterion 5). Strong opinions are expressed with dramatic flair, but underlying rationales are usually vague and diffuse, without supporting facts and details. For example, an individual with histrionic personality disorder may comment that a certain individual is a wonderful human being, yet be unable to provide any specific examples of good qualities to support this opinion. Individuals with this disorder are characterized by self-dramatization, theatricality, and an exaggerated expression of emotion (Criterion 6). They may embarrass friends and acquaintances by an excessive public display of emotions (e.g., embracing casual acquaintances with excessive ardor, sobbing uncontrollably on minor sentimental occasions, having temper tantrums). However, their emotions often seem to be turned on and off too quickly to be deeply felt, which may lead others to accuse the individual of faking these feelings.

Individuals with histrionic personality disorder have a high degree of suggestibility (Criterion 7). Their opinions and feelings are easily influenced by others and by current fads. They may be overly trusting,

especially of strong authority figures whom they see as magically solving their problems. They have a tendency to play hunches and to adopt convictions quickly. Individuals with this disorder often consider relationships more intimate than they actually are, describing almost every acquaintance as “my dear, dear friend” or referring to physicians met only once or twice under professional circumstances by their first names (Criterion 8).

Associated Features

Impairment in general tends to be lower in histrionic personality disorder than in many other personality disorders. However, the impairment most associated with histrionic personality disorder appears to be interpersonal in nature. Individuals with histrionic personality disorder have an interpersonal style characterized by social dominance, which can span a spectrum of behaviors that include a “warmer dominance” that can be intrusive in nature (e.g., need to be center of attention; exhibitionistic) to a “colder dominance” that can include arrogant, controlling, and aggressive behaviors. Romantic relationships appear to be particularly impaired, with evidence suggesting that individuals with histrionic personality disorder symptoms are more likely to get divorced or never get married. Individuals with histrionic personality disorder may have difficulty achieving emotional intimacy in romantic or sexual relationships. Individuals with this disorder often have impaired relationships with same-sex friends because their sexually provocative interpersonal style may seem a threat to their friends’ relationships. These individuals may also alienate friends with demands for constant attention. They often become depressed and

upset when they are not the center of attention. They may crave novelty, stimulation, and excitement and have a tendency to become bored with their usual routine. These individuals are often intolerant of, or frustrated by, situations that involve delayed gratification, and their actions are often directed at obtaining immediate satisfaction. Although they often initiate a job or project with great enthusiasm, their interest may lag quickly. Longer-

term relationships may be neglected to make way for the excitement of new relationships.

Prevalence

The estimated prevalence of histrionic personality disorder based on a probability subsample from Part II of the National Comorbidity Survey Replication was 0.0%.The prevalence of histrionic personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions data was 1.8%. A review of five epidemiological studies (four in the United States) found a median prevalence of 0.9%.

Culture-Related Diagnostic Issues

Norms for interpersonal behavior, personal appearance, and emotional expressiveness vary widely across cultures, genders, and age groups. Before considering the various traits (e.g., emotionality, seductiveness, dramatic interpersonal style, novelty seeking, sociability, charm, impressionability, a tendency to somatization) to be evidence of histrionic personality disorder, it is important to evaluate whether they cause clinically significant impairment or distress. The presence of histrionic personality disorder should be distinguished from reactive and contextual expression of these traits, arising in response to socialization pressures in competitive peer groups, including the “need to be liked,” that do not represent pervasive and enduring traits consistent with a personality disorder.

Sex- and Gender-Related Diagnostic Issues

In clinical settings, this disorder has been diagnosed more frequently in females; however, the gender ratio is not significantly different from the gender ratio of females within the respective clinical setting. In contrast, some studies using structured assessments report similar prevalence rates among males and females.

Association With Suicidal Thoughts or Behavior

The actual risk of suicide is not known, but clinical experience suggests that individuals with this disorder may be at increased risk for suicidal gestures

and threats.

Differential Diagnosis

Other personality disorders and personality traits. Other personality disorders may be confused with histrionic personality disorder because they have certain features in common. It is therefore important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality features that meet criteria for one or more personality disorders in addition to histrionic personality disorder, all can be diagnosed. Although borderline personality disorder can also be characterized by attention seeking, manipulative behavior, and rapidly shifting emotions, it is distinguished by self-destructiveness, angry disruptions in close relationships, and chronic feelings of deep emptiness and identity disturbance. Individuals with antisocial personality disorder and histrionic personality disorder share a tendency to be impulsive, superficial, excitement seeking, reckless, seductive, and manipulative, but persons with histrionic personality disorder tend to be more exaggerated in their emotions and do not characteristically engage in antisocial behaviors. Individuals with histrionic personality disorder are manipulative to gain nurturance, whereas those with antisocial personality disorder are manipu

lative to gain profit, power, or some other material gratification. Although individuals with narcissistic personality disorder also crave attention from others, they usually want praise for their “superiority,” whereas individuals with histrionic personality disorder are willing to be viewed as fragile or dependent if this is instrumental in getting attention. Individuals with narcissistic personality disorder may exaggerate the intimacy of their relationships with other people, but they are more apt to emphasize the “VIP” status or wealth of their friends. In dependent personality disorder, the individual is excessively dependent on others for praise and guidance, but is without the flamboyant, exaggerated, emotional features of individuals with histrionic personality disorder.

Many individuals may display histrionic personality traits. Only when these traits are inflexible, maladaptive, and persisting and cause significant functional impairment or subjective distress do they constitute histrionic personality disorder.

Personality change due to another medical condition. Histrionic personality disorder must be distinguished from personality change due to another medical condition, in which the traits that emerge are a direct physiological consequence of another medical condition.

Substance use disorders. The disorder must also be distinguished from symptoms that may develop in association with persistent substance use.

Comorbidity

Histrionic personality disorder has been associated with higher rates of borderline, narcissistic, paranoid, dependent, and antisocial personality disorders; alcohol and other substance use and misuse; as well as aggression and violence. Histrionic personality disorder is also thought to be related to somatic symptom disorder, functional neurological symptom disorder (conversion disorder), and major depressive disorder.

Narcissistic Personality Disorder

Diagnostic Criteria F60.81

A pervasive pattern of grandiosity (in fantasy or behavior), need for admiration, and lack of empathy, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Has a grandiose sense of self-importance (e.g., exaggerates achievements and talents, expects to be recognized as superior without commensurate achievements).
2. Is preoccupied with fantasies of unlimited success, power, brilliance, beauty, or ideal love.

3. Believes that he or she is “special” and unique and can only be understood by, or should associate with, other special or high-status people (or institutions).
4. Requires excessive admiration.
5. Has a sense of entitlement (i.e., unreasonable expectations of especially favorable treatment or automatic compliance with his or her expectations).
6. Is interpersonally exploitative (i.e., takes advantage of others to achieve his or her own ends).
7. Lacks empathy: is unwilling to recognize or identify with the feelings and needs of others.
8. Is often envious of others or believes that others are envious of him or her.
9. Shows arrogant, haughty behaviors or attitudes.

Diagnostic Features

The essential feature of narcissistic personality disorder is a pervasive pattern of grandiosity, need for admiration, and lack of empathy that begins by early adulthood and is present in a variety of contexts.

Individuals with this disorder have a grandiose sense of self-importance, which may be manifest as an exaggerated or unrealistic sense of superiority, value, or capability (Criterion 1). They tend to overestimate their abilities and amplify their accomplishments, often appearing boastful and pretentious. They may blithely assume that others attribute the same value to their efforts and may be surprised when the praise they expect and feel they deserve is not forthcoming. Often implicit in the inflated judgments of their own accomplishments is an underestimation or devaluation of the contributions of others. Individuals with narcissistic personality disorder are often preoccupied with fantasies of unlimited success, power, brilliance, beauty, or ideal love (Criterion 2). They may ruminate about “long overdue” admiration and privilege and compare themselves favorably with famous or privileged people.

Individuals with narcissistic personality disorder believe that they are special or unique and expect others to recognize them as such (Criterion 3). They can be surprised or even devastated when the recognition of acclaim they expect and feel they deserve from others is not forthcoming. They may feel that they can only be understood by, and should only associate with, people of high status and may attribute “unique,” “perfect,” or “gifted” qualities to those with whom they associate. Individuals with this disorder believe that their needs are special and beyond the ken of ordinary people. Their own self-esteem is enhanced (i.e., “mirrored”) by the idealized value that they assign to those with whom they associate. They are likely to insist on having only the “top” person (doctor, lawyer, hairdresser, instructor) or being affiliated with the “best” institutions but may devalue the credentials of those who disappoint them.

Individuals with this disorder generally require excessive admiration (Criterion 4). Their self-esteem is almost invariably very fragile, and their struggle with severe internal self-doubt, self-criticism, and emptiness results in their need to actively seek others’ admiration. They may be preoccupied with how well they are doing and how favorably they are regarded by others. They may expect their arrival to be greeted with great fanfare and are astonished if others do not covet their possessions. They may constantly fish for compliments, often with great charm.

A sense of entitlement, which is rooted in their distorted sense of self-worth, is evident in these individuals’ unreasonable expectation of especially favorable treatment (Criterion 5). They expect to be catered to and are puzzled or furious when this does not happen. For example, they may assume that they do not have to wait in line and that their priorities are so important that others should defer to them, and then get irritated when others fail to assist “in their very important work.” They expect to be given whatever they want or feel they need, no matter what it might mean to others. For example, these individuals may expect great dedication from others and may overwork them without regard for the impact on their lives. This sense of entitlement, combined with a lack of understanding and sensitivity to the wants and needs of others, may result in the conscious or unwitting exploitation of others (Criterion 6). They tend to form friendships or romantic relationships only if the other person seems likely to advance their purposes or otherwise enhance their self-esteem. They often usurp

special privileges and extra resources that they believe they deserve. Some individuals with narcissistic personality disorder intentionally and purposefully take advantage of others emotionally, socially, intellectually, or financially for their own purposes and gains.

Individuals with narcissistic personality disorder generally have a lack of empathy and are unwilling to recognize or identify with the desires, subjective experiences, and feelings of others (Criterion 7). They tend to have some degree of cognitive empathy

(understanding another person's perspective on an intellectual level) but lack emotional empathy (directly feeling the emotions that another person is feeling). These individuals may be oblivious to the hurt their remarks may inflict (e.g., exuberantly telling a former lover that "I am now in the relationship of a lifetime!"; boasting of health in front of someone who is sick). When recognized, the needs, desires, or feelings of others are likely to be viewed disparagingly as signs of weakness or vulnerability. Those who relate to individuals with narcissistic personality disorder typically find an emotional coldness and lack of reciprocal interest.

These individuals are often envious of others or believe that others are envious of them (Criterion 8). They may begrudge others their successes or possessions, feeling that they better deserve those achievements, admiration, or privileges. They may harshly devalue the contributions of others, particularly when those individuals have received acknowledgment or praise for their accomplishments. Arrogant, haughty behaviors characterize these individuals; they often display snobbish, disdainful, or patronizing attitudes (Criterion 9).

Associated Features

Vulnerability in self-esteem makes individuals with narcissistic personality disorder very sensitive to criticism or defeat. Although they may not show it outwardly, such experiences may leave them feeling ashamed, humiliated, degraded, hollow, and empty. They may react with disdain, rage, or defiant counterattack. However, such experiences can also lead to social

withdrawal or an appearance of humility that may mask and protect the grandiosity. Interpersonal relations are typically impaired because of problems related to self-preoccupation, entitlement, need for admiration, and relative disregard for the sensitivities of others.

Individuals with narcissistic personality disorder can be competent and high functioning with professional and social success, while others can have various levels of functional impairment. Professional capability combined with self-control, stoicism, and interpersonal distancing with minimal self-disclosure can support sustained life engagement and even enable marriage and social affiliations. Sometimes ambition and temporary confidence lead to high achievements, but performance can be disrupted because of fluctuating self-confidence and intolerance of criticism or defeat. Some individuals with narcissistic personality disorder have very low vocational functioning, reflecting an unwillingness to take a risk in competitive or other situations in which failure or defeat can be possible.

Low self-esteem with inferiority, vulnerability, and sustained feelings of shame, envy, and humiliation accompanied by self-criticism and insecurity can make individuals with narcissistic personality disorder susceptible to social withdrawal, emptiness, and depressed mood. High perfectionist standards are often associated with significant fear of exposure to imperfection, failure, and overwhelming emotions.

Prevalence

The estimated prevalence of narcissistic personality disorder based on a probability subsample from Part II of the National Comorbidity Survey Replication was 0.0%.The prevalence of narcissistic personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions data was 6.2%. A review of five epidemiological studies (four in the United States) found a median prevalence of 1.6%.

Development and Course

Narcissistic traits may be particularly common in adolescents but do not necessarily indicate that the individual will develop narcissistic personality disorder in adulthood. Predominant narcissistic traits or manifestations of

the full disorder may first come to clinical attention or be exacerbated in the context of unexpected or extremely challenging life

experiences or crises, such as bankruptcies, demotions or loss of work, or divorces. In addition, individuals with narcissistic personality disorder may have specific difficulties adjusting to the onset of physical and occupational limitations that are inherent in the aging process. However, life experiences, such as new durable relationships, real successful achievements, and tolerable disappointments and setbacks, can all be corrective and contribute to changes and improvements in individuals with this disorder.

Culture-Related Diagnostic Issues

Narcissistic traits may be elevated in sociocultural contexts that emphasize individualism and personal autonomy over collectivistic goals. Compared with collectivistic contexts, in individualistic contexts, narcissistic traits may warrant less clinical attention or less frequently lead to social impairment.

Sex- and Gender-Related Diagnostic Issues

Among adults age 18 and older diagnosed with narcissistic personality disorder, 50%–75% are men. Gender differences in adults with this disorder include stronger reactivity in response to stress and compromised empathic processing in men as opposed to self-focus and withdrawal in women. Culturally based gender patterns and expectations may also contribute to gender differences in narcissistic personality disorder traits and patterns.

Association With Suicidal Thoughts or Behavior

In the context of severe stress, and given the perfectionism often associated with narcissistic personality disorder, exposure to imperfection, failure, and overwhelming emotions can evoke suicidal ideation. Suicide attempts in individuals with narcissistic personality disorder tend to be less impulsive and are characterized by higher lethality compared with suicide attempts by individuals with other personality disorders.

Differential Diagnosis

Other personality disorders and personality traits. Other personality disorders may be confused with narcissistic personality disorder because they have certain features in common. It is, therefore, important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality features that meet criteria for one or more personality disorders in addition to narcissistic personality disorder, all can be diagnosed. The most useful feature in discriminating narcissistic personality disorder from histrionic, antisocial, and borderline personality disorders, in which the interactive styles are coquettish, callous, and needy, respectively, is the grandiosity characteristic of narcissistic personality disorder. The relative stability of self-image and self-control as well as the relative lack of self-destructiveness, impulsivity, separation insecurity, and emotional hyperreactivity also help distinguish narcissistic personality disorder from borderline personality disorder.

Excessive pride in achievements, a relative lack of emotional display, and ignorance of or disdain for others' sensitivities help distinguish narcissistic personality disorder from histrionic personality disorder. Although individuals with borderline, histrionic, and narcissistic personality disorders may require much attention, those with narcissistic personality disorder specifically need that attention to be admiring. Individuals with antisocial and narcissistic personality disorders share a tendency to be tough-minded, glib, superficial, exploitative, and unempathic. However, narcissistic personality disorder does not necessarily include characteristics of impulsive aggressivity and deceitfulness. In addition, individuals with antisocial personality disorder may be more indifferent and less sensitive to others' reactions or criticism, and individuals with narcissistic personality disorder usually lack the history of conduct disorder in childhood or criminal behavior in adulthood.

In both narcissistic personality disorder and obsessive-compulsive personality disorder, the individual may profess a commitment to perfectionism and believe that others cannot do things as well. However,

while those with obsessive-compulsive personality disorder tend to be more immersed in perfectionism related to order and rigidity, individuals with narcissistic personality disorder tend to set high perfectionistic standards, especially for appearance and performance, and to be critically concerned if they are not measuring up.

Suspiciousness and social withdrawal usually distinguish those with schizotypal, avoidant, or paranoid personality disorder from those with narcissistic personality disorder. When these qualities are present in individuals with narcissistic personality disorder, they derive primarily from shame and fear of failure, or fear of having imperfections or flaws revealed.

Many highly successful individuals display personality traits that might be considered narcissistic. Only when these traits are inflexible, maladaptive, and persisting and cause significant functional impairment or subjective distress do they constitute narcissistic personality disorder.

Mania or hypomania. Grandiosity may emerge as part of manic or hypomanic episodes, but the association with mood change or functional impairments helps distinguish these episodes from narcissistic personality disorder.

Substance use disorders. Narcissistic personality disorder must also be distinguished from symptoms that may develop in association with persistent substance use.

Persistent depressive disorder. Experiences that threaten self-esteem can evoke a deep sense of inferiority and sustained feelings of shame, envy, self-criticism, and insecurity in individuals with narcissistic personality disorder that can result in persistent negative feelings resembling those seen in persistent depressive disorder. If criteria are also met for persistent depressive disorder, both conditions can be diagnosed.

Comorbidity

Narcissistic personality disorder is associated with depressive disorders (persistent depressive disorder and major depressive disorder), anorexia nervosa, and substance use disorders (especially related to cocaine). Histrionic, borderline, antisocial, and paranoid personality disorders may also be associated with narcissistic personality disorder.

Cluster C Personality Disorders

Avoidant Personality Disorder

Diagnostic Criteria **F60.6**

A pervasive pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation, beginning by early adulthood and present in a variety of contexts, as indicated by four (or more) of the following:

1. Avoids occupational activities that involve significant interpersonal contact because of fears of criticism, disapproval, or rejection.
2. Is unwilling to get involved with people unless certain of being liked.

3. Shows restraint within intimate relationships because of the fear of being shamed or ridiculed.
4. Is preoccupied with being criticized or rejected in social situations.
5. Is inhibited in new interpersonal situations because of feelings of inadequacy.
6. Views self as socially inept, personally unappealing, or inferior to others.
7. Is unusually reluctant to take personal risks or to engage in any new activities because they may prove embarrassing.

Diagnostic Features

The essential feature of avoidant personality disorder is a pervasive pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative

evaluation that begins by early adulthood and is present in a variety of contexts.

Individuals with avoidant personality disorder avoid work activities that involve significant interpersonal contact because of fears of criticism, disapproval, or rejection (Criterion 1). Offers of job promotions may be declined because failure to manage the new responsibilities might result in criticism from coworkers. These individuals avoid making new friends unless they are certain they will be liked and accepted without criticism (Criterion 2). Until they pass stringent tests proving the contrary, other people are assumed to be critical and disapproving. Individuals with this disorder are highly avoidant of group activities. Interpersonal intimacy is often difficult for these individuals, although they are able to establish intimate relationships when there is assurance of uncritical acceptance. They may act with restraint, be reluctant to talk about themselves, and withhold intimate feelings for fear of being exposed, ridiculed, or shamed (Criterion 3).

Because individuals with this disorder are preoccupied with being criticized or rejected in social situations, they may have a markedly low threshold for detecting such reactions (Criterion 4). If someone is even slightly disapproving or critical, they may feel extremely hurt. They tend to be shy, quiet, inhibited, and “invisible” because of the fear that any attention would be critical or rejecting. They expect that no matter what they say, others will see it as “wrong,” and so they may say nothing at all. They react strongly to subtle cues that are suggestive of mockery or derision, and may misinterpret a neutral gesture or statement as critical or rejecting. Despite their longing to be active participants in social life, they fear placing their psychological welfare in the hands of others. Individuals with avoidant personality disorder are inhibited in new interpersonal situations because they feel inadequate and have low self-esteem (Criterion 5). These individuals believe themselves to be socially inept, personally unappealing, or inferior to others (Criterion 6). Doubts concerning social competence and personal appeal may be most intense for some individuals in settings involving interactions with strangers. But many others report more difficulties with repeated interaction, when sharing of personal information would normally occur, thus, in the individual’s perception, increasing the chances that their inferiority would be revealed and that they

would be rejected. When commencing a new ongoing social or occupational commitment requiring repeated interpersonal interaction, individuals may over weeks or months develop a growing conviction that others or colleagues view them as inferior or lacking worth, resulting in intolerable distress or anxiety that prompts resignation. Thus, a history of repeated job changes may be present. Individuals with this disorder are unusually reluctant to take personal risks or to engage in any new activities because these may prove embarrassing (Criterion 7). They are prone to exaggerate the potential dangers of ordinary situations, and a restricted lifestyle may result from their need for certainty and security.

Associated Features

Individuals with avoidant personality disorder often vigilantly appraise the movements and expressions of those with whom they come into contact. They are likely to

misinterpret social responses as critical, which in turn confirms their self-doubts. They are described by others as being “shy,” “timid,” “lonely,” and “isolated.” The major problems associated with this disorder occur in social and occupational functioning. The low self-esteem and hypersensitivity to rejection are associated with restricted interpersonal contacts. These individuals may become relatively isolated and usually do not have a large social support network that can help them weather crises. They desire affection and acceptance and may fantasize about idealized relationships with others. Avoidant behaviors can also adversely affect occupational functioning because these individuals try to avoid the types of social situations that may be important for meeting the basic demands of the job or for advancement.

Individuals with avoidant personality disorder have been reported as having insecure attachment styles characterized by a desire for emotional attachment (which may include a preoccupation with previous and current relationships), but their fears that others may not value them or may hurt them may lead them to respond with passivity, anger, or fear. These

attachment patterns have been referred to variously as “preoccupied” or “fearful” depending on the model employed by researchers.

Prevalence

The estimated prevalence of avoidant personality disorder based on a probability subsample from Part II of the National Comorbidity Survey Replication was 5.2%. The prevalence of avoidant personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions was 2.4%. A review of six epidemiological studies (four in the United States) found a median prevalence of 2.1%.

Development and Course

The avoidant behavior often starts in infancy or childhood with shyness, isolation, and fear of strangers and new situations. Although shyness in childhood is a common precursor of avoidant personality disorder, in most individuals it tends to gradually dissipate as they get older. In contrast, individuals who go on to develop avoidant personality disorder may become increasingly shy and avoidant during adolescence and early adulthood, when social relationships with new people become especially important. There is some evidence that in adults, avoidant personality disorder tends to become less evident or to remit with age; the prevalence in adults older than 65 years has been estimated at 0.8%. This diagnosis should be used with great caution in children and adolescents, for whom shy and avoidant behavior may be developmentally appropriate.

Culture-Related Diagnostic Issues

There may be variation in the degree to which different cultural and ethnic groups regard diffidence and avoidance as appropriate. Moreover, avoidant behavior may be the result of problems in acculturation following migration. In some sociocultural contexts, marked avoidance might occur following social embarrassment (“loss of face”) or failure to meet major life goals rather than temperamental shyness. In these settings, the goal of avoidance includes deliberate minimization of social interactions in order to preserve social harmony or prevent public offense.

Sex- and Gender-Related Diagnostic Issues

Avoidant personality disorder appears to be more common in women than in men in community surveys. This gender difference in prevalence is small but consistently found in large population-based samples.

Differential Diagnosis

Social anxiety disorder. There appears to be a great deal of overlap between avoidant personality disorder and social anxiety disorder. It has been suggested that they may represent different manifestations of similar underlying problems, or avoidant personality disorder may be a more severe form of social anxiety disorder. However, differences have also been described, especially in relation to self-concept (such as self-esteem and the sense of inferiority in avoidant personality disorder); the latter is indirect evidence as it shows that negative self-concept in social anxiety disorder may be unstable and thus less pervasive and entrenched than in avoidant personality disorder. Additionally, studies have shown that avoidant personality disorder frequently occurs in the absence of social anxiety disorder, and some separate risk factors have been identified, providing support for retaining two separate diagnostic categories.

Agoraphobia. Avoidance characterizes both avoidant personality disorder and agoraphobia, and they often co-occur. They can be distinguished by the motivation for the avoidance (e.g., fear of panic or physical harm in agoraphobia).

Other personality disorders and personality traits. Other personality disorders may be confused with avoidant personality disorder because they have certain features in common. It is, therefore, important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality features that meet criteria for one or more personality disorders in addition to avoidant personality disorder, all can be diagnosed. Both avoidant personality disorder and dependent personality disorder are characterized by feelings of inadequacy, hypersensitivity to criticism, and a need for reassurance. Similar behaviors

(e.g., unassertiveness) and attributes (e.g., low self-esteem and low self-confidence) may be observed in both dependent personality disorder and avoidant personality disorder, although other behaviors are notably divergent, such as avoidance of social proximity in avoidant personality disorder but proximity-seeking in dependent personality disorder. The motivations behind similar behaviors may be quite different. For example, the unassertiveness in avoidant personality disorder is described as more closely related to fears of being rejected or humiliated, whereas in dependent personality disorder it is motivated by the desire to avoid being left to fend for oneself. However, avoidant personality disorder and dependent personality disorder may be particularly likely to co-occur. Like avoidant personality disorder, schizoid personality disorder and schizotypal personality disorder are characterized by social isolation. However, individuals with avoidant personality disorder want to have relationships with others and feel their loneliness deeply, whereas those with schizoid or schizotypal personality disorder may be content with and even prefer their social isolation. Paranoid personality disorder and avoidant personality disorder are both characterized by a reluctance to confide in others. However, in avoidant personality disorder, this reluctance is attributable more to a fear of humiliation or being found inadequate than to a fear of others' malicious intent.

Many individuals display avoidant personality traits. Only when these traits are inflexible, maladaptive, and persisting and cause significant functional impairment or subjective distress do they constitute avoidant personality disorder.

Personality change due to another medical condition. Avoidant personality disorder must be distinguished from personality change due to another medical condition, in which the traits that emerge are a direct physiological consequence of another medical condition.

Substance use disorders. Avoidant personality disorder must also be distinguished from symptoms that may develop in association with persistent substance use.

Comorbidity

Other disorders that are commonly diagnosed with avoidant personality disorder include depressive disorders and anxiety disorders, especially social anxiety disorder. Avoidant personality disorder also tends to be diagnosed with schizoid personality disorder. Avoidant personality disorder is associated with increased rates of substance use disorders at a similar rate to the generalized form of social anxiety disorder.

Dependent Personality Disorder

Diagnostic Criteria F60.7

A pervasive and excessive need to be taken care of that leads to submissive and clinging behavior and fears of separation, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Has difficulty making everyday decisions without an excessive amount of advice and reassurance from others.
2. Needs others to assume responsibility for most major areas of his or her life.
3. Has difficulty expressing disagreement with others because of fear of loss of support or approval. (**Note:** Do not include realistic fears of retribution.)
4. Has difficulty initiating projects or doing things on his or her own (because of a lack of self-confidence in judgment or abilities rather than a lack of motivation or energy).
5. Goes to excessive lengths to obtain nurturance and support from others, to the point of volunteering to do things that are unpleasant.
6. Feels uncomfortable or helpless when alone because of exaggerated fears of being unable to care for himself or herself.

7. Urgently seeks another relationship as a source of care and support when a close relationship ends.
8. Is unrealistically preoccupied with fears of being left to take care of himself or herself.

Diagnostic Features

The essential feature of dependent personality disorder is a pervasive and excessive need to be taken care of that leads to submissive and clinging behavior and fears of separation. This pattern begins by early adulthood and is present in a variety of contexts. The dependent and submissive behaviors are designed to elicit caregiving and arise from a self-perception of being unable to function adequately without the help of others.

Individuals with dependent personality disorder have great difficulty making everyday decisions (e.g., what color shirt to wear to work or whether to carry an umbrella) without an excessive amount of advice and reassurance from others (Criterion 1). These individuals tend to be passive and to allow other people (often a single other person) to take the initiative and assume responsibility for most major areas of their lives (Criterion 2). Adults with this disorder typically depend on a parent or spouse to decide where they should live, what kind of job they should have, and which neighbors to befriend. Adolescents with this disorder may allow their parent(s) to decide what they should wear, with whom they should associate, how they should spend their free time, and what school or college they should attend. This need for others to assume responsibility goes beyond age-appropriate and situation-appropriate requests for assistance from others (e.g., the specific needs of children, elderly persons, and handicapped persons). Dependent personality disorder may occur in an individual who has a serious medical condition or disability, but in such cases the difficulty in taking responsibility must go beyond what would normally be associated with that condition or disability.

Because they fear losing support or approval, individuals with dependent personality disorder often have difficulty expressing

disagreement with other individuals, especially those on whom they are dependent (Criterion 3). These individuals feel so unable to function alone that they will agree with things that they feel are wrong rather than risk losing the help of those to whom they look for guidance. They do not express anger toward others whose support and nurturance they need for fear of alienating them. If the individual's concerns regarding the consequences of expressing disagreement are realistic (e.g., realistic fears of retribution from an abusive spouse), the behavior should not be considered to be evidence of dependent personality disorder.

Individuals with this disorder have difficulty initiating projects or doing things independently (Criterion 4). They lack self-confidence and believe that they need help to begin and carry through tasks. They will wait for others to start things because they believe that as a rule others can do them better. These individuals are convinced that they are incapable of functioning independently and present themselves as inept and requiring constant assistance. They are, however, likely to function adequately if given the assurance that someone else is supervising and approving. There may be a fear of becoming or appearing to be more competent, because they may believe that this will lead to loss of support. Because they rely on others to handle their problems, they often do not learn the skills of independent living, thus perpetuating dependency.

Individuals with dependent personality disorder may go to excessive lengths to obtain nurturance and support from others, even to the point of volunteering for unpleasant tasks if such behavior will bring the care they need (Criterion 5). They are willing to submit to what others want, even if the demands are unreasonable. Their need to maintain an important bond will often result in imbalanced or distorted relationships. They may make extraordinary self-sacrifices or tolerate verbal, physical, or sexual abuse. (It should be noted that this behavior should be considered evidence of dependent personality disorder only when it can clearly be established that other options are available to the individual.) Individuals with this disorder feel uncomfortable or helpless when alone because of their exaggerated fears of being unable to care for themselves (Criterion 6).

When a close relationship ends (e.g., a breakup with a lover; the death of a caregiver), individuals with dependent personality disorder may urgently seek another relationship to provide the care and support they need

(Criterion 7). Their belief that they are unable to function in the absence of a close relationship motivates these individuals to become quickly and indiscriminately attached to another individual. Individuals with this disorder are often preoccupied with fears of being left to care for themselves (Criterion 8). They see themselves as so totally dependent on the advice and help of an important other person that they worry about losing the support of that person when there are no grounds to justify such fears. To be considered as evidence of this criterion, the fears must be excessive and unrealistic. For example, an elderly man with cancer who moves into his son's household for care is exhibiting dependent behavior that is appropriate given this person's life circumstances.

Associated Features

Individuals with dependent personality disorder are often characterized by pessimism and self-doubt and tend to belittle their abilities and assets. They take criticism and disapproval as proof of their worthlessness and lose faith in themselves. They may seek overprotection and dominance from others. Occupational functioning may be impaired if independent initiative is required. They may avoid positions of responsibility and become anxious when faced with decisions.

Prevalence

The estimated prevalence of dependent personality disorder based on a probability subsample from Part II of the National Comorbidity Survey Replication was 0.6%. The

prevalence of dependent personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions was 0.5%. A review of six epidemiological studies (four in the United States) found a median prevalence of 0.4%.

Development and Course

This diagnosis should be used with great caution, if at all, in children and adolescents, for whom dependent behavior may be developmentally appropriate.

Culture-Related Diagnostic Issues

The degree to which dependent behaviors are considered to be appropriate varies substantially across different age and sociocultural groups. Age and cultural factors need to be considered in evaluating the diagnostic threshold of each criterion. Dependent behavior should be considered characteristic of the disorder only when it is clearly in excess of the individual's cultural norms or reflects unrealistic concerns. An emphasis on passivity, politeness, and deferential treatment is characteristic of some societies and may be misinterpreted as traits of dependent personality disorder. Similarly, societies may differentially foster and discourage dependent behavior in males and females. Individuals with dependent personality disorder exhibit a pervasive inability to make decisions, continuous feelings of subjugation, lack of initiative, silence, and social distancing that are far in excess of usual cultural norms of politeness and purposeful passivity.

Sex- and Gender-Related Diagnostic Issues

In clinical and community settings, dependent personality disorder has been diagnosed more frequently in women compared with men.

Differential Diagnosis

Separation anxiety disorder in adults. Adults with separation anxiety disorder are typically overconcerned about their offspring, spouses, parents, and pets, and experience marked discomfort when separated from them. In contrast, individuals with dependent personality disorder feel uncomfortable or helpless when alone because of exaggerated fears of being unable to take care of themselves.

Other mental disorders and medical conditions. Dependent personality disorder must be distinguished from dependency arising as a consequence of other mental disorders (e.g., depressive disorders, panic disorder, agoraphobia) and as a result of other medical conditions.

Other personality disorders and personality traits. Other personality disorders may be confused with dependent personality disorder because they have certain features in common. It is therefore important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality features that meet criteria for one or more personality disorders in addition to dependent personality disorder, all can be diagnosed. Although many personality disorders are characterized by dependent features, dependent personality disorder can be distinguished by its predominantly submissive and clinging behavior and by the person's self-perception of not being able to function adequately without the help and support of others. Both dependent personality disorder and borderline personality disorder are characterized by fear of abandonment; however, the individual with borderline personality disorder reacts to abandonment with feelings of emotional emptiness, rage, and demands, whereas the individual with dependent personality disorder reacts with increasing appeasement and submissiveness and urgently seeks a replacement relationship to provide caregiving and support. Borderline personality disorder can further be distinguished from dependent

personality disorder by a typical pattern of unstable and intense relationships. Individuals with histrionic personality disorder, like those with dependent personality disorder, have a strong need for reassurance and approval and may appear childlike and clinging. However, unlike dependent personality disorder, which is characterized by self-effacing and docile behavior, histrionic personality disorder is characterized by gregarious flamboyance with active demands for attention. Moreover, individuals with histrionic personality disorder typically have less insight regarding their underlying dependency needs than do people with dependent personality disorder. Both dependent personality disorder and avoidant personality disorder are characterized by feelings of inadequacy, hypersensitivity to criticism, and a need for reassurance; however, individuals with avoidant personality disorder have such a strong fear of humiliation and rejection that they withdraw until they are certain they will be accepted. In contrast, individuals with dependent personality disorder have a pattern of seeking

and maintaining connections to important others, rather than avoiding and withdrawing from relationships.

Many individuals display dependent personality traits. Only when these traits are inflexible, maladaptive, and persisting and cause significant functional impairment or subjective distress do they constitute dependent personality disorder.

Personality change due to another medical condition. Dependent personality disorder must be distinguished from personality change due to another medical condition, in which the traits that emerge are a direct physiological consequence of another medical condition.

Substance use disorders. Dependent personality disorder must also be distinguished from symptoms that may develop in association with persistent substance use.

Comorbidity

There may be an increased risk of depressive disorders, anxiety disorders, and adjustment disorders. Dependent personality disorder often co-occurs with other personality disorders, especially borderline, avoidant, and histrionic personality disorders. Chronic physical illness or persistent separation anxiety disorder in childhood or adolescence may predispose the individual to the development of this disorder.

Obsessive-Compulsive Personality Disorder

Diagnostic Criteria F60.5

A pervasive pattern of preoccupation with orderliness, perfectionism, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency, beginning by early adulthood and present in a variety of contexts, as indicated by four (or more) of the following:

1. Is preoccupied with details, rules, lists, order, organization, or schedules to the extent that the major point of the activity is

lost.
2. Shows perfectionism that interferes with task completion (e.g., is unable to complete a project because his or her own overly strict standards are not met).
3. Is excessively devoted to work and productivity to the exclusion of leisure activities and friendships (not accounted for by obvious economic necessity).
4. Is overconscientious, scrupulous, and inflexible about matters of morality, ethics, or values (not accounted for by cultural or religious identification).

5. Is unable to discard worn-out or worthless objects even when they have no sentimental value.
6. Is reluctant to delegate tasks or to work with others unless they submit to exactly his or her way of doing things.
7. Adopts a miserly spending style toward both self and others; money is viewed as something to be hoarded for future catastrophes.
8. Shows rigidity and stubbornness.

Diagnostic Features

The essential feature of obsessive-compulsive personality disorder is a preoccupation with orderliness, perfectionism, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency. This pattern begins by early adulthood and is present in a variety of contexts.

Individuals with obsessive-compulsive personality disorder attempt to maintain a sense of control through painstaking attention to rules, trivial details, procedures, lists, schedules, or form to the extent that the major point of the activity is lost (Criterion 1). They are excessively careful and prone to repetition, paying extraordinary attention to detail and repeatedly checking for possible mistakes, losing track of time in the process. For example, when such individuals misplace a list of things to be done, they will spend an inordinate amount of time looking for the list rather than

spending a few moments trying their best to recreate it from memory and proceeding to accomplish the tasks. They dismiss the fact that other people tend to become very annoyed at the delays and inconveniences that result from this behavior because they preferentially respond to either their anxiety about making a mistake or their insistence on how things should be done. Time is poorly allocated, and the most important tasks are left to the last moment. The perfectionism and self-imposed high standards of performance cause significant dysfunction and distress in these individuals. They may become so involved in making every detail of a project absolutely perfect that the project is never finished (Criterion 2). For example, the completion of a written report is delayed by numerous time-consuming rewrites that all come up short of "perfection." Deadlines are routinely missed or the individual has a pattern of exerting extraordinary effort (e.g., working through the night, skipping meals) in order to make the deadline at the last moment, and aspects of the individual's life that are not the current focus of activity may fall into disarray.

Individuals with obsessive-compulsive personality disorder display excessive devotion to work and productivity to the exclusion or devaluing of leisure activities and friendships (Criterion 3). This behavior is not accounted for by economic necessity. They often feel that they do not have time to take an evening or a weekend day off to go on an outing or to just relax. They may keep postponing a pleasurable activity, such as a vacation, so that it may never occur. When they reluctantly take time for leisure activities or vacations, they are very uncomfortable unless they have taken along something to work on so they do not "waste time." There may be a great concentration on household chores (e.g., repeated excessive cleaning so that "one could eat off the floor"). If they spend time with friends, it is likely to be in some kind of formally organized activity (e.g., sports). Hobbies or recreational activities are approached as serious tasks or with methodical intensity, requiring careful organization and hard work to master. The emphasis is on perfect performance. These individuals turn play into a structured work-like task (e.g., correcting an infant for not putting rings on the post in the right order; telling a toddler to ride their tricycle in a straight line; turning a baseball game into a harsh "lesson").

Individuals with obsessive-compulsive personality disorder may be excessively conscientious, scrupulous, and inflexible about matters of

morality, ethics, or values (Criterion 4). They may force themselves and others to follow rigid moral principles and very strict standards of performance. They may also be mercilessly self-critical about their own

mistakes or harshly judgmental of others' moral or ethical missteps. Individuals with this disorder are rigidly deferential to authority and rules and insist on quite literal compliance, with no rule bending for extenuating circumstances. For example, the individual will not lend a dollar to a friend who is short of the fare needed to get on a bus because "neither a borrower nor a lender be" or because it would be "bad" for the friend's character. These qualities should not be accounted for by the individual's cultural or religious identification.

Individuals with this disorder may be unable to discard worn-out or worthless objects, even when they have no sentimental value (Criterion 5). Often these individuals will admit to being "pack rats." They regard discarding objects as wasteful because "you never know when you might need something." The clutter may also result from an accumulation of partially read learning material or unfinished projects that the individual intends to get to someday but that have been sidelined because of procrastination and/or a meticulous yet slow work style. These individuals will become upset if someone tries to get rid of the things they have saved. Their spouses or roommates may complain about the amount of space taken up by old parts, piles of reading material, broken appliances, and so on.

Individuals with obsessive-compulsive personality disorder are reluctant to delegate tasks or to work with others (Criterion 6). They stubbornly and unreasonably insist that everything be done their way and that people conform to their way of doing things. They often give very detailed instructions about how things should be done (e.g., there is one and only one way to mow the lawn, wash the dishes, load the dishwasher, build a doghouse), even to the point of micromanaging others, and are surprised and irritated if others suggest creative alternatives. At other times they may reject offers of help even when behind schedule because they believe no one else can do it right.

Individuals with this disorder may be miserly and stingy (having difficulty spending money on both themselves and others) and maintain a standard of living far below what they can afford, believing that spending must be tightly controlled to provide for future catastrophes (Criterion 7). Obsessive-compulsive personality disorder is characterized by rigidity and stubbornness (Criterion 8). Individuals with this disorder are so concerned about having things done the one "correct" way that they have trouble going along with anyone else's ideas. These individuals plan ahead in meticulous detail and are unwilling to consider changes to these plans or their usual routines. Totally wrapped up in their own perspective, they have difficulty acknowledging the viewpoints of others. Friends and colleagues may become frustrated by this constant rigidity. Even when individuals with obsessive-compulsive personality disorder recognize that it may be in their interest to compromise, they may stubbornly refuse to do so, arguing that it is "the principle of the thing."

Associated Features

When rules and established procedures do not dictate the correct answer, decision-making may become a time-consuming, often painful process (e.g., exhaustively researching options before making a purchase). Individuals with obsessive-compulsive personality disorder may have such difficulty deciding which tasks take priority or what is the best way of doing some particular task that they may never get started on anything. They are prone to become upset or angry in situations in which they are not able to maintain control of their physical or interpersonal environment, although the anger is typically not expressed directly. For example, an individual may be angry when service in a restaurant is poor, but instead of complaining to the management, the individual ruminates about how much to leave as a tip. On other occasions, anger may be expressed with righteous indignation over a seemingly minor matter. Individuals with this disorder may be especially attentive to their relative status in dominance-submission relationships and may display excessive deference to an authority they respect and excessive resistance to authority they do not respect.

Individuals with this disorder have difficulty relating to and sharing emotions. For example, they may express affection in a highly controlled or stilted fashion and may be very uncomfortable in the presence of others who are emotionally expressive. Their everyday relationships have a formal and serious quality, and they may be stiff in situations in which others would smile and be happy (e.g., greeting a lover at the airport). They carefully hold themselves back until they are sure that whatever they say will be perfect. They may be preoccupied with logic and intellect and intolerant of displays of emotion in others. They often have difficulty expressing tender feelings, rarely paying compliments. Individuals with this disorder may experience occupational difficulties and distress, particularly when confronted with new situations that demand flexibility and compromise.

Prevalence

The estimated prevalence of obsessive-compulsive personality disorder based on a probability subsample from Part II of the National Comorbidity Survey Replication was 2.4%. The prevalence of obsessive-compulsive personality disorder in the National Epidemiologic Survey on Alcohol and Related Conditions was 7.9%. A review of five epidemiological studies (three in the United States) found a median prevalence of 4.7%.

Culture-Related Diagnostic Issues

In assessing an individual for obsessive-compulsive personality disorder, the clinician should not include those behaviors that reflect habits, customs, or interpersonal styles that are culturally sanctioned by the individual's reference group. Certain cultural communities place substantial emphasis on work and productivity, and some members of sociocultural groups (e.g., certain religious groups, professions, migrants) may at times rigidly embrace codes of conduct; work demands; restrictive social environments; rules of behavior; or standards that emphasize overconscientiousness, moral scrupulosity, and striving for perfectionism that may be reinforced by norms of the cultural group. Such behaviors should not on their own be considered indications of obsessive-compulsive personality disorder.

Sex- and Gender-Related Diagnostic Issues

In large population-based studies, obsessive-compulsive personality disorder appears to be equally prevalent in men and women.

Differential Diagnosis

Obsessive-compulsive disorder (OCD). Despite the similarity in names, OCD is usually easily distinguished from obsessive-compulsive personality disorder by the presence of true obsessions and compulsions in OCD. When criteria for both obsessive-compulsive personality disorder and OCD are met, both diagnoses should be recorded.

Hoarding disorder. A diagnosis of hoarding disorder should be considered especially when hoarding is extreme (e.g., accumulated stacks of worthless objects present a fire hazard and make it difficult for others to walk through the house). When criteria for both obsessive-compulsive personality disorder and hoarding disorder are met, both diagnoses should be recorded.

Other personality disorders and personality traits. Other personality disorders may be confused with obsessive-compulsive personality disorder because they have certain features in common. It is, therefore, important to distinguish among these disorders based on differences in their characteristic features. However, if an individual has personality

features that meet criteria for one or more personality disorders in addition to obsessive-compulsive personality disorder, all can be diagnosed. Individuals with narcissistic personality disorder may also profess a commitment to perfectionism and believe that others cannot do things as well, but these individuals are more likely to believe that they have achieved perfection, whereas those with obsessive-compulsive personality disorder are usually self-critical. Individuals with narcissistic or antisocial personality disorder lack generosity but will indulge themselves, whereas those with obsessive-compulsive personality disorder adopt a miserly spending style toward both self and others. Both schizoid personality disorder and obsessive-compulsive personality disorder may be characterized by an apparent formality and social detachment. In obsessive-

compulsive personality disorder, this stems from discomfort with emotions and excessive devotion to work, whereas in schizoid personality disorder there is a fundamental lack of capacity for intimacy.

Obsessive-compulsive personality traits in moderation may be especially adaptive, particularly in situations that reward high performance. Only when these traits are inflexible, maladaptive, and persisting and cause significant functional impairment or subjective distress do they constitute obsessive-compulsive personality disorder.

Personality change due to another medical condition. Obsessive-compulsive personality disorder must be distinguished from personality change due to another medical condition, in which the traits are a direct physiological consequence of another medical condition.

Substance use disorders. Obsessive-compulsive personality disorder must also be distinguished from symptoms that may develop in association with persistent substance use.

Comorbidity

Individuals with anxiety disorders (e.g., generalized anxiety disorder, separation anxiety disorder, social anxiety disorder, specific phobias) and OCD have an increased likelihood of having a personality disturbance that meets criteria for obsessive-compulsive personality disorder. Even so, it appears that the majority of individuals with OCD do not have a pattern of behavior that meets criteria for this personality disorder. Many of the features of obsessive-compulsive personality disorder overlap with “type A” personality characteristics (e.g., preoccupation with work, competitiveness, time urgency), and these features may be present in individuals at risk for myocardial infarction. There may be an association between obsessive-compulsive personality disorder and depressive and bipolar disorders and eating disorders.

Other Personality Disorders

Personality Change Due to Another Medical Condition

Diagnostic Criteria F07.0

A. A persistent personality disturbance that represents a change from the individual's previous characteristic personality pattern.

Note: In children, the disturbance involves a marked deviation from normal development or a significant change in the child's usual behavior patterns, lasting at least 1 year.

B. There is evidence from the history, physical examination, or laboratory findings that the disturbance is the direct pathophysiological consequence of another medical condition.

C. The disturbance is not better explained by another mental disorder (including another mental disorder due to another medical condition).

D. The disturbance does not occur exclusively during the course of a delirium.

E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify whether:

Labile type: If the predominant feature is affective lability.

Disinhibited type: If the predominant feature is poor impulse control as evidenced by sexual indiscretions, etc.

Aggressive type: If the predominant feature is aggressive behavior.

Apathetic type: If the predominant feature is marked apathy and indifference.

Paranoid type: If the predominant feature is suspiciousness or paranoid ideation.

Other type: If the presentation is not characterized by any of the above subtypes.

Combined type: If more than one feature predominates in the clinical picture.

Unspecified type

Coding note: Include the name of the other medical condition (e.g., F07.0 personality change due to temporal lobe epilepsy). The other medical condition should be coded and listed separately immediately before the personality change due to another medical condition (e.g., G40.209 temporal lobe epilepsy; F07.0 personality change due to temporal lobe epilepsy).

Subtypes

The particular personality change can be specified by indicating the symptom presentation that predominates in the clinical presentation.

Diagnostic Features

The essential feature of a personality change due to another medical condition is a persistent personality disturbance that is judged to be a physiological consequence of another medical condition. The personality disturbance represents a change from the individual's previous characteristic personality pattern. In children, this condition may be manifested as a marked deviation from normal development rather than as a change in a stable personality pattern (Criterion A). There must be evidence from the history, physical examination, or laboratory findings that the personality change is the direct physiological consequence of another medical condition (Criterion B). The diagnosis is not given if the disturbance is better explained by another mental disorder (Criterion C).

The diagnosis is not given if the disturbance occurs exclusively during the course of a delirium (Criterion D). The disturbance must also cause clinically significant distress or impairment in social, occupational, or other important areas of functioning (Criterion E).

Common manifestations of the personality change include affective instability, poor impulse control, outbursts of aggression or rage grossly out of proportion to any precipitating psychosocial stressor, marked apathy, suspiciousness, or paranoid ideation. The phenomenology of the change is indicated using the subtypes listed in the criteria set. An individual with the disorder is often characterized by others as "not himself [or herself]." Although it shares the term "personality" with the other personality disorders, this diagnosis is distinct by virtue of its specific etiology, different phenomenology, and more variable onset and course.

The clinical presentation in a given individual may depend on the nature and localization of the pathological process. For example, injury to the frontal lobes may yield

symptoms such as lack of judgment or foresight, facetiousness, disinhibition, and euphoria. In this example, the diagnosis of personality change due to frontal lobe injury would be made if a persistent personality disturbance is a deviation from the individual's previous characteristic personality pattern prior to the injury (Criterion A). Right hemisphere strokes have often been shown to evoke personality changes in association with unilateral spatial neglect, anosognosia (i.e., inability of the individual to recognize a bodily or functional deficit, such as the existence of hemiparesis), motor impersistence, and other neurological deficits.

Associated Features

A variety of neurological and other medical conditions may cause personality changes, including central nervous system neoplasms, head trauma, cerebrovascular disease, Huntington's disease, epilepsy, infectious conditions with central nervous system involvement (e.g., HIV), endocrine conditions (e.g., hypothyroidism, hypo- and hyperadrenocorticism), and

autoimmune conditions with central nervous system involvement (e.g., systemic lupus erythematosus). The associated physical examination findings, laboratory findings, and patterns of prevalence and onset reflect those of the neurological or other medical condition involved.

Differential Diagnosis

Chronic medical conditions associated with pain and disability. Chronic medical conditions associated with pain and disability can also be associated with changes in personality. The diagnosis of personality change due to another medical condition is given only if a direct pathophysiological mechanism can be established. This diagnosis is not given if the change is due to a behavioral or psychological adjustment or response to another medical condition (e.g., dependent behaviors that result from a need for the assistance of others following a severe head trauma, cardiovascular disease, or dementia).

Delirium or major neurocognitive disorder. Personality change is a frequently associated feature of a delirium or major neurocognitive disorder. A separate diagnosis of personality change due to another medical condition is not given if the change occurs exclusively during the course of a delirium. However, the diagnosis of personality change due to another medical condition may be given in addition to the diagnosis of major neurocognitive disorder if the personality change is judged to be a physiological consequence of the pathological process causing the neurocognitive disorder and if the personality change is a prominent part of the clinical presentation.

Another mental disorder due to another medical condition. The diagnosis of personality change due to another medical condition is not given if the disturbance is better explained by another mental disorder due to another medical condition (e.g., depressive disorder due to brain tumor).

Substance use disorders. Personality changes may also occur in the context of substance use disorders, especially if the disorder is long-standing. The clinician should inquire carefully about the nature and extent of substance use. If the clinician wishes to indicate an etiological relationship between the personality change and substance use, the other specified category for

the specific substance can be used (e.g., other specified stimulant-related disorder with personality change).

Other mental disorders. Marked personality changes may also be an associated feature of other mental disorders (e.g., schizophrenia; delusional disorder; depressive and bipolar disorders; other specified and unspecified disruptive behavior, impulse-control, and conduct disorders; panic disorder). However, in these disorders, no specific physiological factor is judged to be etiologically related to the personality change.

Other personality disorders. Personality change due to another medical condition can be distinguished from a personality disorder by the requirement for a clinically significant change from baseline personality functioning and the presence of a specific etiological medical condition.

Other Specified Personality Disorder

F60.89

This category applies to presentations in which symptoms characteristic of a personality disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the personality disorders diagnostic class. The other specified personality disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific personality disorder. This is done by recording “other specified personality disorder” followed by the specific reason (e.g., “mixed personality features”).

Unspecified Personality Disorder

F60.9

This category applies to presentations in which symptoms characteristic of a personality disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the personality disorders diagnostic class. The unspecified personality disorder category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific personality disorder and includes presentations in which there is insufficient information to make a more specific diagnosis.

Paraphilic Disorders

Paraphilic disorders included in this manual are voyeuristic disorder (spying on others in private activities), exhibitionistic disorder (exposing the genitals), frotteuristic disorder (touching or rubbing against a nonconsenting person), sexual masochism disorder (undergoing humiliation, bondage, or suffering), sexual sadism disorder (inflicting humiliation, bondage, or suffering), pedophilic disorder (sexual focus on children), fetishistic disorder (using nonliving objects or having a highly specific focus on nongenital body parts), and transvestic disorder (engaging in sexually arousing cross-dressing). These disorders have traditionally been selected for specific listing and assignment of explicit diagnostic criteria in DSM for two main reasons: they are relatively common, in relation to other paraphilic disorders, and some of them entail actions for their satisfaction that, because of their noxiousness or potential harm to others, are classed as criminal offenses. The eight listed disorders do not exhaust the list of possible paraphilic disorders. Many dozens of distinct paraphilias have been identified and named, and almost any of them could, by virtue of its negative consequences for the individual or for others, rise to the level of a paraphilic disorder.

In this chapter, the order of presentation of the listed paraphilic disorders generally corresponds to common classification schemes for these conditions. The first group of disorders is based on *anomalous activity preferences.* These disorders are subdivided into *courtship disorders,* which resemble distorted components of human courtship behavior (voyeuristic disorder, exhibitionistic disorder, and frotteuristic disorder), and *algolagnic disorders,* which involve pain and suffering (sexual masochism disorder and sexual sadism disorder). The second group of disorders is based on *anomalous target preferences.* These disorders include one directed at other

humans (pedophilic disorder) and two directed elsewhere (fetishistic disorder and transvestic disorder).

The term *paraphilia* denotes any intense and persistent sexual interest other than sexual interest in genital stimulation or preparatory fondling with phenotypically normal, physically mature, consenting human partners. In some circumstances, the criteria "intense and persistent" may be difficult to apply, such as in the assessment of persons who are very old or medically ill and who may not have "intense" sexual interests of any kind. In such circumstances, the term *paraphilia* may be defined as any sexual interest greater than or equal to nonparaphilic sexual interests. There are also specific paraphilias that are generally better described as *preferential* sexual interests than as intense sexual interests.

Some paraphilias primarily concern the individual's erotic activities, and others primarily concern the individual's erotic targets. Examples of the former would include intense and persistent interests in spanking, whipping, cutting, binding, or strangulating another person, or an interest in these activities that equals or exceeds the individual's interest in copulation or equivalent interaction with another person. Examples of the latter would include intense or preferential sexual interest in children, corpses, or amputees (as a class), as well as intense or preferential interest in nonhuman animals, such as horses or dogs, or in inanimate objects, such as shoes or articles made of rubber. An individual's pattern of paraphilic interests is often reflected in his or her choice of pornography.

A *paraphilic disorder* is a paraphilia that is currently causing distress or impairment to the individual or a paraphilia whose satisfaction has entailed personal harm, or risk of harm, to others. A paraphilia is a necessary but not a sufficient condition for having a paraphilic disorder, and a paraphilia by itself does not necessarily justify or require clinical intervention.

In the diagnostic criteria set for each of the listed paraphilic disorders, Criterion A specifies the qualitative nature of the paraphilia (e.g., an erotic focus on children or on exposing the genitals to strangers), and Criterion B specifies the negative consequences of the paraphilia (i.e., distress, impairment, or harm to others). In keeping with the distinction between

paraphilias and paraphilic disorders, the term *diagnosis* should be reserved for individuals whose paraphilic interests or behaviors meet both Criteria A and B (i.e., individuals who have a paraphilic disorder). If an individual's paraphilic interests or behaviors meet Criterion A but not Criterion B for a particular paraphilia—a circumstance that might arise when a benign paraphilia is discovered during the clinical investigation of some other condition—then the individual may be said to have that paraphilia but not a paraphilic disorder.

It is not rare for an individual to manifest two or more paraphilias. In some cases, the paraphilic foci are closely related and the connection between the paraphilias is intuitively comprehensible (e.g., foot fetishism and shoe fetishism). In other cases, the connection between the paraphilias is not obvious, and the presence of multiple paraphilias may be coincidental or else related to some generalized vulnerability to anomalies of psychosexual development. In any event, comorbid diagnoses of separate paraphilic disorders may be warranted if more than one paraphilia is causing suffering to the individual or harm to others.

Because of the two-pronged nature of diagnosing paraphilic disorders, clinician-rated or self-rated measures and severity assessments could address either the strength of the paraphilia itself or the seriousness of its consequences. Although the distress and impairment stipulated in the Criterion B are special in being the immediate or ultimate result of the paraphilia and not primarily the result of some other factor, the phenomena of reactive depression, anxiety, guilt, poor work history, impaired social relations, and so on are not unique in themselves and may be quantified with multipurpose measures of psychosocial functioning or quality of life.

Voyeuristic Disorder

Diagnostic Criteria F65.3

A. Over a period of at least 6 months, recurrent and intense sexual arousal from observing an unsuspecting person who

is naked, in the process of disrobing, or engaging in sexual activity, as manifested by fantasies, urges, or behaviors.

B. The individual has acted on these sexual urges with a nonconsenting person, or the sexual urges or fantasies cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The individual experiencing the arousal and/or acting on the urges is at least 18 years of age.

Specify if:

In a controlled environment: This specifier is primarily applicable to individuals living in institutional or other settings where opportunities to engage in voyeuristic behavior are restricted.

In full remission: The individual has not acted on the urges with a nonconsenting person, and there has been no distress or impairment in social, occupational, or other areas of functioning, for at least 5 years while in an uncontrolled environment.

Specifiers

The “in full remission” specifier does not address the continued presence or absence of voyeurism per se, which may still be present after behaviors and distress have remitted.

Diagnostic Features

The diagnostic criteria for voyeuristic disorder can apply both to individuals who more or less freely disclose this paraphilic interest and to those who categorically deny any sexual arousal from observing an unsuspecting person who is naked, disrobing, or engaged in sexual activity despite substantial objective evidence to the contrary. If disclosing individuals also report distress or psychosocial problems because of their voyeuristic sexual

preferences, they could be diagnosed with voyeuristic disorder. On the other hand, if they declare no distress, demonstrated by lack of anxiety, obsessions, guilt, or shame, about these paraphilic impulses and are not impaired in other important areas of functioning because of this sexual interest, and their psychiatric or legal histories indicate that they do not act on it, they could be ascertained as having voyeuristic sexual interest but should *not* be diagnosed with voyeuristic disorder.

Nondisclosing individuals include, for example, individuals known to have been spying repeatedly on unsuspecting persons who are naked or engaging in sexual activity on separate occasions but who deny any urges or fantasies concerning such sexual behavior, and who may report that known episodes of watching unsuspecting naked or sexually active persons were all accidental and nonsexual. Others may disclose past episodes of observing unsuspecting naked or sexually active persons but contest any significant or sustained sexual interest in this behavior. Since these individuals deny having fantasies or impulses about watching others nude or involved in sexual activity, it follows that they would also reject feeling subjectively distressed or socially impaired by such impulses. Despite their nondisclosing stance, such individuals may be diagnosed with voyeuristic disorder. Recurrent voyeuristic behavior constitutes sufficient support for voyeurism (by fulfilling Criterion A) and simultaneously demonstrates that this paraphilically motivated behavior is causing harm to others (by fulfilling Criterion B).

“Recurrent” spying on unsuspecting persons who are naked or engaging in sexual activity may be interpreted as requiring multiple victims, each on a separate occasion; this requirement for multiple victims on separate occasions is relevant because it increases the confidence in the clinical inference that the individual is motivated by voyeuristic disorder. Fewer victims can be interpreted as satisfying this criterion if there were multiple occasions of watching the same victim or if there is corroborating evidence of a distinct or preferential interest in secret watching of naked or sexually active unsuspecting persons. Note that multiple victims, as suggested earlier, are a sufficient but not a necessary condition for diagnosis; the criteria may also be met if the individual acknowledges intense voyeuristic sexual interest.

Adolescence and puberty generally increase sexual curiosity and activity. To reduce the risk of pathologizing normative sexual interest and behavior during pubertal adolescence, the minimum age for the diagnosis of voyeuristic disorder is 18 years (Criterion C).

Prevalence

The population prevalence of individuals whose presentations meet the full criteria for voyeuristic disorder is unknown. Voyeuristic acts, however, are the most common of potentially law-breaking sexual behaviors. For example, in a Quebec Internet and phone survey sample, the lifetime prevalence of voyeuristic behaviors was reported to be as high as 34.5% (50.3% in men, 21.2% in women). Because this same study found that an "intense desire" and "persistent behavior" occur with much less frequency (9.6% and 2.1%, respectively), the prevalence of voyeuristic disorder is likely much lower. The ratio of voyeuristic behavior in men to women was approximately 2:1 in the Quebec sample and 3:1 in a Swedish general population sample. In a study determining which particular disorders were prevalent

in individuals incarcerated for sexual offenses, a study of 1,346 incarcerated sex offenders from Austria found a prevalence of voyeuristic disorder of 3.7%.

Development and Course

Adult men with voyeuristic disorder often first become aware of their sexual interest in secretly watching unsuspecting persons during adolescence. However, the minimum age for a diagnosis of voyeuristic disorder is 18 years because there is substantial difficulty in differentiating it from age-appropriate puberty-related sexual curiosity and activity. The persistence of voyeurism over time is unclear. With or without treatment of voyeuristic disorder, the subjective distress (e.g., guilt, shame, intense sexual frustration, loneliness) or impairment from the disorder may change over time, as may a number of factors that may potentially affect the course

of the disorder, such as psychiatric morbidity, hypersexuality, and sexual impulsivity. Thus, the severity and course may vary over time. As with other sexual preferences, advancing age may be associated with decreasing voyeuristic sexual preferences and behavior.

Risk and Prognostic Factors

Temperamental. Because voyeurism is a necessary precondition for voyeuristic disorder, risk factors for voyeurism should also increase the risk of voyeuristic disorder.

Environmental. Childhood sexual abuse, substance misuse, and sexual preoccupation/hypersexuality have been suggested as risk factors, although the causal relationship to voyeuristic behavior is uncertain and the specificity unclear.

Sex- and Gender-Related Diagnostic Issues

Voyeuristic disorder is very uncommon among women in clinical settings, whereas the ratio in men to women for single sexually arousing voyeuristic acts is less extreme and may be 2:1–3:1.

Differential Diagnosis

Voyeurism. Individuals with voyeurism experience recurrent, intense sexual arousal from the act of observing an unsuspecting person who is naked, in the process of disrobing, or engaging in sexual activity. Unless the individual acts on these urges with an unsuspecting person (e.g., surreptitiously peeping through a neighbor's window) or unless there is accompanying clinically significant distress or impairment in social, occupational, or other important areas of functioning, a diagnosis of voyeuristic disorder is not warranted.

Manic episode, major neurocognitive disorder, intellectual developmental disorder, personality change due to another medical condition, substance intoxication, and schizophrenia. Individuals with a major neurocognitive disorder, intellectual developmental disorder, personality change due to another medical condition, or schizophrenia, or who are in a manic episode or experiencing substance intoxication, may become sexually disinhibited or have impaired

judgment or impulse control and engage in voyeuristic behavior. Unless that behavior occurs at times other than in the context of one of these disorders, a diagnosis of voyeuristic disorder should not be made.

Conduct disorder and antisocial personality disorder. Conduct disorder in adolescents and antisocial personality disorder would be characterized by additional norm-breaking and antisocial behaviors, and the specific sexual interest in secretly watching unsuspecting others who are naked or engaging in sexual activity will usually be lacking.

Comorbidity

Known comorbidities in voyeuristic disorder are largely based on research with males suspected of or convicted for acts involving the secret watching of unsuspecting nude or

sexually active persons. Hence, these comorbidities might not apply to all individuals with voyeuristic disorder. Conditions that occur comorbidly with voyeuristic disorder include hypersexuality and other paraphilic disorders, particularly exhibitionistic disorder. Depressive, bipolar, anxiety, and substance use disorders; attention-deficit/hyperactivity disorder; and conduct disorder and antisocial personality disorder are also frequent comorbid conditions.

Exhibitionistic Disorder

Diagnostic Criteria	**F65.2**

A. Over a period of at least 6 months, recurrent and intense sexual arousal from the exposure of one’s genitals to an unsuspecting person, as manifested by fantasies, urges, or behaviors.

B. The individual has acted on these sexual urges with a nonconsenting person, or the sexual urges or fantasies cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify whether:

Sexually aroused by exposing genitals to prepubertal children

Sexually aroused by exposing genitals to physically mature individuals

Sexually aroused by exposing genitals to prepubertal children and to physically mature individuals

Specify if:

In a controlled environment: This specifier is primarily applicable to individuals living in institutional or other settings where opportunities to expose one's genitals are restricted.

In full remission: The individual has not acted on the urges with a nonconsenting person, and there has been no distress or impairment in social, occupational, or other areas of functioning, for at least 5 years while in an uncontrolled environment.

Subtypes

The subtypes for exhibitionistic disorder are based on the age or physical maturity of the nonconsenting persons to whom the individual prefers to expose his or her genitals. The nonconsenting persons could be prepubescent children, adults, or both. This specifier should help draw adequate attention to characteristics of victims of individuals with exhibitionistic disorder to prevent co-occurring pedophilic disorder from being overlooked. However, indications that the individual with exhibitionistic disorder is sexually attracted to exposing his or her genitals to children should not preclude a diagnosis of pedophilic disorder.

Specifiers

The “in full remission” specifier does not address the continued presence or absence of exhibitionism per se, which may still be present after behaviors and distress have remitted.

Diagnostic Features

The diagnostic criteria for exhibitionistic disorder can apply both to individuals who more or less freely disclose this paraphilia and to those who categorically deny any sexual arousal from exposing their genitals to unsuspecting persons despite substantial objective evidence to the contrary. If disclosing individuals also report psychosocial difficulties because of their sexual attractions or preferences for exposing, they may be diagnosed with exhibitionistic

disorder. In contrast, if they declare no distress (exemplified by absence of anxiety, obsessions, and guilt or shame about these paraphilic impulses) and are not impaired by this sexual interest in other important areas of functioning, and their self-reported, psychiatric, or legal histories indicate that they do not act on them, they could be ascertained as having exhibitionistic sexual interest but *not* be diagnosed with exhibitionistic disorder.

Examples of nondisclosing individuals include those who have exposed themselves repeatedly to unsuspecting persons on separate occasions but who deny any urges or fantasies about such sexual behavior and who report that known episodes of exposure were all accidental and nonsexual. Others may disclose past episodes of sexual behavior involving genital exposure but refute any significant or sustained sexual interest in such behavior. Since these individuals deny having urges or fantasies involving genital exposure, it follows that they would also deny feeling subjectively distressed or socially impaired by such impulses. Such individuals may be diagnosed with exhibitionistic disorder despite their negative self-report. Recurrent exhibitionistic behavior constitutes sufficient support for exhibitionism (Criterion A) and simultaneously demonstrates that this paraphilically motivated behavior is causing harm to others (Criterion B).

“Recurrent” genital exposure to unsuspecting others may be interpreted as requiring multiple victims, each on a separate occasion; this requirement for multiple victims on separate occasions is relevant because it increases the confidence in the clinical inference that the individual is motivated by exhibitionistic disorder. Fewer victims can be interpreted as satisfying this criterion if there were multiple occasions of exposure to the same victim, or if there is corroborating evidence of a strong or preferential interest in genital exposure to unsuspecting persons. Note that multiple victims, as suggested earlier, are a sufficient but not a necessary condition for diagnosis, as criteria may be met by an individual’s acknowledging intense exhibitionistic sexual interest with distress or impairment.

Prevalence

The population prevalence of individuals whose presentations meet the full criteria for exhibitionistic disorder is unknown, although the disorder is highly unusual in women. Exhibitionistic acts, however, are not uncommon, and single sexually arousing exhibitionistic acts occur up to half as often among women compared with men. In a Quebec Internet and phone survey sample, lifetime prevalence of exhibitionistic behaviors was reported to be 30.9% (32.6% in men, 29.4% in women). Because this same study found that an “intense desire” and “persistent behavior” occur with much less frequency (4.8% and 0.8%, respectively), the prevalence of exhibitionistic disorder is likely much lower. For example, a Swedish study suggested that the lifetime prevalence of exhibitionistic disorder in the general population was 4.1% in men and 2.1% in women.

Development and Course

Adult men with exhibitionistic disorder often report that they first became aware of sexual interest in exposing their genitals to unsuspecting persons during adolescence, at a somewhat later time than the typical development of normative sexual interest in women or men. Although there is no minimum age requirement for the diagnosis of exhibitionistic disorder, it may be difficult to differentiate exhibitionistic behaviors from age-appropriate sexual curiosity in adolescents. Whereas exhibitionistic impulses appear to emerge in adolescence or early adulthood, very little is

known about persistence over time. With or without treatment of exhibitionistic disorder, the subjective distress (e.g., guilt, shame, intense sexual frustration, loneliness) or impairment from the disorder may change over time, as may a number of factors that may potentially affect the course of the disorder, such as psychiatric morbidity, hypersexuality, and sexual impulsivity. Thus, the severity and course may vary over time. As with other sexual preferences, advancing age may be associated with decreasing exhibitionistic sexual preferences and behavior.

Risk and Prognostic Factors

Temperamental. Because exhibitionism is a necessary precondition for exhibitionistic disorder, risk factors for exhibitionism should also increase the risk of exhibitionistic disorder. Antisocial history, antisocial personality disorder, alcohol misuse, and pedophilic sexual preference might increase risk of sexual recidivism in exhibitionistic offenders. Hence, antisocial personality disorder, alcohol use disorder, and pedophilic interest may be considered risk factors for exhibitionistic disorder in men with exhibitionistic sexual preferences.

Environmental. Childhood sexual and emotional abuse and sexual preoccupation/hypersexuality have been suggested as risk factors for exhibitionism, although the causal relationship to exhibitionism is uncertain and the specificity unclear.

Differential Diagnosis

Exhibitionism. Individuals with exhibitionism experience recurrent, intense sexual arousal from the act of exposing their genitals to an unsuspecting person. Unless the individual acts on these urges with an unsuspecting person (e.g., exposing his genitals to riders on a train) or unless there is accompanying clinically significant distress or impairment in social, occupational, or other important areas of functioning, a diagnosis of exhibitionistic disorder is not warranted.

Manic episode, major neurocognitive disorder, intellectual developmental disorder, personality change due to another medical condition, substance intoxication, and

schizophrenia. Individuals with a major neurocognitive disorder, intellectual developmental disorder, personality change due to another medical condition, or schizophrenia, or who are in a manic episode or experiencing substance intoxication, may become sexually disinhibited or have impaired judgment or impulse control and engage in exhibitionistic behavior. Unless that behavior occurs at times other than in the context of one of these disorders, a diagnosis of exhibitionistic disorder should not be made.

Conduct disorder and antisocial personality disorder. Conduct disorder in adolescents and antisocial personality disorder would be characterized by additional norm-breaking and antisocial behaviors, and the specific sexual interest in exposing the genitals will usually be lacking.

Comorbidity

Known comorbidities in exhibitionistic disorder are largely based on research with individuals (almost all men) convicted for criminal acts involving genital exposure to nonconsenting persons. Hence, these comorbidities might not apply to all individuals who qualify for a diagnosis of exhibitionistic disorder. Conditions that occur comorbidly with exhibitionistic disorder at high rates include depressive, bipolar, anxiety, and substance use disorders; hypersexuality; attention-deficit/hyperactivity disorder; other paraphilic disorders; and antisocial personality disorder.

Frotteuristic Disorder

Diagnostic Criteria F65.81

A. Over a period of at least 6 months, recurrent and intense sexual arousal from touching or rubbing against a nonconsenting person, as manifested by fantasies, urges, or behaviors.
B. The individual has acted on these sexual urges with a nonconsenting person, or the sexual urges or fantasies cause

clinically significant distress or impairment in social, occupational, or other important areas of functioning.

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Specify if:

In a controlled environment: This specifier is primarily applicable to individuals living in institutional or other settings where opportunities to touch or rub against a nonconsenting person are restricted.

In full remission: The individual has not acted on the urges with a nonconsenting person, and there has been no distress or impairment in social, occupational, or other areas of functioning, for at least 5 years while in an uncontrolled environment.

Specifiers

The “in remission” specifier does not address the continued presence or absence of frotteurism per se, which may still be present after behaviors and distress have remitted.

Diagnostic Features

The diagnostic criteria for frotteuristic disorder can apply both to individuals who relatively freely disclose this paraphilia and to those who firmly deny any sexual arousal from touching or rubbing against a nonconsenting person regardless of considerable objective evidence to the contrary. If disclosing individuals also report psychosocial impairment because of their sexual preferences for touching or rubbing against a nonconsenting person, they could be diagnosed with frotteuristic disorder. In contrast, if they declare no distress (demonstrated by lack of anxiety, obsessions, guilt, or shame) about these paraphilic impulses and are not impaired in other important areas of functioning because of this sexual interest, and their psychiatric or legal histories indicate that they do not act

on it, they could be ascertained as having frotteuristic sexual interest but should *not* be diagnosed with frotteuristic disorder.

Nondisclosing individuals include, for instance, individuals known to have been touching or rubbing against nonconsenting persons on separate occasions but who contest any urges or fantasies concerning such sexual behavior. Such individuals may report that identified episodes of touching or rubbing against an unwilling individual were all unintentional and nonsexual. Others may disclose past episodes of touching or rubbing against nonconsenting persons but contest any major or persistent sexual interest in this. Since these individuals deny having fantasies or impulses about touching or rubbing, they would consequently reject feeling distressed or psychosocially impaired by such impulses. Despite their nondisclosing position, such individuals may be diagnosed with frotteuristic disorder. Recurrent frotteuristic behavior constitutes satisfactory support for frotteurism (by fulfilling Criterion A) and concurrently demonstrates that this paraphilically motivated behavior is causing harm to others (by fulfilling Criterion B).

"Recurrent" touching or rubbing against a nonconsenting person may be interpreted as requiring multiple victims, each on a separate occasion; this requirement for multiple victims on separate occasions is relevant because it increases the confidence in the clinical inference that the individual is motivated by frotteuristic disorder. Fewer victims can be interpreted as satisfying this criterion if there were multiple occasions of touching or rubbing against the same unwilling individual, or corroborating evidence of a strong or preferential interest in touching or rubbing against nonconsenting persons. Note that multiple victims are a sufficient but not a necessary condition for diagnosis; criteria may also be met if the individual acknowledges intense frotteuristic sexual interest with clinically significant distress and/or impairment.

Prevalence

The population prevalence of individuals whose presentations meet the full criteria for frotteuristic disorder is unknown, but frotteuristic acts, including the uninvited sexual touching of or rubbing against another individual, may

occur in up to 30% of adult men in the U.S. and Canadian general population. Prevalence of frotteuristic disorder is certainly

much lower, considering the finding that “intense desire” and “persistent behavior” were reported infrequently (3.8% and 0.7%, respectively). In outpatient settings for men with paraphilic disorders and hypersexuality, approximately 10%–14% have a presentation that meets diagnostic criteria for frotteuristic disorder. Prevalence among women is likely lower.

Development and Course

Adult men with frotteuristic disorder often report first becoming aware of their sexual interest in surreptitiously touching unsuspecting persons during late adolescence or emerging adulthood. However, children and adolescents may also touch or rub against unwilling others in the absence of a diagnosis of frotteuristic disorder. Although there is no minimum age for the diagnosis, frotteuristic disorder can be difficult to differentiate from conduct-disordered behavior without sexual motivation in individuals at younger ages. The persistence of frotteurism over time is unclear. With or without treatment of frotteuristic disorder, the subjective distress (e.g., guilt, shame, intense sexual frustration, loneliness) or impairment from the disorder may change over time, as may a number of factors that may potentially affect the course of the disorder, such as psychiatric morbidity, hypersexuality, and sexual impulsivity. Thus, the severity and course may vary over time. As with other sexual preferences, advancing age may be associated with decreasing frotteuristic sexual preferences and behavior.

Risk and Prognostic Factors

Temperamental. Nonsexual antisocial behavior and sexual preoccupation/hypersexuality might be nonspecific risk factors, although the causal relationship to frotteurism is uncertain and the specificity unclear. However, because frotteurism is a necessary precondition for frotteuristic disorder, risk factors for frotteurism should also increase the risk of frotteuristic disorder.

Differential Diagnosis

Frotteurism. Individuals with frotteurism experience recurrent intense sexual arousal from the act of touching or rubbing against a nonconsenting person. Unless the individual acts on these urges with a nonconsenting person (e.g., rubbing his genitals against a passenger on a crowded subway car) or unless there is accompanying clinically significant distress or impairment in social, occupational, or other important areas of functioning, a diagnosis of frotteuristic disorder is not warranted.

Manic episode, major neurocognitive disorder, intellectual developmental disorder, personality change due to another medical condition, substance intoxication, and schizophrenia. Individuals with a major neurocognitive disorder, intellectual developmental disorder, personality change due to another medical condition, or schizophrenia, or who are in a manic episode or experiencing substance intoxication, may become sexually disinhibited or have impaired judgment or impulse control and engage in frotteuris-tic behavior. Unless that behavior occurs at times other than in the context of one of these disorders, a diagnosis of frotteuristic disorder should not be made.

Conduct disorder and antisocial personality disorder. Conduct disorder in adolescents and antisocial personality disorder would be characterized by additional norm-breaking and antisocial behaviors, and the specific sexual interest in touching or rubbing against a nonconsenting person will usually be lacking.

Comorbidity

Known comorbidities in frotteuristic disorder are largely based on research with men suspected of or convicted for criminal acts involving sexually motivated touching of or

rubbing against a nonconsenting person. Hence, these comorbidities might not apply to other individuals with a diagnosis of frotteuristic disorder based on subjective distress over their sexual interest. Conditions that occur comorbidly with frotteuristic disorder include hypersexuality and other

paraphilic disorders, particularly exhibitionistic disorder and voyeuristic disorder. Conduct disorder, antisocial personality disorder, depressive disorders, bipolar disorders, anxiety disorders, and substance use disorders also co-occur.

Sexual Masochism Disorder

Diagnostic Criteria F65.51

A. Over a period of at least 6 months, recurrent and intense sexual arousal from the act of being humiliated, beaten, bound, or otherwise made to suffer, as manifested by fantasies, urges, or behaviors.

B. The fantasies, sexual urges, or behaviors cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

With asphyxiophilia: If the individual engages in the practice of achieving sexual arousal related to restriction of breathing.

Specify if:

In a controlled environment: This specifier is primarily applicable to individuals living in institutional or other settings where opportunities to engage in masochistic sexual behaviors are restricted.

In full remission: There has been no distress or impairment in social, occupational, or other areas of functioning for at least 5 years while in an uncontrolled environment.

Diagnostic Features

The diagnostic criteria for sexual masochism disorder are intended to apply to individuals who freely admit to having such paraphilic interests. Such individuals openly acknowledge intense sexual arousal from the act of

being humiliated, beaten, bound, or otherwise made to suffer, as manifested by fantasies, urges, or behaviors. If these individuals also report psychosocial difficulties because of their sexual attractions or preferences for being humiliated, beaten, bound, or otherwise made to suffer, they may be diagnosed with sexual masochism disorder. In contrast, if they declare no distress, exemplified by anxiety, obsessions, guilt, or shame, about these paraphilic impulses, and are not hampered by them in pursuing other personal goals, they could be ascertained as having masochistic sexual interest but should *not* be diagnosed with sexual masochism disorder.

The term *bondage-domination-sadism-masochism* (BDSM) is broadly used to refer to a wide range of behaviors that individuals with sexual masochism and/or sexual sadism (as well as other individuals with similar sexual interests) engage in, such as restraints or restriction, discipline, spanking, slapping, sensory deprivation (e.g., using blindfolds), and dominance-submission role-play involving themes such as master/enslaved person, owner/pet, or kidnapper/victim.

Associated Features

The extensive use of pornography involving the act of being humiliated, beaten, bound, or otherwise made to suffer is sometimes an associated feature of sexual masochism disorder. Those who engage in sadomasochistic sexual behavior may experience a hyposensitivity to pain, although it is unknown whether this finding applies to those with sexual masochism disorder. Additionally, although it is often assumed that individuals with

masochistic sexual interest have a history of childhood sexual abuse experiences, there is insufficient evidence to support this association.

Prevalence

The population prevalence of individuals whose presentations meet the full criteria for sexual masochism disorder is unknown. In Australia, it has been

estimated that 2.2% of men and 1.3% of women had been involved in BDSM behavior in the past 12 months.

Development and Course

Individuals with paraphilias living in the community have reported a mean age at onset for masochism of 19.3 years, although earlier ages, including puberty and childhood, have also been reported for the onset of masochistic fantasies. Very little is known about persistence over time. With or without treatment of sexual masochism disorder, the subjective distress (e.g., guilt, shame, intense sexual frustration, loneliness) or impairment from the disorder may change over time, as may a number of factors that may potentially affect the course of the disorder, such as psychiatric morbidity, hypersexuality, and sexual impulsivity. Thus, the severity and course may vary over time. As with other sexual preferences, advancing age may be associated with decreasing sexual masochistic preferences and behavior.

Culture-Related Diagnostic Issues

It is important to distinguish self-harming behaviors that occur during collectively accepted religious and spiritual practices from sadomasochistic behavior conducted for sexual arousal. For example, collective rituals in various religions and societies include suspension from hooks, self-flagellation, self-mortification, and other painful ordeals. The role of sexual arousal or pleasure in these practices remains unknown.

Association With Suicidal Thoughts or Behavior

Association of sexual masochism disorder with suicidal thoughts or behavior is unknown. However, a study of 321 adults who endorsed BDSM involvement found an association of stigma-related shame and guilt with suicidal ideation.

Functional Consequences of Sexual Masochism Disorder

The functional consequences of sexual masochism disorder are unknown. Individuals reporting sexual interest in asphyxiophilia seem to experience

more sexual distress and psychological maladjustment than the general population. Individuals engaging in masochistic behavior are at risk for accidental death while practicing asphyxiophilia or other autoerotic procedures. However, the proportion of these decedents whose sexual interests and behavior fulfill diagnostic criteria for sexual masochism is unknown.

Differential Diagnosis

Sexual masochism. Individuals with sexual masochism experience recurrent, intense sexual arousal from the act of being humiliated, beaten, bound, or otherwise made to suffer. Unless the sexual urges, fantasies, or behaviors involving being humiliated or made to suffer are accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning, a diagnosis of sexual masochism disorder is not warranted.

Comorbidity

Known comorbidities with sexual masochism disorder are largely based on individuals in treatment. Disorders that occur comorbidly with sexual masochism disorder typically

include other paraphilic disorders, such as transvestic fetishism. There is some indication of an association of sexual masochism disorder with borderline personality disorder (based on data from a small clinical sample of women with and without borderline personality disorder).

Sexual Sadism Disorder

Diagnostic Criteria	F65.52

A. Over a period of at least 6 months, recurrent and intense sexual arousal from the physical or psychological suffering of another person, as manifested by fantasies, urges, or behaviors.

B. The individual has acted on these sexual urges with a nonconsenting person, or the sexual urges or fantasies cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

In a controlled environment: This specifier is primarily applicable to individuals living in institutional or other settings where opportunities to engage in sadistic sexual behaviors are restricted.

In full remission: The individual has not acted on the urges with a nonconsenting person, and there has been no distress or impairment in social, occupational, or other areas of functioning, for at least 5 years while in an uncontrolled environment.

Diagnostic Features

The diagnostic criteria for sexual sadism disorder are intended to apply both to individuals who freely admit to having such paraphilic interests and to those who deny any sexual interest in the physical or psychological suffering of another individual despite substantial objective evidence to the contrary. Individuals who openly acknowledge intense sexual interest in the physical or psychological suffering of others are referred to as "admitting individuals." If these individuals also report psychosocial difficulties because of their sexual attractions or preferences for the physical or psychological suffering of another individual, they may be diagnosed with sexual sadism disorder. In contrast, if admitting individuals declare no distress, exemplified by anxiety, obsessions, guilt, or shame, about these paraphilic impulses, and are not hampered by them in pursuing other goals, and their self-reported, psychiatric, or legal histories indicate that they do not act on them with nonconsenting persons, then they could be ascertained

as having sadistic sexual interest but their presentation would *not* meet criteria for sexual sadism disorder.

Examples of individuals who deny any interest in the physical or psychological suffering of another individual include individuals known to have inflicted pain or suffering on multiple victims on separate occasions but who deny any urges or fantasies about such sexual behavior and who may further claim that known episodes of sexual assault were either unintentional or nonsexual. Others may admit past episodes of sexual behavior involving the infliction of pain or suffering on a nonconsenting person but do not report any significant or sustained sexual interest in the physical or psychological suffering of another person. Since these individuals deny having urges or fantasies involving sexual arousal to pain and suffering, it follows that they would also deny feeling subjectively distressed or socially impaired by such impulses. Such individuals may be diagnosed with sexual sadism disorder despite their negative self-report. Their recurrent behavior constitutes clinical support for the presence of the paraphilia of sexual sadism (by satisfying Criterion A) and simultaneously demonstrates that their paraphilically motivated behavior is causing clinically significant distress, harm, or risk of harm to others (satisfying Criterion B).

"Recurrent" sexual sadism involving nonconsenting others may be interpreted as requiring multiple victims, each on a separate occasion; this requirement for multiple victims on separate occasions is relevant because it increases the confidence in the clinical inference that the individual is motivated by sexual sadism disorder. Fewer victims can be interpreted as satisfying this criterion, if there are multiple instances of infliction of pain and suffering to the same victim, or if there is corroborating evidence of a strong or preferential interest in pain and suffering involving multiple victims. Note that multiple victims, as suggested earlier, are a sufficient but not a necessary condition for diagnosis, as the criteria may be met if the individual acknowledges intense sadistic sexual interest.

The term *bondage-domination-sadism-masochism* (BDSM) is broadly used to refer to a wide range of behaviors that individuals with sexual

masochism and/or sexual sadism (as well as other individuals with similar sexual interests) engage in, such as restraints or restriction, discipline, spanking, slapping, sensory deprivation (e.g., using blindfolds), and dominance-submission role-play involving themes such as master/enslaved person, owner/pet, or kidnapper/victim.

Associated Features

The extensive use of pornography involving the infliction of pain and suffering is sometimes an associated feature of sexual sadism disorder.

Prevalence

The population prevalence of individuals whose presentations meet the full criteria for sexual sadism disorder is unknown and is largely based on individuals in forensic settings. Among civilly committed sexual offenders in the United States, less than 10% have sexual sadism disorder. Among individuals who have committed sexually motivated homicides, the proportion of sexually sadistic behavior is about one-third.

Individuals with sexual sadism disorder in forensic samples are almost exclusively men, but a representative sample of the population in Australia reported that 2.2% of men and 1.3% of women said that they had been involved in BDSM behavior in the previous year. In a population-based sample in Finland, the lifetime prevalence for sexually sadistic behavior was 2.7% among men and 2.3% among women.

Development and Course

Information on the development and course of sexual sadism disorder is extremely limited. Whereas sexually sadistic preferences per se are probably a lifelong characteristic, sexual sadism disorder may fluctuate according to the individual's subjective distress or his or her propensity to harm nonconsenting others. As with other sexual preferences, advancing age may be associated with decreasing sexually sadistic preferences and behavior. Regarding sexually sadistic preference, many individuals who engage in BDSM behavior became aware of their corresponding interest in their teenage years.

Culture-Related Diagnostic Issues

The legal status of sexually sadistic behavior ranges across countries and societies, suggesting the potential for variation in distress (because of variation in cultural acceptance) and functional impairment (because of legal status).

Association With Suicidal Thoughts or Behavior

Association of sexual sadism disorder with suicidal thoughts or behavior is unknown. However, a study of 321 adults who endorsed BDSM involvement found an association of stigma-related shame and guilt with suicidal ideation.

Differential Diagnosis

Sexual sadism. Individuals with sexual sadism experience recurrent, intense sexual arousal from the physical or psychological suffering of another person. Unless the sexual urges to make another person suffer physically or psychologically are acted on with a non-consenting person, or unless there is accompanying clinically significant distress or impairment in social, occupational, or other important areas of functioning, a diagnosis of sexual sadism disorder is not warranted. The majority of individuals who are active in community networks that practice sadistic and masochistic behaviors do not express any dissatisfaction with their sexual interests, and their behavior would not meet DSM-5 criteria for sexual sadism disorder.

Infliction of physical or psychological suffering during the commission of a sex crime.

Individuals who commit rape or other sexual assaults might inflict pain on their victims as a result of the act of rape, or in the course of subduing victims or restraining them to commit the sexual assault. Such instrumental infliction of pain should not be considered to be indicative of sexual sadism disorder unless there is evidence that the individual is deriving pleasure from the infliction of pain and the resulting suffering of the victim (e.g., admission of specifically being aroused by the pain, evidence of a preference for pornography involving themes of sexual sadism, excessive

use of pain-inducing violence that goes beyond what might be necessary in the course of committing the sexual assault).

Conduct disorder and antisocial personality disorder. Individuals with conduct disorder and antisocial personality disorder may be physically cruel to people and force others to engage in sexual activity. Coercive or sadistic sexual behaviors that occur in the context of conduct disorder or antisocial personality disorder but that do not reflect an underlying pattern of sexual arousal from the physical or psychological suffering of another person should not be used as a basis for diagnosing sexual sadism disorder. In cases in which the diagnostic criteria are met for both sexual sadism disorder and conduct disorder/antisocial personality disorder, both disorders may be diagnosed.

Comorbidity

Known comorbidities with sexual sadism disorder are largely based on individuals (almost all men) convicted for criminal acts involving sadistic acts against nonconsenting victims. Hence, these comorbidities might not apply to all individuals who never engaged in sadistic activity with a nonconsenting victim but who qualify for a diagnosis of sexual sadism disorder based on subjective distress over their sexual interest. Disorders that are commonly comorbid with sexual sadism disorder include other paraphilic disorders. According to a population-based study in Finland, individuals who had engaged in sexually sadistic behavior had also engaged in other types of paraphilic behavior, namely (in descending order of co-occurrence) masochism (68.8%), voyeurism (33.3%), transvestic fetishism (9.2%), and exhibitionism (6.4%).

Pedophilic Disorder

Diagnostic Criteria	F65.4

A. Over a period of at least 6 months, recurrent, intense sexually arousing fantasies, sexual urges, or behaviors involving

sexual activity with a prepubescent child or children (generally age 13 years or younger).

B. The individual has acted on these sexual urges, or the sexual urges or fantasies cause marked distress or interpersonal difficulty.

C. The individual is at least age 16 years and at least 5 years older than the child or children in Criterion A.

Note: Do not include an individual in late adolescence involved in an ongoing sexual relationship with a 12- or 13-year-old.

Specify whether:

Exclusive type (attracted only to children)

Nonexclusive type

Specify if:

Sexually attracted to males

Sexually attracted to females

Sexually attracted to both

Specify if:

Limited to incest

Diagnostic Features

The diagnostic criteria for pedophilic disorder are intended to apply both to individuals who freely disclose this paraphilia and to individuals who deny any sexual attraction to prepubertal children (generally age 13 years or younger), despite substantial objective evidence to the contrary. The age guideline of 13 or younger is approximate only, because the onset of puberty varies from person to person, and there is good evidence the average age at onset of puberty has been declining over time and differs across ethnicities and cultures. Examples of disclosing this paraphilia

include candidly acknowledging an intense sexual interest in children and indicating that sexual interest in children is greater than or equal to sexual interest in physically mature persons. If individuals also complain that their sexual attractions or preferences for children are causing marked distress or psychosocial difficulties, they may be diagnosed with pedophilic disorder. However, if they report an absence of feelings of guilt, shame, or anxiety about these impulses and are not functionally limited by their paraphilic impulses (according to self-report, objective assessment, or both), and their self-reported and legally recorded histories indicate that they have never acted on their impulses, then these individuals have a pedophilic sexual interest but not pedophilic disorder. When trying to differentiate child offenders with pedophilic disorder from child offenders without pedophilic disorder, factors that suggest a diagnosis of pedophilic disorder in the offender include self-reported interest in children, use of child pornography, a history of multiple child victims, boy victims, and unrelated child victims.

Examples of individuals who deny attraction to children include individuals who are known to have sexually approached multiple children on separate occasions but who deny any urges or fantasies about sexual behavior involving children, and who may further claim that the known episodes of physical contact were all unintentional and nonsexual. Other individuals may acknowledge past episodes of sexual behavior involving children but deny any significant or sustained sexual interest in children. Because these individuals may deny experiences, impulses, or fantasies involving children, they may also deny feeling subjectively distressed. Such individuals may still be diagnosed with pedophilic disorder despite the absence of self-reported distress, provided that there is evidence of recurrent behaviors persisting for 6 months (Criterion A) and evidence that the individual has acted on sexual urges or experienced interpersonal difficulties as a consequence of the disorder (Criterion B). Behaviors include sexual interactions with children, whether or not they involve physical contact (e.g., some pedophilic individuals expose themselves to children). Although the use of sexually explicit content depicting prepubescent children is typical of individuals with pedophilic sexual interests and thus might contribute important information relevant to the evaluation of Criterion A, such behavior in the absence of the individual’s

sexual interactions with children (i.e., acting on these sexual urges in person) is insufficient to conclude that Criterion B is met.

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Presence of multiple victims, as discussed above, is sufficient but not necessary for diagnosis; that is, the individual can still meet Criterion A by merely acknowledging intense or preferential sexual interest in children.

Associated Features

Individuals with pedophilic disorder may experience an emotional and cognitive affinity with children, sometimes referred to as *emotional congruence* with children. Emotional congruence with children can manifest in different ways, including preferring social interactions with children over adults, feeling like one has more in common with children than with adults, and choosing occupations or volunteer roles in order to be around children more often. Studies show that emotional congruence with children is related to both pedophilic sexual interest and the likelihood of sexually reoffending among individuals who have sexually offended.

Prevalence

The population prevalence of individuals whose presentations meet the full criteria for pedophilic disorder is unknown but is likely less than 3% among men in international studies. The population prevalence of pedophilic disorder in women is even more uncertain, but it is likely a small fraction of the prevalence in men.

Development and Course

Adult men with pedophilic disorder may indicate that they became aware of strong or preferential sexual interest in children around the time of puberty —the same time frame in which men who later prefer physically mature partners became aware of their sexual interest in women or men. Attempting to diagnose pedophilic disorder at the age at which it first manifests is problematic because of the difficulty during adolescent development in differentiating it from age-appropriate sexual interest in

peers or from sexual curiosity. Hence, Criterion C requires for diagnosis a minimum age of 16 years and at least 5 years older than the child or children in Criterion A.

Pedophilia per se appears to be a lifelong condition. Pedophilic disorder, however, necessarily includes other elements that may change over time with or without treatment: subjective distress (e.g., guilt, shame, intense sexual frustration, or feelings of isolation) or psychosocial impairment, or the propensity to act out sexually with children, or both. Therefore, the course of pedophilic disorder may fluctuate, or the intensity might increase or decrease with age.

Adults with pedophilic disorder may report an awareness of sexual interest in children that preceded engaging in sexual behavior involving children or self-identification as an individual with pedophilia. Advanced age is as likely to similarly diminish the frequency of sexual behavior involving children as it does other paraphilically motivated and nonparaphilic sexual behavior.

Risk and Prognostic Factors

Temperamental. There appears to be an interaction between pedophilia and antisocial personality traits such as callousness, impulsivity, and a willingness to take risks without adequate regard for the consequences. Men with pedophilic interest and antisocial personality traits are more likely to act out sexually with children and thus qualify for a diagnosis of pedophilic disorder. Thus, antisocial personality disorder may be considered a risk factor for pedophilic disorder in males with pedophilia.

Environmental. Adult men with pedophilia sometimes report that they were sexually abused as children. It is unclear, however, whether this correlation reflects a causal influence of childhood sexual abuse on adult pedophilia.

Genetic and physiological. Since pedophilia is a necessary condition for pedophilic disorder, any factor that increases the probability of pedophilia also increases the risk of pedophilic disorder. There is some evidence that

neurodevelopmental perturbation in utero increases the probability of development of a pedophilic interest.

Sex- and Gender-Related Diagnostic Issues

Laboratory measures of sexual interest, in terms of psychophysiological responses to sexual stimuli depicting children, which are sometimes useful in diagnosing pedophilic disorder in men, are not necessarily useful in diagnosing this disorder in women because there has been very limited research on the assessment of pedophilic sexual interest in women.

Diagnostic Markers

Psychophysiological measures of sexual interest may sometimes be useful when an individual's history suggests the possible presence of pedophilic disorder but the individual denies strong or preferential attraction to children. The most thoroughly researched and longest used of such measures is *penile plethysmography*, although the sensitivity and specificity of diagnosis may vary across sites, which frequently use different stimuli, procedures, and scoring. *Viewing time*, using photographs of nude or minimally clothed persons as visual stimuli, is also used to diagnose pedophilic disorder, especially in combination with self-report measures. U.S. clinicians, however, should be aware that possession of visual sexual stimuli depicting children, even for diagnostic purposes, may violate American law regarding possession of child pornography and leave the clinician susceptible to criminal prosecution. The option exists to use audio stimuli describing sexual interactions in penile plethysmography. Across psychophysiological methods, the diagnostic marker is relative sexual response to stimuli depicting children compared with stimuli depicting adults, rather than absolute response to child stimuli.

Differential Diagnosis

Pedophilia. Individuals with pedophilia experience recurrent, intense, sexually arousing fantasies or sexual urges involving sexual activity with a prepubescent child or children. Unless the individual has acted on these sexual urges with a prepubescent child or unless the sexual urges or

fantasies cause marked distress or interpersonal difficulty, a diagnosis of pedophilic disorder is not warranted.

Other paraphilic disorders. Sometimes individuals present with a different paraphilic disorder but are referred for an evaluation regarding possible pedophilic disorder (e.g., when an individual with a diagnosis of exhibitionistic disorder exposes himself to children as well as adults). In some cases, both diagnoses may apply, whereas in others, it may be the case that one paraphilic disorder diagnosis is sufficient. For example, an individual who exposes himself exclusively to prepubescent children may have both exhibitionistic disorder and pedophilic disorder, whereas another individual who exposes himself to victims, irrespective of the victims' age, may be considered to have only exhibitionistic disorder.

Antisocial personality disorder. Some individuals with antisocial personality disorder sexually abuse children, reflecting the fact that the presence of antisocial personality disorder increases the likelihood that an individual who is primarily attracted to mature persons will approach a child sexually, on the basis of relative access to the child. An additional diagnosis of pedophilic disorder should only be considered if there is evidence that over a period of at least 6 months, the individual has also had recurrent, intense, sexually arousing fantasies, sexual urges, or behaviors involving sexual activity with a prepubescent child.

Substance intoxication. The disinhibiting effects of substance intoxication may also increase the likelihood that an individual who is primarily attracted to mature persons will sexually approach a child.

Obsessive-compulsive disorder. There are occasional individuals who complain about ego-dystonic thoughts and worries about possible attraction to children. Clinical interviewing usually reveals an absence of positive feelings about these thoughts, no connection between these thoughts and sexual behavior (e.g., masturbating to these thoughts), and sometimes additional ego-dystonic, intrusive sexual ideas (e.g., concerns about homosexuality).

Comorbidity

Psychiatric comorbidity of pedophilic disorder includes substance use disorders; depressive, bipolar, and anxiety disorders; antisocial personality disorder; and other paraphilic disorders. However, findings on comorbid disorders are largely among individuals convicted for sexual offenses involving children (almost all males) and may not be generalizable to other individuals with pedophilic disorder (e.g., individuals who have never approached a child sexually but who qualify for the diagnosis of pedophilic disorder on the basis of subjective distress).

Fetishistic Disorder

Diagnostic Criteria F65.0

A. Over a period of at least 6 months, recurrent and intense sexual arousal from either the use of nonliving objects or a highly specific focus on nongenital body part(s), as manifested by fantasies, urges, or behaviors.

B. The fantasies, sexual urges, or behaviors cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The fetish objects are not limited to articles of clothing used in cross-dressing (as in transvestic disorder) or devices specifically designed for the purpose of tactile genital stimulation (e.g., vibrator).

Specify:

Body part(s)

Nonliving object(s)

Other

Specify if:

In a controlled environment: This specifier is primarily applicable to individuals living in institutional or other settings

where opportunities to engage in fetishistic behaviors are restricted.

In full remission: There has been no distress or impairment in social, occupational, or other areas of functioning for at least 5 years while in an uncontrolled environment.

Specifiers

Although individuals with fetishistic disorder may report intense and recurrent sexual arousal to inanimate objects or a specific body part, it is not unusual for non–mutually exclusive combinations of fetishistic sexual interests to occur. Thus, an individual may have fetishistic disorder associated with an inanimate object (e.g., female undergarments) or an exclusive focus on an intensely eroticized body part (e.g., feet, hair), or his or her fetishistic interest may meet criteria for various combinations of these specifiers (e.g., socks, shoes, and feet).

Diagnostic Features

The paraphilic focus of fetishistic disorder involves the persistent and repetitive use of or dependence on nonliving objects or a highly specific focus on a (typically nongenital) body part as a primary element associated with sexual arousal (Criterion A). A diagnosis of fetishistic disorder must include clinically significant personal distress or impairment in social, occupational, or other important areas of functioning (Criterion B). Common fetish objects include women's undergarments, men's or women's footwear, rubber articles, leather clothing, diapers, or other wearing apparel. Highly eroticized body parts associated with fetishistic disorder include feet, toes, and hair. It is not uncommon for sexualized fetishes to include both inanimate objects and body parts (e.g., dirty socks and feet), and for this reason the definition of fetishistic disorder now re-incorporates *partialism* (i.e., an exclusive focus on a body part) into its boundaries. Partialism, previously considered in DSM-IV-TR to be a paraphilia not

otherwise specified, had historically been subsumed in fetishism prior to DSM-III.

Many individuals who self-identify as fetishist practitioners do not necessarily report clinical impairment in association with their fetish-associated behaviors. Such individuals could be considered as having a fetishistic sexual interest (i.e., a recurrent and intense sexual arousal from either the use of nonliving objects or a highly specific focus on a nongenital body part, as manifested by fantasies, urges, or behaviors), but not fetishistic disorder. A diagnosis of fetishistic disorder requires concurrent fulfillment of both the behaviors in Criterion A and the clinically significant distress or impairment in functioning noted in Criterion B.

Associated Features

Fetishistic disorder can be a multisensory experience, including holding, tasting, rubbing, inserting, or smelling the fetish object while masturbating, or preferring that a sexual partner wear or utilize a fetish object during sexual encounters. It should be noted that many individuals with fetishistic sexual interests also enjoy sexual experiences with their partner(s) without using their fetish object. However, it should also be noted that individuals with a fetishistic sexual interest often find that sexual experiences that involve their fetish object are more sexually satisfying than sexual experience without it. And for a minority of people with a fetishistic sexual interest, their fetish object is obligatory to becoming sexually aroused and/or satisfied. Some individuals may acquire extensive collections of highly desired fetish objects.

Development and Course

Usually paraphilias have an onset during puberty, but fetishistic sexual interests can develop prior to adolescence. Once established, fetishistic disorder tends to have a continuous course that fluctuates in intensity and frequency of urges or behavior.

Culture-Related Diagnostic Issues

Knowledge of and appropriate consideration for normative aspects of sexual behavior are important factors to explore to establish a clinical diagnosis of fetishistic disorder and to distinguish a clinical diagnosis from a socially acceptable sexual behavior.

Sex- and Gender-Related Diagnostic Issues

Fetishistic behaviors have been reported more in men, but also occur in women. This gender difference is smaller for fetishistic fantasy than for actual fetishistic behavior. In clinical samples, fetishistic disorder is nearly exclusively reported in men.

Functional Consequences of Fetishistic Disorder

Typical impairments associated with fetishistic disorder include sexual dysfunction during romantic reciprocal relationships when the preferred fetish object or body part is

unavailable during foreplay or coitus. Some individuals with fetishistic disorder may prefer solitary sexual activity associated with their fetishistic preference(s) even while involved in a meaningful reciprocal and affectionate relationship.

Differential Diagnosis

Transvestic disorder. The nearest diagnostic neighbor of fetishistic disorder is transvestic disorder. As noted in the diagnostic criteria, fetishistic disorder is not diagnosed when fetish objects are limited to articles of clothing exclusively worn during cross-dressing (as in transvestic disorder), or when the object is genitally stimulating because it has been designed for that purpose (e.g., a vibrator).

Sexual masochism disorder or other paraphilic disorders. Fetishistic disorder can co-occur with other paraphilic disorders, especially sadomasochistic behavior or interests and transvestic disorder. When an individual fantasizes about or engages in “forced cross-dressing” and is primarily sexually aroused by the domination or humiliation associated with such fantasy or

repetitive activity, and experiences distress or functional impairment, the diagnosis of sexual masochism disorder should be made.

Fetishism. Use of a fetish object for sexual arousal (fetishism) without any associated distress or psychosocial role impairment or other adverse consequence would not meet criteria for fetishistic disorder, as the threshold required by Criterion B would not be met. For example, an individual whose sexual partner either shares or can successfully incorporate his interest in caressing, smelling, or licking feet or toes as an important element of foreplay would not be diagnosed with fetishistic disorder; nor would an individual who prefers, and is not distressed or impaired by, solitary sexual behavior associated with wearing rubber garments or leather boots.

Comorbidity

Fetishistic disorder may co-occur with other paraphilic disorders as well as hypersexuality. Rarely, fetishistic disorder may be associated with neurological conditions.

Transvestic Disorder

Diagnostic Criteria F65.1

A. Over a period of at least 6 months, recurrent and intense sexual arousal from cross-dressing, as manifested by fantasies, urges, or behaviors.

B. The fantasies, sexual urges, or behaviors cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

With fetishism: If sexually aroused by fabrics, materials, or garments.

With autogynephilia: If sexually aroused by thoughts or images of self as a woman.

Specify if:

In a controlled environment: This specifier is primarily applicable to individuals living in institutional or other settings where opportunities to cross-dress are restricted.

In full remission: There has been no distress or impairment in social, occupational, or other areas of functioning for at least 5 years while in an uncontrolled environment.

Specifiers

The presence of fetishism decreases the likelihood of gender dysphoria in men with transvestic disorder. The presence of autogynephilia increases the likelihood of gender dysphoria in men with transvestic disorder.

Diagnostic Features

The diagnosis of transvestic disorder does not apply to all individuals who dress as the opposite sex, even those who do so habitually. It applies to individuals whose cross-dressing or thoughts of cross-dressing are always or often accompanied by sexual excitement (Criterion A) and who are emotionally distressed by this pattern or for whom it impairs their social or interpersonal functioning (Criterion B). The cross-dressing may involve only one or two articles of clothing (e.g., for men, it may pertain only to women's undergarments), or it may involve dressing completely in the inner and outer garments of the other sex and (in men) may include the use of women's wigs and makeup. Sexual arousal, in its most obvious form of penile erection, may co-occur with cross-dressing in various ways. In younger men, cross-dressing often leads to masturbation, following which any women's clothing is removed. Older men often learn to avoid masturbating or doing anything to stimulate the penis so that the avoidance of ejaculation allows them to prolong their cross-dressing session. Men and women sometimes complete a cross-dressing session by having intercourse

with their partners, and some have difficulty maintaining sufficient sexual arousal for sexual activity without cross-dressing (or having private fantasies of cross-dressing).

Clinical assessment of distress or impairment, like clinical assessment of transvestic sexual arousal, is usually dependent on the individual's self-report. The pattern of behavior "purging and acquisition" often signifies the presence of distress in individuals with transvestic disorder. During this behavioral pattern, an individual (usually a man) who has spent a great deal of money on women's clothes and other apparel (e.g., shoes, wigs) discards the items (i.e., purges them) in an effort to overcome urges to cross-dress, and then begins acquiring a woman's wardrobe all over again.

Associated Features

Transvestic disorder in men is often accompanied by *autogynephilia* (i.e., a man's paraphilic tendency to be sexually aroused by the thought or image of himself as a woman). Autogynephilic fantasies and behaviors may focus on the idea of exhibiting female physiological functions (e.g., lactation, menstruation), engaging in stereotypically feminine behavior (e.g., knitting), or possessing female anatomy (e.g., breasts).

Prevalence

The prevalence of transvestic disorder is unknown; however, it appears to be much more prevalent in men than in women. Fewer than 3% of Swedish men report having ever been sexually aroused by dressing in women's attire. The percentage of individuals who have cross-dressed with sexual arousal more than once or a few times in their lifetimes would be even lower.

Development and Course

In men, the first signs of transvestic disorder may begin in childhood, in the form of strong fascination with a particular item of women's attire. Prior to puberty, cross-dressing produces generalized feelings of pleasurable excitement. With the arrival of puberty, dressing in women's clothes begins to elicit penile erection and, in some cases, leads directly to first ejaculation.

In many cases, cross-dressing elicits less and less sexual excitement as the individual grows older; eventually it may produce no discernible penile response at all. The desire to cross-dress, at the same time, remains the same or grows even stronger.

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Individuals who report such a diminution of sexual response typically report that the sexual excitement of cross-dressing has been replaced by feelings of comfort or well-being.

In some cases, the course of transvestic disorder is continuous, and in others it is episodic. It is not rare for men with transvestic disorder to lose interest in cross-dressing when they first fall in love with a woman and begin a relationship, but such abatement usually proves temporary. When the desire to cross-dress returns, so does the associated distress.

Some cases of transvestic disorder progress to gender dysphoria. The men in these cases, who may be indistinguishable from others with transvestic disorder in adolescence or early childhood, gradually develop desires to remain in the woman's role for longer periods and to feminize their anatomy. The development of gender dysphoria is usually accompanied by a (self-reported) reduction or elimination of sexual arousal in association with cross-dressing.

The manifestation of transvestism in penile erection and stimulation, like the manifestation of other paraphilic as well as nonparaphilic sexual interests, is most intense in adolescence and early adulthood. The severity of transvestic disorder is highest in adulthood, when the transvestic drives are most likely to conflict with performance in heterosexual intercourse and desires to marry and start a family. Middle-age and older men with a history of transvestism are less likely to present with transvestic disorder than with gender dysphoria.

Functional Consequences of Transvestic Disorder

Engaging in transvestic behaviors can interfere with, or detract from, heterosexual relationships. This can be a source of distress to men who wish to maintain conventional marriages or romantic partnerships with women.

Differential Diagnosis

Transvestism. Individuals with transvestism experience recurrent and intense sexual arousal from cross-dressing. Unless the fantasies, sexual urges, or behaviors involving cross-dressing are accompanied by clinically significant distress or impairment in social, occupational, or other important areas of functioning, a diagnosis of transvestic disorder is not warranted.

Fetishistic disorder. This disorder may resemble transvestic disorder, in particular, in men with fetishism who put on women's undergarments while masturbating with them. Distinguishing transvestic disorder depends on the individual's specific thoughts during such activity (e.g., are there any ideas of being a woman, being like a woman, or being dressed as a woman?) and on the presence of other fetishes (e.g., soft, silky fabrics, whether these are used for garments or for something else).

Gender dysphoria. Individuals with transvestic disorder do not report an incongruence between their experienced gender and their assigned gender or a desire to be of the other gender; and they typically do not have a history of childhood cross-gender behaviors, which would be present in individuals with gender dysphoria. Individuals with a presentation that meets full criteria for transvestic disorder as well as gender dysphoria should be given both diagnoses.

Comorbidity

Transvestic disorder is often found in association with other paraphilias. The most frequently co-occurring paraphilias are fetishistic sexual interests or behavior and masochistic sexual interests or behavior. One particularly dangerous form of masochistic sexual interests or behavior, *autoerotic asphyxia*, is associated with transvestic sexual interests or behavior in a substantial proportion of fatal cases.

Other Specified Paraphilic Disorder

F65.89

This category applies to presentations in which symptoms characteristic of a paraphilic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the paraphilic disorders diagnostic class. The other specified paraphilic disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific paraphilic disorder. This is done by recording "other specified paraphilic disorder" followed by the specific reason (e.g., "zoophilia").

Examples of presentations that can be specified using the "other specified" designation include, but are not limited to, recurrent and intense sexual arousal involving *telephone scatologia* (obscene phone calls), *necrophilia* (corpses), *zoophilia* (animals), *coprophilia* (feces), *klismaphilia* (enemas), or *urophilia* (urine) that has been present for at least 6 months and causes marked distress or impairment in social, occupational, or other important areas of functioning. Other specified paraphilic disorder can be specified as in remission and/or as occurring in a controlled environment.

Unspecified Paraphilic Disorder

F65.9

This category applies to presentations in which symptoms characteristic of a paraphilic disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any of the disorders in the paraphilic disorders diagnostic class. The unspecified paraphilic disorder category is

used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific paraphilic disorder, and includes presentations in which there is insufficient information to make a more specific diagnosis.

Other Mental Disorders and Additional Codes

This chapter provides diagnostic codes for psychiatric presentations that are mental disorders (i.e., symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning) but that do not meet diagnostic requirements for any of the mental disorders in the prior Section II chapters. These codes allow for the documentation and coding of these otherwise unclassified mental disorders. This chapter also includes an additional code, "No diagnosis or condition," for situations in which the individual has been evaluated and it is determined that no mental disorder or condition is present.

The categories 1) other specified mental disorder due to another medical condition and 2) unspecified mental disorder due to another medical condition are for presentations for which it has been determined that the psychiatric symptoms (e.g., dissociative symptoms) are a direct physiological consequence of another medical condition but do not otherwise meet diagnostic criteria for any of the prior Section II mental disorders due to another medical condition. For the diagnosis of other specified or unspecified mental disorder due to another medical condition, it is necessary to code and list the medical condition first (e.g., B20 HIV disease), followed by the applicable code for either other specified or unspecified mental disorder due to another medical condition.

The categories 1) other specified mental disorder and 2) unspecified mental disorder are residual categories used when all of the following considerations are met: the psychiatric presentation is a mental disorder (i.e., the symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning); the presentation does not meet diagnostic criteria for any of the specific mental

disorders in Section II; neither does the presentation meet the definitional requirements of any of the other specified and unspecified mental disorder categories presented in Section II; and no other mental disorder diagnosis applies.

As is the case with other specified and unspecified categories throughout DSM-5, the other specified category is used when the clinician chooses to specify the specific reason that the presentation does not meet the criteria for any of the existing categories (e.g., other specified mental disorder due to complex partial seizures, with dissociative symptoms), and the unspecified category is used when the clinician chooses not to specify the reason.

Other Specified Mental Disorder Due to Another Medical Condition

F06.8

This category applies to presentations in which symptoms characteristic of a mental disorder due to another medical condition that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific mental disorder attributable to another medical

condition. The other specified mental disorder due to another medical condition category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific mental disorder attributable to another medical condition. This is done by recording the name of the disorder, with the specific etiological medical condition inserted in place of “another medical condition,” followed by the specific symptomatic manifestation that does not

meet the criteria for any specific mental disorder due to another medical condition. Furthermore, the diagnostic code for the specific medical condition must be listed immediately before the code for the other specified mental disorder due to another medical condition. For example, dissociative symptoms due to complex partial seizures would be coded and recorded as G40.209 complex partial seizures, F06.8 other specified mental disorder due to complex partial seizures, dissociative symptoms.

An example of a presentation that can be specified using the "other specified" designation is the following:

Dissociative symptoms: This includes symptoms occurring, for example, in the context of complex partial seizures.

Unspecified Mental Disorder Due to Another Medical Condition

F09

This category applies to presentations in which symptoms characteristic of a mental disorder due to another medical condition that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific mental disorder due to another medical condition. The unspecified mental disorder due to another medical condition category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific mental disorder due to another medical condition, and includes presentations for which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings). This is done by recording the name of the disorder, with the specific

etiological medical condition inserted in place of “another medical condition.” Furthermore, the diagnostic code for the specific medical condition must be listed immediately before the code for the unspecified mental disorder due to another medical condition. For example, dissociative symptoms due to complex partial seizures would be coded and recorded as G40.209 complex partial seizures, F09 unspecified mental disorder due to complex partial seizures.

Other Specified Mental Disorder

F99

This category applies to presentations in which symptoms characteristic of a mental disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any specific mental disorder. The other specified mental disorder category is used in situations in which the clinician chooses to communicate the specific reason that the presentation does not meet the criteria for any specific mental disorder. This is done by recording “other specified mental disorder” followed by the specific reason.

Unspecified Mental Disorder

F99

This category applies to presentations in which symptoms characteristic of a mental disorder that cause clinically significant distress or impairment in social, occupational, or other important areas of functioning predominate but do not meet the full criteria for any mental disorder. The unspecified mental disorder

category is used in situations in which the clinician chooses *not* to specify the reason that the criteria are not met for a specific mental disorder, and includes presentations for which there is insufficient information to make a more specific diagnosis (e.g., in emergency room settings).

Additional Codes

Z03.89 No Diagnosis or Condition

This code applies to situations in which the person has been evaluated and it is determined that no mental disorder or condition is present.

Medication-Induced Movement Disorders and Other Adverse Effects of Medication

Medication-induced movement disorders are included in Section II because of their frequent importance in 1) the management by medication of mental disorders or other medical conditions and 2) the differential diagnosis of mental disorders (e.g., anxiety disorder vs. medication-induced akathisia; malignant catatonia [a particularly severe and potentially life-threatening form of catatonia] vs. neuroleptic malignant syndrome; tardive dyskinesia vs. chorea). Although these movement disorders are labeled "medication induced," it is often difficult to establish the causal relationship between medication exposure and the development of the movement disorder, especially because some of these movement disorders also occur in the absence of medication exposure. The conditions and problems listed in this chapter are not mental disorders.

The term *neuroleptic* is becoming outdated because it highlights the propensity of antipsychotic medications to cause abnormal movements, and it is being replaced with the term *antipsychotic medications and other dopamine receptor blocking agents* in many contexts. Although newer antipsychotic medications may be less likely to cause some medication-induced movement disorders, those disorders still occur. Antipsychotic medications and other dopamine receptor blocking agents include so-called conventional, "typical," or first-generation antipsychotic agents (e.g., chlorpromazine, haloperidol, fluphenazine); "atypical" or second-generation antipsychotic agents (e.g., clozapine, risperidone, olanzapine, quetiapine); certain dopamine receptor blocking drugs used in the treatment of symptoms such as nausea and gastroparesis (e.g., prochlorperazine,

promethazine, trimethobenzamide, thiethylperazine, metoclopramide); and amoxapine, which is indicated for the treatment of depression.

Medication-Induced Parkinsonism

G21.11 Antipsychotic Medication– and Other Dopamine Receptor Blocking Agent–Induced Parkinsonism

G21.19 Other Medication-Induced Parkinsonism

Medication-induced parkinsonism (MIP), the second most common cause of parkinsonism after Parkinson's disease, is associated with significant morbidity, disability, and treatment nonadherence, particularly in individuals with psychiatric disorders. Because early recognition is important, any new case of parkinsonism should prompt a thorough medication history, which is essential for diagnosis of MIP. A temporal relationship between medication initiation and onset of parkinsonism should be evident. A host of agents that may be prescribed in individuals with psychiatric disorders may also induce parkinsonism, but MIP is most often seen upon exposure to antipsychotic medications that block dopamine D_2 receptors. MIP occurs at higher rates with antipsychotics that have higher potency for the dopamine D_2 receptor, such as haloperidol, fluphenazine, and risperidone, but there are no

differences in the clinical features of parkinsonism between first- and second-generation antipsychotics.

Other medications that can cause MIP include calcium channel antagonists (e.g., flunarizine, cinnarizine), dopamine depleters (e.g., reserpine, tetrabenazine), antiepileptics (e.g., phenytoin, valproate, levetiracetam), antidepressants (e.g., selective serotonin reuptake inhibitors, monoamine oxidase inhibitors), lithium, chemotherapeutic drugs (e.g., cystosine arabinoside, cyclophosphamide, vincristine, doxorubicin, paclitaxel, etoposide), and immunosuppressants (e.g., cyclosporine,

tacrolimus). Toxins (e.g., 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine [MPTP], organophosphate pesticides, manganese, methanol, cyanide, carbon monoxide, and carbon disulfide) may also cause MIP.

The time course for development of MIP varies. Usually, MIP develops a few weeks after starting or raising the dose of a medication known to cause parkinsonism or after reducing an antiparkinsonian medication (e.g., an anticholinergic agent) that is being used to treat or prevent medication-induced dystonia or parkinsonian symptoms. However, MIP may also develop rapidly after starting or raising the dose of a medication or have an insidious onset after many months of exposure. With antipsychotic medications or other dopamine receptor blocking agents, MIP typically develops 2–4 weeks after starting the medication and usually by 3 months. Mainly with calcium channel blockers, a second peak of symptom onset is reported after about 1 year.

Reported rates of MIP are affected by absence of standard diagnostic criteria, incorrect diagnosis or misattribution of MIP signs to Lewy body disease (e.g., Parkinson's disease), or a psychiatric condition, and overall lack of recognition, especially in milder cases. It is estimated that at least 50% of outpatients receiving long-term antipsychotic treatment with typical agents develop parkinsonian signs or symptoms at some point in their course of treatment.

There are no clinical characteristics that distinguish MIP reliably from Parkinson's disease. Because motor signs and symptoms in Parkinson's disease begin unilaterally and progress asymmetrically, the subacute onset of bilateral parkinsonism within weeks of starting an antipsychotic or other MIP-causing agent is highly suggestive for MIP. Parkinsonian signs are often symmetric in MIP, but asymmetric patterns are not uncommon and should not exclude a diagnosis of MIP. In addition, the course and presentation of parkinsonism should not be better accounted for by psychiatric phenomena, such as catatonia, negative symptoms of schizophrenia, or psychomotor retardation in a major depressive episode; other nonparkinsonian medication-induced movement disorders; another neurological or medical condition (e.g., Parkinson's disease, Wilson's disease); or antipsychotic-exacerbated Parkinson's disease.

In MIP, rigidity and bradykinesia are more often present, whereas tremor is somewhat less common and may be absent. Parkinsonian tremor,

also referred to as a “pill-rolling tremor,” is a steady, rhythmic oscillatory movement (3–6 cycles per second) that is apparent at rest and is typically slower than other tremors. It may be intermittent, unilateral or bilateral, or dependent on limb position (i.e., positional tremor). The tremor may involve the limbs, head, jaw, mouth, lip (“rabbit syndrome”), or tongue. As it is present at rest, the tremor can be suppressed, especially when the individual attempts to perform a task with the tremulous limb. Individuals may describe the tremor as “shaking” and report that it may worsen with anxiety, stress, or fatigue.

Parkinsonian rigidity is experienced as an involuntary stiffness and inflexibility of the muscles of the limbs, shoulders, neck, or trunk. Rigidity is evaluated by assessing muscle tone, or the amount of resistance present when the examiner moves a limb (and stretches the muscles) passively around a joint. In lead-pipe rigidity, increased tone is constant throughout range of motion (in contrast to the clasp-knife rigidity spasticity). Cogwheel rigidity is thought to represent a tremor superimposed on rigidity. Most common in the wrists and elbows, it is experienced as a rhythmic, ratchet-like resistance (cogwheeling) when the muscles are passively moved around a joint. Individuals with parkinsonian rigidity may

complain of generalized muscle tenderness or stiffness, tightness in their limbs, muscle or joint pain, body aching, or lack of coordination.

Bradykinesia and akinesia are observable states of decreased or absent spontaneous motor activity, respectively. There is global slowing as well as slowness in initiating and executing movements. Everyday behaviors (e.g., grooming) can be difficult to perform normally and may be reduced. Individuals may complain of listlessness, lack of spontaneity and drive, or fatigue. Parkinsonian rigidity and bradykinesia manifest as gait abnormalities, including decreased stride length, arm swing, or overall spontaneity of walking. Other signs include a hunched posture with bent-over neck and stooped shoulders, a staring facial expression, and small shuffling steps. Drooling can arise as a result of reduced pharyngeal motor activity and swallowing, but because of anticholinergic properties of these

medications, it may be less common in antipsychotic-induced parkinsonism as compared with other medications that cause MIP.

MIP is associated with increased gait dysfunction, falls, and nursing home placement. As such, MIP is a serious iatrogenic movement disorder in older individuals that warrants recognition and early diagnosis. Associated behavioral symptoms may include depression and worsening of negative signs of schizophrenia. Other parkinsonian signs and symptoms include small handwriting (micrographia), reduced motor dexterity, hypophonia, decreased gag reflex, dysphagia, postural instability, reduced facial expression and blinking, and seborrhea. When parkinsonism is associated with severe decreased motor activity, medical complications of parkinsonism include contractures, bedsores, pulmonary emboli, urinary incontinence, aspiration pneumonia, weight loss, and hip fractures.

Consistent risk factors are female gender, older age, cognitive impairment, other concurrent neurological conditions, HIV infection, family history of Parkinson's disease, and severe psychiatric disease. MIP secondary to antipsychotic use is also reported in children. The risk of MIP is reduced if individuals are taking anticholinergic medications.

Differential Diagnosis

Parkinson's disease and Parkinson's-plus conditions such as multiple system atrophy, progressive supranuclear palsy, and Wilson's disease are distinguished from MIP by their other signs and symptoms that accompany parkinsonism. For example, Parkinson's disease is suggested by evidence of three or more cardinal features of Parkinson's disease (e.g., resting tremor, rigidity, bradykinesia, postural instability), hyposmia, sleep disturbances such as rapid eye movement (REM) sleep behavior disorder, and urinary and other autonomic symptoms common to Parkinson's disease. These features are less likely to be present in MIP. Individuals with primary neurological causes of parkinsonism are also susceptible to worsening symptoms if treated with medications causing MIP.

Nonparkinsonian tremors tend to be finer (e.g., smaller amplitude) and faster (10 cycles per second) and worsen on intention (e.g., when reaching out to grab an object). With substance withdrawal, there is usually associated hyperreflexia and increased autonomic signs. In cerebellar

disease, tremor worsens on intention and may be associated with nystagmus, ataxia, or scanning speech. Choreiform movements associated with tardive dyskinesia lack the steady rhythmicity of a parkinsonian tremor. Strokes and other central nervous system lesions can cause focal neurological signs or immobility from flaccid or spastic paralysis, which is characterized by decreased muscle strength and increased tone on passive movement that gives way with further pressure (i.e., clasp-knife rigidity). This contrasts with the lead-pipe rigidity and normal muscle strength in MIP.

Diagnostic alternatives to MIP are also suggested by a family history of an inherited neurological condition, rapidly progressive parkinsonism not accounted for by recent psychopharmacological changes, or presence of focal neurological signs (e.g., frontal release signs, cranial nerve abnormalities, a positive Babinski sign). Neuroleptic malignant syndrome involves severe akinesia and rigidity, but also characteristic physical and laboratory findings (e.g., fever, increased creatine phosphokinase).

Psychomotor slowing, inactivity, and apathy seen in major depressive disorder can be indistinguishable from the motor slowness or akinesia of MIP, but major depressive disorder is more likely to include vegetative signs (e.g., early-morning awakening), hopelessness, and despair. Negative symptoms of schizophrenia, catatonia associated with schizophrenia, or mood disorders with catatonic features may also be difficult to distinguish from medication-induced akinesia. Rigidity may also manifest in psychotic disorders, delirium, major neurocognitive disorder, anxiety disorders, and functional neurological symptom disorder (conversion disorder). In parkinsonian rigidity, resistance to passive motion is constant through the full range of motion, whereas it is inconsistent in psychiatric disorders or other neurological conditions presenting with rigidity. In general, the constellation of associated physical signs on examination and symptoms associated with the tremor, rigidity, and bradykinesia of parkinsonism helps distinguish MIP-related rigidity and bradykinesia from other primary psychiatric causes of rigidity and decreased movement.

Neuroleptic Malignant Syndrome

G21.0 Neuroleptic Malignant Syndrome

Individuals with neuroleptic malignant syndrome have generally been exposed to a dopamine antagonist within 72 hours prior to symptom development. Hyperthermia (>100.4°F or >38.0°C on at least two occasions, measured orally), associated with profuse diaphoresis, is a distinguishing feature of neuroleptic malignant syndrome, setting it apart from other neurological side effects of antipsychotic medications and other dopamine receptor blocking agents. Extreme elevations in temperature, reflecting a breakdown in central thermoregulation, are more likely to support the diagnosis of neuroleptic malignant syndrome. Generalized rigidity, described as “lead pipe” in its most severe form and usually unresponsive to antiparkinsonian agents, is a cardinal feature of the disorder and may be associated with other neurological symptoms (e.g., tremor, sialorrhea, akinesia, dystonia, trismus, myoclonus, dysarthria, dysphagia, rhabdomyolysis). Creatine kinase elevation of at least four times the upper limit of normal is commonly seen. Changes in mental status, characterized by delirium or altered consciousness ranging from stupor to coma, are often an early sign of neuroleptic malignant syndrome. Affected individuals may appear alert but dazed and unresponsive, consistent with catatonic stupor. Autonomic activation and instability—manifested by tachycardia (rate > 25% above baseline), diaphoresis, blood pressure elevation (systolic or diastolic ≥ 25% above baseline) or fluctuation (≥ 20 mmHg diastolic change or ≥25 mmHg systolic change within 24 hours), urinary incontinence, and pallor—may be seen at any time but provide an early clue to the diagnosis. Tachypnea (rate > 50% above baseline) is common, and respiratory distress —resulting from metabolic acidosis, hypermetabolism, chest wall restriction, aspiration pneumonia, or pulmonary emboli—can occur and lead to sudden respiratory arrest.

Although several laboratory abnormalities are associated with neuroleptic malignant syndrome, no single abnormality is specific to the diagnosis. Individuals with neuroleptic malignant syndrome may have leukocytosis, metabolic acidosis, hypoxia, decreased serum iron concentrations, and elevations in serum muscle enzymes and

catecholamines. Findings from cerebrospinal fluid analysis and neuroimaging studies are generally normal, whereas electroencephalography shows generalized slowing. Autopsy findings in fatal cases have been nonspecific and variable, depending on complications.

Evidence from database studies suggests incidence rates for neuroleptic malignant syndrome of 0.01%–0.02% among individuals treated with antipsychotics. A population-based study conducted in Hong Kong found an incidence risk of 0.11% in individuals treated with antipsychotic medication.

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The temporal progression of signs and symptoms provides important clues to the diagnosis and prognosis of neuroleptic malignant syndrome. Alteration in mental status and other neurological signs typically precede systemic signs. The onset of symptoms varies from hours to days after drug initiation. Some cases develop within 24 hours after drug initiation, most within the first week, and virtually all cases within 30 days. Once the syndrome is diagnosed and oral antipsychotic drugs and other dopamine receptor blocking agents are discontinued, neuroleptic malignant syndrome is self-limited in most cases. The mean recovery time after drug discontinuation is 7–10 days, with most individuals recovering within 1 week and nearly all within 30 days. The duration may be prolonged when long-acting antipsychotic medications are implicated. There have been reports of individuals in whom residual neurological signs persisted for weeks after the acute hypermetabolic symptoms resolved. Total resolution of symptoms can be obtained in most cases of neuroleptic malignant syndrome; however, fatality rates of 10%–20% have been reported when the disorder is not recognized. Although many individuals do not experience a recurrence of neuroleptic malignant syndrome when rechallenged with antipsychotic medication, some do, especially when antipsychotic medications are reinstituted soon after an episode.

Neuroleptic malignant syndrome is a potential risk in any individual after administration of an antipsychotic medication or other dopamine receptor blocking agent. It is not specific to any neuropsychiatric diagnosis and may occur in persons without a diagnosable mental disorder who

receive dopamine antagonists. Clinical, systemic, and metabolic factors associated with a heightened risk of neuroleptic malignant syndrome include agitation, exhaustion, dehydration, and iron deficiency. A prior episode associated with antipsychotic medication and other dopamine receptor blocking agents has been described in 15%–20% of index cases, suggesting underlying vulnerability in some individuals; however, genetic findings based on neurotransmitter receptor polymorphisms have not been replicated consistently.

Nearly all antipsychotic medication and other dopamine receptor blocking agents have been associated with neuroleptic malignant syndrome, although high-potency antipsychotics pose a greater risk compared with low-potency agents and atypical antipsychotics. Partial or milder forms may be associated with newer antipsychotics, but neuroleptic malignant syndrome varies in severity even with older drugs. Dopamine receptor blocking agents used in medical settings (e.g., metoclopramide, prochlorperazine) have also been implicated. Parenteral administration routes, rapid titration rates, and higher total drug dosages have been associated with increased risk; however, neuroleptic malignant syndrome usually occurs within the therapeutic dosage range of antipsychotic medications and other dopamine receptor blocking agents.

Differential Diagnosis

Neuroleptic malignant syndrome should be distinguished from other serious neurological or medical conditions, including central nervous system infections, inflammatory or autoimmune conditions, status epilepticus, subcortical structural lesions, and systemic conditions (e.g., pheochromocytoma, thyrotoxicosis, tetanus, heat stroke).

Neuroleptic malignant syndrome also should be distinguished from similar syndromes resulting from the use of other substances or medications, such as serotonin syndrome; parkinsonian hyperthermia syndrome following abrupt discontinuation of dopamine agonists; alcohol or sedative withdrawal; malignant hyperthermia occurring during anesthesia; hyperthermia associated with misuse of stimulants and hallucinogens; and atropine poisoning from anticholinergics.

In rare instances, individuals with schizophrenia or a mood disorder may present with malignant catatonia, which may be indistinguishable from neuroleptic malignant syndrome. Some investigators consider neuroleptic malignant syndrome to be a drug-induced form of malignant catatonia.

Medication-Induced Acute Dystonia

G24.02 Medication-Induced Acute Dystonia

The essential feature of medication-induced acute dystonia is sustained abnormal muscle contractions (increased muscle tone) and postures that develop in association with use of a medication known to cause acute dystonia. Any medication that blocks dopamine D_2-like receptors can induce an acute dystonic reaction (ADR). Most commonly, ADRs occur after exposure to antipsychotics and antiemetic and promotility agents. A variety of other medication classes are also reported to have induced ADRs, including selective serotonin reuptake inhibitors, cholinesterase inhibitors, opioids, and methylphenidate.

Dystonic reactions vary greatly in severity and location and can be focal, segmented, or generalized. They most often affect head and neck muscles, but can extend to upper and lower limbs or trunk. A common presentation is acute oro-mandibular (jaw) dystonia involving the tongue and mouth with tongue protrusion, or gaping or grimacing postures that can impair speech (dysarthria) and swallowing (dysphagia) and may evolve into frank trismus (lockjaw). Involvement of ocular muscles (oculogyric crisis) manifests as involuntary forced and sustained conjugate deviations of eyes upward, downward, or sideways that can last minutes to hours. Blepharospasm can also occur. Cervical (neck) dystonia presents as abnormal forward, backward, lateral, or twisting positions of the head and neck in relation to the body (e.g., antecollis, retrocollis, laterocollis, and torticollis). Focal limb dystonia, generally more distal than proximal, Pisa syndrome (lateral bending of the trunk with a tendency to lean to one side), and back arching that may evolve into opisthotonos (backward arching of head, neck, and spine) can also occur. Acute laryngeal dystonia is life-

threatening, causing airway obstruction, and manifests as a “clutching of the throat,” stridor, dysphonia, dysphagia, dyspnea, and respiratory distress from the medication effects on vocal cords and laryngeal muscles.

At least 50% of individuals develop ADR signs or symptoms within 24–48 hours of starting or rapidly raising the dose of antipsychotic medication or other dopamine receptor blocking agent or of reducing a medication being used to treat or prevent acute extrapyramidal symptoms (e.g., anticholinergic agents). Approximately 90% of affected individuals have onset of ADRs within 5 days. The symptoms must not be better accounted for by a mental disorder (e.g., catatonia) and must not be due to a primary neurological or other medical condition, or a tardive medication-induced movement disorder.

Fear and anxiety often accompany ADRs given their intense nature, inability of the individual to control or stop the movements, and, when present, difficulty breathing, speaking, or swallowing. Some individuals experience pain or cramps in affected muscles. Individuals who are unaware of the possibility of developing a medication-induced dystonia can be especially distressed, increasing the likelihood of subsequent medication nonadherence. Thought disorder, delusions, or mannerisms in an individual with psychosis may cause the affected individual or others to mistakenly regard his or her dystonic symptoms as a feature of the psychiatric condition, which could lead to increased doses of the causative medication. The risk of developing ADRs is greatest in children and in adults younger than age 40 with psychosis, with a greater incidence in males than females in both children and adults. Other risk factors for developing ADRs include prior dystonic reactions to antipsychotic medications or other dopamine receptor blocking agents and use of high-potency typical antipsychotic medications.

Differential Diagnosis

It is important to distinguish between medication-induced ADRs and other causes of dystonia, especially in individuals being treated with antipsychotic or other dopamine receptor blocking medications. A primary neurological or other medical condition is evident

based on the time course and evolution of the dystonic phenomena (e.g., dystonia precedes exposure to the antipsychotic medication or progresses in the absence of change in medication) and, possibly, other evidence of focal neurological signs. Idiopathic focal or segmental dystonias usually persist for several days or weeks independent of medication. A family history of dystonia may also be present. Tardive dystonia secondary to medication exposure, including antipsychotic medication or other dopamine receptor blocking agents, does not have acute onset and may become evident when the dose of an antipsychotic medication is lowered. Other neurological conditions (e.g., epileptic seizures, viral and bacterial infections, trauma, space-occupying lesions in the peripheral or central nervous system) and endocrinopathies (e.g., hypoparathyroidism) can also produce symptoms (e.g., tetany) that resemble a medication-induced acute dystonia. Other diagnoses that mimic an acute medication-induced dystonia include anaphylaxis, tardive laryngeal dystonia, and respiratory dyskinesia. Neuroleptic malignant syndrome can produce dystonia but differs in that it is also accompanied by fever and generalized rigidity.

Catatonia associated with a mood disorder or schizophrenia can be distinguished by the temporal relationship between the symptoms and the exposure to antipsychotic treatment (e.g., dystonia preceding exposure to antipsychotic medication) and response to pharmacological intervention (e.g., no improvement after lowering of dose of the antipsychotic medication or in response to anticholinergic administration). Furthermore, individuals with medication-induced acute dystonia are generally distressed about the dystonic reaction and usually seek intervention. In contrast, individuals with the retarded type of catatonia are typically mute and withdrawn and do not express subjective distress about their condition.

Medication-Induced Acute Akathisia

G25.71 Medication-Induced Acute Akathisia

The essential features of medication-induced acute akathisia are subjective complaints of restlessness and at least one of the following observed

movements: fidgety movements or swinging of the legs while seated, rocking from foot to foot or “walking on the spot” while standing, pacing to relieve the restlessness, or an inability to sit or stand still for at least several minutes. Individuals experiencing the most severe form of medication-induced acute akathisia may be unable to maintain any position for more than a few seconds. The subjective complaints include a sense of inner restlessness, most often in the legs; a compulsion to move one’s legs; distress if one is asked not to move one’s legs; and dysphoria and anxiety. The symptoms typically occur within 4 weeks of initiating or increasing the dose of a medication that can cause akathisia, which includes antipsychotic medications and other dopamine receptor blocking agents, tricyclic antidepressants, selective serotonin reuptake inhibitors, dopamine agonists, and calcium channel blockers, and can occasionally follow the reduction of medication used to treat or prevent acute extrapyramidal symptoms (e.g., anticholinergic agents). The symptoms are not better explained by a mental disorder (e.g., schizophrenia, substance withdrawal, agitation from a major depressive or manic episode, hyperactivity in attention-deficit/hyperactivity disorder) and are not due to a neurological or other medical condition (e.g., Parkinson’s disease, iron-deficiency anemia).

The subjective distress resulting from akathisia is significant and can lead to noncompliance with antipsychotic or antidepressant treatment. Akathisia may be associated with dysphoria, irritability, aggression, or suicide attempts. Worsening of psychotic symptoms or behavioral dyscontrol may lead to an increase in medication dose, which may exacerbate the problem. Akathisia can develop very rapidly after initiating or increasing the causative medication. The development of akathisia appears to be dose dependent and to be more frequently associated with particular high-potency antipsychotic medications or drugs with higher affinity for central dopamine receptors. Acute akathisia tends to persist for

as long as the causative medication is continued, although the intensity may fluctuate over time. The reported prevalence of akathisia among individuals receiving antipsychotic medication or other dopamine receptor blocking agents has varied widely (20%–75%). Variations in reported prevalence

may be attributable to a lack of consistency in the definition, antipsychotic prescribing practices, study design, and the demographics of the population being studied.

Differential Diagnosis

Medication-induced acute akathisia may be clinically indistinguishable from syndromes of restlessness due to certain neurological or other medical conditions, and to agitation presenting as part of a mental disorder (e.g., a manic episode). The akathisia of Parkinson's disease and iron-deficiency anemia is phenomenologically similar to medication-induced acute akathisia. The frequently abrupt appearance of restlessness soon after initiation or increase in medication usually distinguishes medication-induced acute akathisia.

Serotonin-specific reuptake inhibitor antidepressant medications may produce akathisia that appears to be identical in phenomenology and treatment response to akathisia induced by antipsychotic medication or other dopamine receptor blocking agents. Tardive dyskinesia also often has a component of generalized restlessness that may coexist with akathisia in an individual receiving antipsychotic medications or other dopamine blocking agents. Antipsychotic medication and other dopamine blocking agent–induced acute akathisia is differentiated from antipsychotic medication and other dopamine blocking agent–induced tardive dyskinesia by the nature of the movements and their relationship to the initiation of medication. The time course of symptomatic presentation relative to medication dose changes may aid in this distinction. An increase in antipsychotic medication will often exacerbate akathisia, whereas it often temporarily relieves the symptoms of tardive dyskinesia.

Medication-induced acute akathisia should be distinguished from symptoms that are better accounted for by a mental disorder. Individuals with depressive episodes, manic episodes, generalized anxiety disorder, schizophrenia spectrum and other psychotic disorders, attention-deficit/hyperactivity disorder, major neurocognitive disorder, delirium, substance intoxication (e.g., with cocaine), or substance withdrawal (e.g., from an opioid) may also display agitation that is difficult to distinguish from akathisia. Some of these individuals are able to differentiate akathisia

from the anxiety, restlessness, and agitation characteristic of a mental disorder by their experience of akathisia as being different from previously experienced feelings. Other evidence that restlessness or agitation may be better accounted for by a mental disorder includes the onset of agitation prior to exposure to the causative medication, absence of increasing restlessness with increasing doses of the causative medication, and absence of relief with pharmacological interventions (e.g., no improvement after decreasing the dose of the causative medication or treatment with another medication intended to treat the akathisia).

Tardive Dyskinesia

G24.01 Tardive Dyskinesia

The essential features of tardive dyskinesia are abnormal, involuntary movements of the tongue, jaw, trunk, or extremities that develop in association with the use of medications that block postsynaptic dopamine receptors, such as first- and second-generation antipsychotic medications and other medications such as metoclopramide for gastrointestinal disorders. The movements are present over a period of at least 4 weeks and may be choreiform (rapid, jerky, nonrepetitive), athetoid (slow, sinuous, continual), or semirhythmic (e.g., stereotypies) in nature; however, the movements are distinctly different from the rhythmic

(3-6 Hz) tremors commonly seen in medication-induced parkinsonism. Signs or symptoms of tardive dyskinesia develop during exposure to the antipsychotic medication or other dopamine blocking agent, or within 4 weeks of withdrawal from an oral agent (or within 8 weeks of withdrawal from a long-acting injectable agent). There must be a history of the use of the offending agent for at least 3 months (or 1 month in individuals age 60 years or older). Although a large number of epidemiological studies have established the etiological relationship between dopamine blocking drug use and tardive dyskinesia, any dyskinesia in an individual who is receiving antipsychotic medication is not necessarily tardive dyskinesia.

Abnormal orofacial movements are the most obvious manifestations of tardive dyskinesia and have been observed in most individuals afflicted with tardive dyskinesia; however, approximately one-half can have limb involvement, and up to one-quarter can have axial dyskinesia of the neck, shoulders, or trunk. Involvement of other muscle groups (e.g., pharyngeal, diaphragm, abdominal) may occur but is uncommon, especially in the absence of dyskinesia of the orofacial region, limbs, or trunk. Limb or truncal dyskinesia without orofacial involvement may be more common in younger individuals, whereas orofacial dyskinesias are typical in older individuals.

The symptoms of tardive dyskinesia tend to be worsened by stimulants, antipsychotic medication withdrawal, and anticholinergic medications (such as benztropine, commonly used to manage medication-induced parkinsonism) and may be transiently worsened by emotional arousal, stress, and distraction during voluntary movements in unaffected parts of the body. The abnormal movements of dyskinesia are transiently reduced by relaxation and by voluntary movements in affected parts of the body. They are generally absent during sleep. Dyskinesia may be suppressed, at least temporarily, by increased doses of antipsychotic medication.

The overall prevalence of tardive dyskinesia in individuals who have received long-term antipsychotic medication treatment ranges from 20% to 30%. The overall incidence among younger individuals ranges from 3% to 5% per year. Middle-age and elderly individuals appear to develop tardive dyskinesia more often, with prevalence figures reported up to 50% and an incidence of 25%–30% after an average of 1 year’s cumulative exposure to antipsychotic medication. Prevalence also varies depending on setting, with tardive dyskinesia tending to be more common among chronically institutionalized individuals. Variations in reported prevalence may be attributable to a lack of consistency in the definition of tardive dyskinesia, antipsychotic prescribing practices, study design, and the demographics of the population being studied.

There is no obvious gender difference in the susceptibility to tardive dyskinesia, although the risk may be somewhat greater in postmenopausal women. Greater cumulative amounts of antipsychotic medications and early development of acute extrapyramidal side effects (such as medication-induced parkinsonism) are two of the most consistent risk factors for tardive

dyskinesia. Mood disorders (especially major depressive disorder), neurological conditions, and alcohol use disorder have also been found to be risk factors in some groups of individuals. Second-generation antipsychotics are associated with a somewhat lower incidence of tardive dyskinesia compared with first-generation antipsychotics, but the difference is not as large as once thought, especially when the dose of the first-generation antipsychotic is taken into account; the most important risk factors are age and cumulative exposure.

Onset of tardive dyskinesia may occur at any age and is almost always insidious. The signs are typically minimal to mild at onset and escape notice except by a keen observer. In many cases, tardive dyskinesia is objectively mild but, although it has been thought of as a cosmetic problem, can be associated with significant distress and social avoidance. In severe cases, it may be associated with medical complications (e.g., ulcers in cheeks and tongue; loss of teeth; macroglossia; difficulty in walking, swallowing, or breathing; muffled speech; weight loss; depression; suicidal ideation). In older individuals there is a greater

likelihood that tardive dyskinesia may become more severe or more generalized with continued antipsychotic medication use. When antipsychotic medications are discontinued, some individuals experience symptom improvement over time; however, for others tardive dyskinesia can be enduring.

Differential Diagnosis

It is imperative to distinguish medication-induced parkinsonism from tardive dyskinesia because the treatments commonly used to manage medication-induced parkinsonism (i.e., anticholinergic medications) may worsen the abnormal motor movements associated with tardive dyskinesia. Moreover, treatments used to manage tardive dyskinesia (i.e., VMAT2 inhibitors) may worsen the symptoms of medication-induced parkinsonism.

Dyskinesia that emerges during withdrawal from an antipsychotic medication or other dopamine receptor blocking agent may remit with

continued withdrawal from the medication. If the dyskinesia persists for at least 4 weeks, a diagnosis of tardive dyskinesia may be warranted. Tardive dyskinesia must be distinguished from other causes of orofacial and body dyskinesia. These conditions include Huntington's disease, Wilson's disease, Sydenham's (rheumatic) chorea, systemic lupus erythematosus, thyrotoxicosis, heavy metal poisoning, ill-fitting dentures, dyskinesia due to other medications such as L-dopa or bromocriptine, and spontaneous dyskinesias. Factors that may be helpful in making the distinction are evidence that the symptoms preceded the exposure to the antipsychotic medication or other dopamine receptor blocking agent or that other focal neurological signs are present. It should be noted that other movement disorders may coexist with tardive dyskinesia. Because spontaneous dyskinesia can occur in more than 5% of individuals and is also more common in elderly persons, it may be difficult to prove that antipsychotic medications produced tardive dyskinesia in a given individual. Tardive dyskinesia must be distinguished from symptoms that are due to a medication-induced acute movement disorder (e.g., medication-induced parkinsonism, acute dystonia, acute akathisia). Acute dystonia and acute akathisia can develop quickly within hours to days, and medication-induced parkinsonism develops within weeks of initiating or increasing the dose of an antipsychotic medication or other dopamine receptor blocking agent (or reducing the dose of a medication used to treat the acute extrapyramidal symptoms). Tardive dyskinesia, on the other hand, generally develops after more prolonged exposure to antipsychotic medication (months to years) and can appear after the withdrawal of antipsychotic medication; the minimum exposure history required for the diagnosis of tardive dyskinesia is antipsychotic medication use for at least 3 months (or 1 month in middle-age and elderly individuals).

Tardive Dystonia Tardive Akathisia

G24.09 Tardive Dystonia

G25.71 Tardive Akathisia

This category is for tardive syndromes involving other types of movement problems, such as dystonia or akathisia, which are distinguished by their late emergence in the course of treatment and their potential persistence for months to years, even in the face of discontinuation of an antipsychotic medication or other dopamine receptor blocking agent or dosage reduction.

Medication-Induced Postural Tremor

G25.1 Medication-Induced Postural Tremor

The essential feature of this condition is a fine tremor occurring during attempts to maintain a posture, which develops in association with the use of medication. Medications with which such a tremor may be associated include lithium, β-adrenergic medications (e.g., isoproterenol), stimulants (e.g., amphetamine), dopaminergic medications, anticonvulsant medications (e.g., valproic acid), antidepressant medications, and methylxanthines (e.g., caffeine, theophylline). The tremor is a regular, rhythmic oscillation of the limbs (most commonly hands and fingers), head, mouth, or tongue, most commonly with a frequency of between 8 and 12 cycles per second. It is most easily observed when the affected body part is held in a sustained posture (e.g., hands outstretched, mouth held open). The tremor may worsen in severity when the affected body part is moved intentionally (kinetic or action tremor). When an individual describes a tremor that is consistent with postural tremor but the clinician does not directly observe the tremor, it may be helpful to try to re-create the situation in which the tremor occurred (e.g., drinking from a cup and saucer).

Most available information concerns lithium-induced tremor. Lithium tremor is a common, usually benign, and well-tolerated side effect of therapeutic doses. However, it may cause social embarrassment, occupational difficulties, and noncompliance in some individuals. As serum lithium levels approach toxic levels, the tremor may become coarser and be accompanied by muscle twitching, fasciculations, or ataxia. Nontoxic lithium tremor may improve spontaneously over time. A variety of factors may increase the risk of lithium tremor (e.g., increasing age, high serum

lithium levels, concurrent antidepressant or antipsychotic medication or another dopamine receptor blocking agent, excessive caffeine intake, personal or family history of tremor, presence of alcohol use disorder, and associated anxiety). The frequency of complaints about tremor appears to decrease with duration of lithium treatment. Factors that may exacerbate the tremor include anxiety, stress, fatigue, hypoglycemia, thyrotoxicosis, pheochromocytoma, hypothermia, and alcohol withdrawal. Tremor can also be an early feature of serotonin syndrome.

Differential Diagnosis

Medication-induced postural tremor should be distinguished from a preexisting tremor that is not caused by the effects of a medication. Factors that help to establish that the tremor was preexisting include its temporal relationship to the initiation of medication, lack of correlation with serum levels of the medication, and persistence after the medication is discontinued. If a preexisting, nonpharmacologically induced tremor is present (e.g., essential tremor) that worsens with medication, such a tremor would not be considered to be medication-induced postural tremor. The factors described above that may contribute to the severity of a medication-induced postural tremor (e.g., anxiety, stress, fatigue, hypoglycemia, thyrotoxicosis, pheochromocytoma, hypothermia, alcohol withdrawal) may also be a cause of tremor independent of the medication.

Medication-induced postural tremor is not diagnosed if the tremor is better accounted for by medication-induced parkinsonism. A medication-induced postural tremor is usually absent at rest and intensifies when the affected part is brought into action or held in a sustained position. In contrast, the tremor related to medication-induced parkinsonism is usually lower in frequency (3–6 Hz), worse at rest, and suppressed during intentional movement and usually occurs in association with other symptoms of medication-induced parkinsonism (e.g., akinesia, rigidity).

G25.79 Other Medication-Induced Movement Disorder

This category is for medication-induced movement disorders not captured by any of the specific disorders listed earlier. Examples include 1) presentations resembling neuroleptic malignant syndrome that are associated with medications other than antipsychotic medications and other dopamine receptor blocking agents and 2) other medication-induced tardive conditions.

Antidepressant Discontinuation Syndrome

T43.205A Initial encounter

T43.205D Subsequent encounter

T43.205S Sequelae

Discontinuation symptoms may occur following treatment with all types of antidepressants. The incidence of this syndrome depends on the dosage and half-life of the medication being taken, as well as the rate at which the medication is tapered. Short half-life medications that are abruptly discontinued (or when the dose is significantly reduced) rather than tapered gradually may pose the greatest risk. The short-acting antidepressants paroxetine and venlafaxine are the agents most commonly associated with discontinuation symptoms. Antidepressant discontinuation syndrome may occur in the context of intermittent non-adherence to treatment and therefore may be irregularly present in some individuals who have not actually stopped taking the medication. This is especially true for very short half-life medications (e.g., venlafaxine). By contrast, long half-life medications like fluoxetine seldom produce significant discontinuation effects.

Unlike withdrawal syndromes associated with opioids, alcohol, and other substances, antidepressant discontinuation syndrome has no pathognomonic symptoms. Instead, the symptoms tend to be vague and variable. Symptoms typically begin 2–4 days after the last dose of the antidepressant. For selective serotonin reuptake inhibitors, symptoms such as dizziness, tinnitus, “electric shock”–like sensations, insomnia, and acute

anxiety are described. The antidepressant use before discontinuation must not have incurred hypomania or mixed state (i.e., there should be confidence that the discontinuation syndrome is not the result of fluctuations in mood stability associated with the previous treatment). For the tricyclic antidepressants, sudden discontinuation has been associated with gastrointestinal symptoms (cramping—reflecting cholinergic overactivity after stopping an anticholinergic tricyclic antidepressant) as well as rebound hypomania.

The antidepressant discontinuation syndrome is based solely on pharmacological factors and is not related to the reinforcing effects of an antidepressant. Unlike the discontinuation of substances with reinforcing effects like opioids, drug craving does not occur. Also, when a stimulant is used to augment an antidepressant, abrupt cessation may result in stimulant withdrawal symptoms (see “Stimulant Withdrawal” in the chapter “Substance-Related and Addictive Disorders”) rather than the antidepressant discontinuation syndrome described here.

The prevalence of antidepressant discontinuation syndrome is unknown but is thought to vary according to any of the following factors: the dosage before discontinuation, the half-life (i.e., occurring more commonly with short half-life medications) and receptor-binding affinity of the medication (e.g., more likely to occur with serotonin reuptake inhibitors), and possibly the individual’s genetically influenced rate of metabolism for this

medication. Therefore, discontinuation reactions occur more frequently with short half-life medications, but may also be influenced by rapid or ultrarapid metabolizer status of cytochrome enzymes that metabolize the antidepressant.

Because longitudinal studies are lacking, little is known about the clinical course of antidepressant discontinuation syndrome. Symptoms appear to abate over time with very gradual dosage reductions. Symptoms are usually short-lived, lasting no more than 2 weeks, and are seldom present more than 3 weeks after discontinuation.

Differential Diagnosis

The differential diagnosis of antidepressant discontinuation syndrome includes a relapse of the disorder for which the medication was prescribed (e.g., depression or panic disorder), somatic symptom disorder, bipolar I or bipolar II disorder with mixed features, substance use disorders, migraine, or cerebrovascular accident. Discontinuation symptoms often resemble symptoms of a persistent anxiety disorder or a return of somatic symptoms of depression for which the medication was initially given. It is important not to confuse discontinuation syndrome with a relapse of the original depressive or anxiety disorder for which the medication was being prescribed. Antidepressant discontinuation syndrome differs from substance withdrawal in that antidepressants themselves have no reinforcing or euphoric effects. Individuals typically do not escalate the dose of medications on their own, and they generally do not engage in drug-seeking behavior to obtain additional medication. Criteria for a substance use disorder are not met.

Other Adverse Effect of Medication

T50.905A Initial encounter

T50.905D Subsequent encounter

T50.905S Sequelae

This category is available for optional use by clinicians to code side effects of medication (other than movement symptoms) when these adverse effects become a main focus of clinical attention. Examples include severe hypotension, cardiac arrhythmias, and priapism.

Other Conditions That May Be a Focus of Clinical Attention

This chapter includes conditions and psychosocial or environmental problems that may be a focus of clinical attention or otherwise affect the diagnosis, course, prognosis, or treatment of an individual's mental disorder. These conditions are presented with their corresponding codes from ICD-10-CM (usually Z codes). A condition or problem in this chapter may be coded 1) if it is a reason for the current visit; 2) if it helps to explain the need for a test, procedure, or treatment; 3) if it plays a role in the initiation or exacerbation of a mental disorder; or 4) if it constitutes a problem that should be considered in the overall management plan.

The conditions and problems listed in this chapter are not mental disorders. Their inclusion in DSM-5-TR is meant to draw attention to the scope of additional issues that may be encountered in routine clinical practice and to provide a systematic listing that may be useful to clinicians in documenting these issues.

For quick reference to all codes in this section, see the DSM-5-TR Classification. Conditions and problems that may be a focus of clinical attention are listed in the subsequent text as follows:

1. **Suicidal behavior** (potentially self-injurious behavior with at least some intent to die) **and nonsuicidal self-injury** (intentional self-inflicted damage to the body in the absence of suicidal intent).
2. **Abuse and neglect** (e.g., child and adult maltreatment and neglect problems, including physical abuse, sexual abuse, neglect, and psychological abuse).
3. **Relational problems** (e.g., parent-child relational problem, sibling relational problem, relationship distress with spouse or intimate partner, disruption by separation or divorce).

4. **Educational problems** (e.g., illiteracy or low-level literacy, schooling unavailable or unattainable, failed school examinations, underachievement in school).
5. **Occupational problems** (e.g., unemployment, change of job, threat of job loss, stressful work schedule, discord with boss and workmates).
6. **Housing problems** (e.g., homelessness; inadequate housing; discord with neighbor, lodger, or landlord).
7. **Economic problems** (e.g., lack of adequate food or safe drinking water, extreme poverty, low income).
8. **Problems related to the social environment** (e.g., problem related to living alone, acculturation difficulty, social exclusion or rejection).
9. **Problems related to interaction with the legal system** (e.g., conviction in criminal proceedings, imprisonment or other incarceration, problems related to release from prison, problems related to other legal circumstances).
10. **Problems related to other psychosocial, personal, and environmental circumstances** (e.g., problems related to unwanted pregnancy, victim of crime, victim of terrorism).
11. **Problems related to access to medical and other health care** (e.g., unavailability or inaccessibility of health care facilities).
12. **Circumstances of personal history** (e.g., personal history of psychological trauma, military deployment).

13. **Other health service encounters for counseling and medical advice** (e.g., sex counseling, other counseling or consultation).
14. **Additional conditions or problems that may be a focus of clinical attention** (e.g., wandering associated with a mental disorder, uncomplicated bereavement, phase of life problem).

Suicidal Behavior and Nonsuicidal Self-Injury

Coding Note for ICD-10-CM Suicidal Behavior

For T codes only, the 6th character should be coded as follows:

A (initial encounter)—Use while the individual is receiving active treatment for the condition (e.g., emergency department encounter, evaluation and treatment by a new clinician); or

D (subsequent encounter)—Use for encounters after the individual has received active treatment for the condition and when he or she is receiving routine care for the condition during the healing or recovery phase (e.g., medication adjustment, other aftercare and follow-up visits).

Suicidal Behavior

This category may be used for individuals who have engaged in potentially self-injurious behavior with at least some intent to die as a result of the act. Evidence of intent to end one's life can be explicit or inferred from the behavior or circumstances. A suicide attempt may or may not result in actual self-injury. If the individual is dissuaded by another person or changes his or her mind before initiating the behavior, this category does not apply.

Current Suicidal Behavior

T14.91A Initial encounter: If suicidal behavior is part of the initial encounter with the clinical presentation

T14.91D Subsequent encounter: If suicidal behavior is part of subsequent encounters with the clinical presentation

Z91.51 History of Suicidal Behavior

If suicidal behavior has occurred during the individual's lifetime

Nonsuicidal Self-Injury

This category may be used for individuals who have engaged in intentional self-inflicted damage to their body of a sort likely to induce bleeding, bruising, or pain (e.g., cutting, burning, stabbing, hitting, excessive rubbing) in the absence of suicidal intent.

R45.88 Current Nonsuicidal Self-Injury

If nonsuicidal self-injurious behavior is part of the clinical presentation

Z91.52 History of Nonsuicidal Self-Injury

If nonsuicidal self-injurious behavior has occurred during the individual's lifetime

Abuse and Neglect

Maltreatment by a family member (e.g., caregiver, intimate adult partner) or by a nonrelative can be the area of current clinical focus, or such maltreatment can be an important factor in the assessment and treatment of individuals with mental disorders or other medical conditions. Because of the legal implications of abuse and neglect, care should be used in assessing these conditions and assigning these codes. Having a past history of abuse or

neglect can influence diagnosis and treatment response in a number of mental disorders, and may also be noted along with the diagnosis.

For the following categories, in addition to listings of the confirmed or suspected event of abuse or neglect, other codes are provided for use if the current clinical encounter is to provide mental health services to either the victim or the perpetrator of the abuse or neglect. A separate code is also provided for designating a past history of abuse or neglect.

Coding Note for ICD-10-CM Abuse and Neglect Conditions

For T codes only, the 7th character should be coded as follows:

A (initial encounter)—Use while the individual is receiving active treatment for the condition (e.g., surgical treatment, emergency department encounter, evaluation and treatment by a new clinician); or

D (subsequent encounter)—Use for encounters after the individual has received active treatment for the condition and when he or she is receiving routine care for the condition during the healing or recovery phase (e.g., cast change or removal, removal of external or internal fixation device, medication adjustment, other aftercare and follow-up visits).

Child Maltreatment and Neglect Problems

Child Physical Abuse

This category may be used when physical abuse of a child is a focus of clinical attention. Child physical abuse is nonaccidental physical injury to a child—ranging from minor bruises to severe fractures or death—occurring as a result of punching, beating, kicking, biting, shaking, throwing, stabbing, choking, hitting (with a hand, stick, strap, or other object), burning, or any other method that is inflicted by a parent, caregiver, or other individual who has responsibility for the child. Such injury is considered abuse regardless of whether the caregiver intended to hurt the child. Physical discipline, such as spanking or paddling, is not considered abuse as long as it is reasonable and causes no bodily injury to the child.

Child Physical Abuse, Confirmed

T74.12XA Initial encounter

T74.12XD Subsequent encounter

Child Physical Abuse, Suspected

T76.12XA Initial encounter

T76.12XD Subsequent encounter

Other Circumstances Related to Child Physical Abuse

Z69.010 Encounter for mental health services for victim of child physical abuse by parent

Z69.020 Encounter for mental health services for victim of nonparental child physical abuse

Z62.810 Personal history (past history) of physical abuse in childhood

Z69.011 Encounter for mental health services for perpetrator of parental child physical abuse

Z69.021 Encounter for mental health services for perpetrator of nonparental child physical abuse

Child Sexual Abuse

This category may be used when sexual abuse of a child is a focus of clinical attention. Child sexual abuse encompasses any sexual act involving a child that is intended to provide sexual gratification to a parent, caregiver, or other individual who has responsibility

for the child. Sexual abuse includes activities such as fondling a child's genitals, penetration, incest, rape, sodomy, and indecent exposure. Sexual abuse also includes noncontact exploitation of a child by a parent or caregiver—for example, forcing, tricking, enticing, threatening, or pressuring a child to participate in acts for the sexual gratification of others, without direct physical contact between child and abuser.

Child Sexual Abuse, Confirmed

T74.22XA Initial encounter

T74.22XD Subsequent encounter

Child Sexual Abuse, Suspected

T76.22XA Initial encounter

T76.22XD Subsequent encounter

Other Circumstances Related to Child Sexual Abuse

Z69.010 Encounter for mental health services for victim of child sexual abuse by parent

Z69.020 Encounter for mental health services for victim of nonparental child sexual abuse

Z62.810 Personal history (past history) of sexual abuse in childhood

Z69.011 Encounter for mental health services for perpetrator of parental child sexual abuse

Z69.021 Encounter for mental health services for perpetrator of nonparental child sexual abuse

Child Neglect

This category may be used when child neglect is a focus of clinical attention. Child neglect is defined as any confirmed or suspected egregious act or omission by a child's parent or other caregiver that deprives the child of basic age-appropriate needs and thereby results, or has reasonable potential to result, in physical or psychological harm to the child. Child neglect encompasses abandonment; lack of appropriate supervision; failure to attend to necessary emotional or psychological needs; and failure to provide necessary education, medical care, nourishment, shelter, and/or clothing.

Child Neglect, Confirmed

T74.02XA Initial encounter

T74.02XD Subsequent encounter

Child Neglect, Suspected

T76.02XA Initial encounter

T76.02XD Subsequent encounter

Other Circumstances Related to Child Neglect

Z69.010 Encounter for mental health services for victim of child neglect by parent

Z69.020 Encounter for mental health services for victim of nonparental child neglect

Z62.812 Personal history (past history) of neglect in childhood

Z69.011 Encounter for mental health services for perpetrator of parental child neglect

Z69.021 Encounter for mental health services for perpetrator of nonparental child neglect

Child Psychological Abuse

This category may be used when psychological abuse of a child is a focus of clinical attention. Child psychological abuse is nonaccidental verbal or symbolic acts by a child's parent or caregiver that result, or have reasonable potential to result, in significant psychological harm to the child. (Physical and sexual abusive acts are not included in this category.) Examples of psychological abuse of a child include berating, disparaging, or humiliating the child; threatening the child; harming/abandoning—or indicating that the alleged offender will harm/abandon—people or things that the child cares about; confining the child (as by tying a child's arms or legs together or binding a child to furniture or another object, or confining a child to a small enclosed area [e.g., a closet]); egregious scapegoating of the child; coercing the child to inflict pain on himself or herself; and disciplining the child excessively (i.e., at an extremely high frequency or duration, even if not at a level of physical abuse) through physical or nonphysical means.

Child Psychological Abuse, Confirmed

T74.32XA Initial encounter

T74.32XD Subsequent encounter

Child Psychological Abuse, Suspected

T76.32XA Initial encounter

T76.32XD Subsequent encounter

Other Circumstances Related to Child Psychological Abuse

Z69.010 Encounter for mental health services for victim of child psychological abuse by parent

Z69.020 Encounter for mental health services for victim of nonparental child psychological abuse

Z62.811 Personal history (past history) of psychological abuse in childhood

Z69.011 Encounter for mental health services for perpetrator of parental child psychological abuse

Z69.021 Encounter for mental health services for perpetrator of nonparental child psychological abuse

Adult Maltreatment and Neglect Problems

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Spouse or Partner Violence, Physical

This category may be used when spouse or partner physical violence is a focus of clinical attention. Spouse or partner physical violence is nonaccidental acts of physical force that result, or have reasonable potential to result, in physical harm to an intimate partner or that evoke significant fear in the partner. Nonaccidental acts of physical force include shoving, slapping, hair pulling, pinching, restraining, shaking, throwing, biting, kicking, hitting with the fist or an object, burning, poisoning, applying force to the throat, cutting off the air supply, holding the head under water, and using a weapon. Acts for the purpose of physically protecting oneself or one's partner are excluded.

Spouse or Partner Violence, Physical, Confirmed

T74.11XA Initial encounter

T74.11XD Subsequent encounter

Spouse or Partner Violence, Physical, Suspected

T76.11XA Initial encounter

T76.11XD Subsequent encounter

Other Circumstances Related to Spouse or Partner Violence, Physical

Z69.11 Encounter for mental health services for victim of spouse or partner violence, physical

Z91.410 Personal history (past history) of spouse or partner violence, physical

Z69.12 Encounter for mental health services for perpetrator of spouse or

partner violence, physical

Spouse or Partner Violence, Sexual

This category may be used when spouse or partner sexual violence is a focus of clinical attention. Spouse or partner sexual violence involves the use of physical force or psychological coercion to compel the partner to engage in a sexual act against his or her will, whether or not the act is completed. Also included in this category are sexual acts with an intimate partner who is unable to consent.

Spouse or Partner Violence, Sexual, Confirmed

T74.21XA Initial encounter

T74.21XD Subsequent encounter

Spouse or Partner Violence, Sexual, Suspected

T76.21XA Initial encounter

T76.21XD Subsequent encounter

Other Circumstances Related to Spouse or Partner Violence, Sexual

Z69.81 Encounter for mental health services for victim of spouse or partner violence, sexual

Z91.410 Personal history (past history) of spouse or partner violence, sexual

Z69.12 Encounter for mental health services for perpetrator of spouse or partner violence, sexual

Spouse or Partner Neglect

This category may be used when spouse or partner neglect is a focus of clinical attention. Spouse or partner neglect is any egregious act or omission by one partner that deprives a dependent partner of basic needs and thereby results, or has reasonable potential to result, in physical or psychological harm to the dependent partner. This category may be used in the context of relationships in which one partner is extremely dependent on the other

partner for care or for assistance in navigating ordinary daily activities—for example, a partner who is incapable of self-care because of substantial physical, psychological/intellectual, or cultural limitations (e.g., inability to communicate with others and manage everyday activities as a result of living in a foreign culture).

Spouse or Partner Neglect, Confirmed

T74.01XA Initial encounter

T74.01XD Subsequent encounter

Spouse or Partner Neglect, Suspected

T76.01XA Initial encounter

T76.01XD Subsequent encounter

Other Circumstances Related to Spouse or Partner Neglect

Z69.11 Encounter for mental health services for victim of spouse or partner neglect

Z91.412 Personal history (past history) of spouse or partner neglect

Z69.12 Encounter for mental health services for perpetrator of spouse or partner neglect

Spouse or Partner Abuse, Psychological

This category may be used when spouse or partner psychological abuse is a focus of clinical attention. Spouse or partner psychological abuse encompasses nonaccidental verbal or symbolic acts by one partner that result, or have reasonable potential to result, in significant harm to the other partner. Acts of psychological abuse include berating or humiliating the victim; interrogating the victim; restricting the victim's ability to come and go freely; obstructing the victim's access to assistance (e.g., law enforcement; legal, protective, or medical resources); threatening the victim with physical harm or sexual assault; harming, or threatening to harm,

people or things that the victim cares about; unwarranted restriction of the victim's access to or use of economic resources; isolating the victim from family, friends, or social support resources; stalking the victim; and trying to make the victim question his or her sanity ("gaslighting").

Spouse or Partner Abuse, Psychological, Confirmed

T74.31XA Initial encounter

T74.31XD Subsequent encounter

Spouse or Partner Abuse, Psychological, Suspected

T76.31XA Initial encounter

T76.31XD Subsequent encounter

Other Circumstances Related to Spouse or Partner Abuse, Psychological

Z69.11 Encounter for mental health services for victim of spouse or partner psychological abuse

Z91.411 Personal history (past history) of spouse or partner psychological abuse

Z69.12 Encounter for mental health services for perpetrator of spouse or partner psychological abuse

Adult Abuse by Nonspouse or Nonpartner

This category may be used when the abuse of an adult by another adult who is not an intimate partner is a focus of clinical attention. Such maltreatment may involve acts of physical, sexual, or emotional abuse. Examples of adult abuse include nonaccidental acts of physical force (e.g., pushing/shoving, scratching, slapping, throwing something that could hurt, punching, biting) that have resulted—or have reasonable potential to result—in physical harm or have caused significant fear; forced or coerced sexual acts; and verbal or symbolic acts with the potential to cause psychological harm (e.g., berating or humiliating the person; interrogating the person; restricting the person's ability to come and go freely; obstructing the person's access to assistance;

threatening the person; harming or threatening to harm people or things that the person cares about; restricting the person's

access to or use of economic resources; isolating the person from family, friends, or social support resources; stalking the person; trying to make the person think that he or she is crazy). Acts for the purpose of physically protecting oneself or the other person are excluded.

Adult Physical Abuse by Nonspouse or Nonpartner, Confirmed

T74.11XA Initial encounter

T74.11XD Subsequent encounter

Adult Physical Abuse by Nonspouse or Nonpartner, Suspected

T76.11XA Initial encounter

T76.11XD Subsequent encounter

Adult Sexual Abuse by Nonspouse or Nonpartner, Confirmed

T74.21XA Initial encounter

T74.21XD Subsequent encounter

Adult Sexual Abuse by Nonspouse or Nonpartner, Suspected

T76.21XA Initial encounter

T76.21XD Subsequent encounter

Adult Psychological Abuse by Nonspouse or Nonpartner, Confirmed

T74.31XA Initial encounter

T74.31XD Subsequent encounter

Adult Psychological Abuse by Nonspouse or Nonpartner, Suspected

T76.31XA Initial encounter

T76.31XD Subsequent encounter

Other Circumstances Related to Adult Abuse by Nonspouse or Nonpartner

Z69.81 Encounter for mental health services for victim of nonspousal or nonpartner adult abuse

Z69.82 Encounter for mental health services for perpetrator of nonspousal or nonpartner adult abuse

Relational Problems

Key relationships, especially intimate adult partner relationships and parent/caregiver-child relationships, have a significant impact on the health of the individuals in these relationships. These relationships can be health promoting and protective, neutral, or detrimental to health outcomes. In the extreme, these close relationships can be associated with maltreatment or neglect, which has significant medical and psychological consequences for the affected individual. A relational problem may come to clinical attention either as the reason that the individual seeks health care or as a problem that affects the course, prognosis, or treatment of the individual's mental disorder or other medical condition.

Parent-Child Relational Problem

Z62.820 Parent–Biological Child

Z62.821 Parent–Adopted Child

Z62.822 Parent–Foster Child

Z62.898 Other Caregiver–Child

For this category, the term *parent* is used to refer to one of the child's primary caregivers, who may be a biological, adoptive, or foster parent or may be another relative (such as a grandparent) who fulfills a parental role for the child. This category may be used when the main focus of clinical attention is to address the quality of the parent-child relationship or when

the quality of the parent-child relationship is affecting the course, prognosis, or treatment of a mental disorder or other medical condition. Typically, the parent-child relational problem is associated with impaired functioning in behavioral, cognitive, or affective domains. Examples of behavioral problems include inadequate parental control, supervision, and involvement with the child; parental overprotection; excessive parental pressure; arguments that escalate to threats of physical violence; and avoidance without resolution of problems. Cognitive problems may include negative attributions of the other's intentions, hostility toward or scapegoating of the other, and unwarranted feelings of estrangement. Affective problems may include feelings of sadness, apathy, or anger about the other individual in the relationship. Clinicians should take into account the developmental needs of the child and the cultural context.

Z62.891 Sibling Relational Problem

This category may be used when the focus of clinical attention is a pattern of interaction among siblings that is associated with significant impairment in individual or family functioning or with development of symptoms in one or more of the siblings, or when a sibling relational problem is affecting the course, prognosis, or treatment of a sibling's mental disorder or other medical condition. This category may be used for either children or adults if the focus is on the sibling relationship. Siblings in this context include full, half-, step-, foster, and adopted siblings.

Z63.0 Relationship Distress With Spouse or Intimate Partner

This category may be used when the major focus of the clinical contact is to address the quality of the intimate (spouse or partner) relationship or when the quality of that relationship is affecting the course, prognosis, or treatment of a mental disorder or other medical condition. Partners can be of the same or different genders. Typically, the relationship distress is associated with impaired functioning in behavioral, cognitive, or affective domains. Examples of behavioral problems include conflict resolution difficulty, withdrawal, and overinvolvement. Cognitive problems can manifest as chronic negative attributions of the other's intentions or dismissals of the partner's positive behaviors. Affective problems would include chronic sadness, apathy, and/or anger about the other partner.

Problems Related to the Family Environment

Z62.29 Upbringing Away From Parents

This category may be used when the main focus of clinical attention pertains to issues regarding a child being raised away from the parents or when this separate upbringing affects the course, prognosis, or treatment of a mental disorder or other medical condition. The child could be one who is under state custody and placed in kin care or foster care. The child could also be one who is living in a nonparental relative's home, or with friends, but whose out-of-home placement is not mandated or sanctioned by the courts. Problems related to a child living in a group home or orphanage are also included. This category excludes issues related to Z59.3 Problem Related to Living in a Residential Institution.

Z62.898 Child Affected by Parental Relationship Distress

This category may be used when the focus of clinical attention is the negative effects of parental relationship discord (e.g., high levels of conflict, distress, or disparagement) on a child in the family, including effects on the child's mental disorder or other medical condition.

Z63.5 Disruption of Family by Separation or Divorce

This category may be used when partners in an intimate adult couple are living apart because of relationship problems or are in the process of divorce.

Z63.8 High Expressed Emotion Level Within Family

Expressed emotion is a construct used as a qualitative measure of the "amount" of emotion—in particular, hostility, emotional overinvolvement, and criticism directed toward a family member who is an identified patient—displayed in the family environment. This category may be used when a family's high level of expressed emotion is the focus of clinical attention or is affecting the course, prognosis, or treatment of a family member's mental disorder or other medical condition.

Educational Problems

These categories may be used when an academic or educational problem is the focus of clinical attention or has an impact on the individual's diagnosis, treatment, or prognosis. Problems to be considered include illiteracy or low-level literacy; lack of access to schooling owing to unavailability or unattainability; problems with academic performance (e.g., failing school examinations, receiving failing marks or grades) or underachievement (below what would be expected given the individual's intellectual capacity); discord with teachers, school staff, or other students; problems related to inadequate teaching; and any other problems related to education and/or literacy.

Z55.0 Illiteracy and Low-Level Literacy

Z55.1 Schooling Unavailable and Unattainable

Z55.2 Failed School Examinations

Z55.3 Underachievement in School

Z55.4 Educational Maladjustment and Discord With Teachers and Classmates

Z55.8 Problems Related to Inadequate Teaching

Z55.9 Other Problems Related to Education and Literacy

Occupational Problems

These categories may be used when an occupational problem is the focus of clinical attention or has an impact on the individual's treatment or prognosis. Areas to be considered include problems with employment or in the work environment, including problems related to current military deployment status; unemployment; recent change of job; threat of job loss; stressful work schedule; uncertainty about career choices; sexual harassment on the job; other discord with boss, supervisor, co-workers, or others in the work environment; uncongenial or hostile work environments; other physical or mental strain related to work; sexual harassment on the job; and any other problems related to employment and/or occupation.

Z56.82 Problem Related to Current Military Deployment Status

This category may be used when an occupational problem directly related to an individual's military deployment status is the focus of clinical attention or has an impact on the individual's diagnosis, treatment, or prognosis. Psychological reactions to deployment are not included in this category; such reactions would be better captured as an adjustment disorder or another mental disorder.

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Z56.0 Unemployment

Z56.1 Change of Job

Z56.2 Threat of Job Loss

Z56.3 Stressful Work Schedule

Z56.4 Discord With Boss and Workmates

Z56.5 Uncongenial Work Environment

Z56.6 Other Physical and Mental Strain Related to Work

Z56.81 Sexual Harassment on the Job

Z56.9 Other Problem Related to Employment

Housing Problems

Z59.01 Sheltered Homelessness

This category may be used when sheltered homelessness has an impact on an individual's treatment or prognosis. An individual is considered to be experiencing sheltered homelessness if the primary nighttime residence is a homeless shelter, a warming shelter, a domestic violence shelter, a motel, or in a temporary or transitional living situation.

Z59.02 Unsheltered Homelessness

This category may be used when unsheltered homelessness has an impact on an individual's treatment or prognosis. An individual is considered to be

experiencing unsheltered homelessness if residing in a place not meant for human habitation, such as a public space (e.g., tunnel, transportation station, mall), a building not intended for residential use (e.g., abandoned structure, unused factory), a car, a cave, a cardboard box, or some other ad hoc housing situation.

Z59.1 Inadequate Housing

This category may be used when lack of adequate housing has an impact on an individual's treatment or prognosis. Examples of inadequate housing conditions include lack of heat (in cold temperatures) or electricity, infestation by insects or rodents, inadequate plumbing and toilet facilities, overcrowding, lack of adequate sleeping space, and excessive noise. It is important to consider cultural norms before assigning this category.

Z59.2 Discord With Neighbor, Lodger, or Landlord

This category may be used when discord with neighbors, lodgers, or a landlord is a focus of clinical attention or has an impact on the individual's treatment or prognosis.

Z59.3 Problem Related to Living in a Residential Institution

This category may be used when a problem (or problems) related to living in a residential institution is a focus of clinical attention or has an impact on the individual's treatment or prognosis. Psychological reactions to a change in living situation are not included in this category; such reactions would be better captured as an adjustment disorder.

Z59.9 Other Housing Problem

This category may be used when there is a problem related to housing circumstances other than as specified above.

Economic Problems

These categories may be used when an economic problem is the focus of clinical attention or has an impact on the individual's treatment or prognosis. Areas to be considered include lack of adequate food (food insecurity) or safe drinking water, extreme poverty, low income, insufficient social or health insurance or welfare support, or any other economic problems.

Z59.41 Food Insecurity

Z58.6 Lack of Safe Drinking Water

Z59.5 Extreme Poverty

Z59.6 Low Income

Z59.7 Insufficient Social or Health Insurance or Welfare Support

This category may be used for individuals who meet eligibility criteria for social or welfare support but are not receiving such support, who receive support that is insufficient to address their needs, or who otherwise lack access to needed insurance or support programs. Examples include inability to qualify for welfare support because of lack of proper documentation or evidence of address, inability to obtain adequate health insurance because of age or a preexisting condition, and denial of support owing to excessively stringent income or other requirements.

Z59.9 Other Economic Problem

This category may be used when there is a problem related to economic circumstances other than as specified above.

Problems Related to the Social Environment

Z60.2 Problem Related to Living Alone

This category may be used when a problem associated with living alone is the focus of clinical attention or has an impact on the individual's treatment or prognosis. Examples of such problems include chronic feelings of loneliness, isolation, and lack of structure in carrying out activities of daily living (e.g., irregular meal and sleep schedules, inconsistent performance of home maintenance chores).

Z60.3 Acculturation Difficulty

This category may be used when difficulty in adjusting to a new culture (e.g., following migration) is the focus of clinical attention or has an impact on the individual's treatment or prognosis.

Z60.4 Social Exclusion or Rejection

This category may be used when there is an imbalance of social power such that there is recurrent social exclusion or rejection by others. Examples of social rejection include bullying, teasing, and intimidation by others; being targeted by others for verbal abuse and humiliation; and being purposefully excluded from the activities of peers, workmates, or others in one's social environment.

Z60.5 Target of (Perceived) Adverse Discrimination or Persecution

This category may be used when there is perceived or experienced discrimination against or persecution of the individual based on his or her membership (or perceived membership) in a specific category. Typically, such categories include gender or gender identity, race, ethnicity, religion, sexual orientation, country of origin, political beliefs, disability status, caste, social status, weight, and physical appearance.

Z60.9 Other Problem Related to Social Environment

This category may be used when there is a problem related to the individual's social environment other than as specified above.

Problems Related to Interaction With the Legal System

These categories may be used when a problem related to interaction with the legal system is the focus of clinical attention or has an impact on the individual's treatment or prognosis. Areas to be considered include conviction in criminal proceedings, imprisonment or other incarceration, problems related to release from prison, and problems related to other legal circumstances (e.g., civil litigation, child custody or support proceedings).

Z65.0 Conviction in Criminal Proceedings Without Imprisonment

Z65.1 Imprisonment or Other Incarceration

Z65.2 Problems Related to Release From Prison

Z65.3 Problems Related to Other Legal Circumstances (e.g., civil litigation, child custody or support proceedings)

Problems Related to Other Psychosocial, Personal, and Environmental Circumstances

Z72.9 Problem Related to Lifestyle

This category may be used when a lifestyle problem is a specific focus of treatment or directly affects the course, prognosis, or treatment of a mental disorder or other medical condition. Examples of lifestyle problems include lack of physical exercise, inappropriate diet, high-risk sexual behavior, and poor sleep hygiene. A problem that is attributable to a symptom of a mental disorder should not be coded unless that problem is a specific focus of treatment or directly affects the course, prognosis, or treatment of the individual. In such cases, both the mental disorder and the lifestyle problem should be coded.

Z64.0 Problems Related to Unwanted Pregnancy

Z64.1 Problems Related to Multiparity

Z64.4 Discord With Social Service Provider, Including Probation Officer, Case Manager, or Social Services Worker

Z65.4 Victim of Crime

Z65.4 Victim of Terrorism or Torture

Z65.5 Exposure to Disaster, War, or Other Hostilities

Problems Related to Access to Medical and Other Health Care

These categories may be used when a problem related to access to medical or other health care is the focus of clinical attention or has an impact on the individual’s treatment or prognosis.

Z75.3 Unavailability or Inaccessibility of Health Care Facilities

Z75.4 Unavailability or Inaccessibility of Other Helping Agencies

Circumstances of Personal History

Z91.49 Personal History of Psychological Trauma

Z91.82 Personal History of Military Deployment

Other Health Service Encounters for Counseling and Medical Advice

Z31.5 Genetic Counseling

This category may be used for individuals seeking genetic counseling to understand the risks of developing a mental disorder with a significant genetic component (e.g., bipolar disorder) for themselves and other family members, including their existing children, as well as the risks for their future children.

Z70.9 Sex Counseling

This category may be used when the individual seeks counseling related to sex education, sexual behavior, sexual orientation, sexual attitudes (embarrassment, timidity), others' sexual behavior or orientation (e.g., spouse, partner, child), sexual enjoyment, or any other sex-related issue.

Z71.3 Dietary Counseling

This category may be used when the individual seeks counseling related to dietary issues like weight management.

Z71.9 Other Counseling or Consultation

This category may be used when counseling is provided or advice/consultation is sought for a problem that is not specified above or elsewhere in this chapter (e.g., counseling regarding drug abuse prevention in an adolescent).

Additional Conditions or Problems That May Be a Focus of Clinical Attention

Z91.83 Wandering Associated With a Mental Disorder

This category may be used for individuals with a mental disorder whose desire to walk about leads to significant clinical management or safety concerns. For example, individuals with major neurocognitive or neurodevelopmental disorders may experience a restless urge to wander that places them at risk for falls and causes them to leave supervised settings without needed accompaniment. This category excludes individuals whose intent is to escape an unwanted housing situation (e.g., children who are running away from home, individuals who no longer wish to remain in the hospital) or those who walk or pace as a result of medication-induced akathisia.

> **Coding note:** First code associated mental disorder (e.g., major neurocognitive disorder, autism spectrum disorder), then code Z91.83 wandering associated with [specific mental disorder].

Z63.4 Uncomplicated Bereavement

This category may be used when the focus of clinical attention is a normal reaction to the death of a loved one. As part of their reaction to such a loss, some grieving individuals present with symptoms characteristic of a major depressive episode—for example, feelings of sadness and associated symptoms such as insomnia, poor appetite, and weight loss. The bereaved individual typically regards the depressed mood as "normal," although the individual may seek professional help for relief of associated symptoms such as insomnia or anorexia. The duration and expression of "normal" bereavement vary considerably among different cultural groups. Further guidance in distinguishing grief from a major depressive episode and from prolonged grief disorder is provided in their respective texts.

Z60.0 Phase of Life Problem

This category may be used when a problem adjusting to a life-cycle transition (a particular developmental phase) is the focus of clinical attention or has an impact on the individual's treatment or prognosis. Examples of such transitions include entering or completing school, leaving

parental control, getting married, starting a new career, becoming a parent, adjusting to an “empty nest” after children leave home, and retiring.

Z65.8 Religious or Spiritual Problem

This category may be used when the focus of clinical attention is a religious or spiritual problem. Examples include distressing experiences that involve loss or questioning of faith, problems associated with conversion to a new faith, or questioning of spiritual values that may not necessarily be related to an organized church or religious institution.

Z72.811 Adult Antisocial Behavior

This category may be used when the focus of clinical attention is adult antisocial behavior that is not attributable to a mental disorder (e.g., conduct disorder, antisocial personality disorder). Examples include the behavior of some professional thieves, racketeers, or dealers in illegal substances.

Z72.810 Child or Adolescent Antisocial Behavior

This category may be used when the focus of clinical attention is antisocial behavior in a child or adolescent that is not attributable to a mental disorder (e.g., intermittent explosive disorder, conduct disorder). Examples include isolated antisocial acts by children or adolescents (not a pattern of antisocial behavior).

Z91.19 Nonadherence to Medical Treatment

This category may be used when the focus of clinical attention is nonadherence to an important aspect of treatment for a mental disorder or another medical condition. Reasons for such nonadherence may include discomfort resulting from treatment (e.g., medication side effects), expense of treatment, personal value judgments or religious or cultural beliefs about the proposed treatment, age-related debility, and the presence of a mental disorder (e.g., schizophrenia, personality disorder). This category may be used only when the problem is sufficiently severe to warrant independent clinical attention and does not meet diagnostic criteria for psychological factors affecting other medical conditions.

E66.9 Overweight or Obesity

This category may be used when overweight or obesity is a focus of clinical attention.

Z76.5 Malingering

The essential feature of malingering is the intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by external incentives such as avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution, or obtaining drugs. Under some circumstances, malingering may represent adaptive behavior—for example, feigning illness while a captive of the enemy during wartime. Malingering should be strongly considered if any combination of the following is noted:

1. Medicolegal context of presentation (e.g., the individual is referred by an attorney to the clinician for examination, or the individual self-refers while litigation or criminal charges are pending).
2. Marked discrepancy between the individual's claimed stress or disability and the objective findings and observations.
3. Lack of cooperation during the diagnostic evaluation and in complying with the prescribed treatment regimen.
4. The presence of antisocial personality disorder.

Malingering differs from factitious disorder in that the motivation for the symptom production in malingering is an external incentive, whereas in factitious disorder external incentives are absent. Malingering is differentiated from functional neurological symptom disorder (conversion disorder) and other somatic symptom–related mental disorders by the intentional production of symptoms and by the obvious external incentives associated with it. Definite evidence of feigning (such as clear evidence that loss of function is present during the examination but not at home) would suggest a diagnosis of factitious disorder if the individual's apparent aim is to assume the sick role, or malingering if it is to obtain an incentive, such as money.

R41.81 Age-Related Cognitive Decline

This category may be used when the focus of clinical attention is an objectively identified decline in cognitive functioning consequent to the aging process that is within normal limits

given the individual’s age. Individuals with this condition may report problems remembering names or appointments or may experience difficulty in solving complex problems. This category should be considered only after it has been determined that the cognitive impairment is not better explained by a specific mental disorder or attributable to a neurological condition.

R41.83 Borderline Intellectual Functioning

This category may be used when an individual’s borderline intellectual functioning is the focus of clinical attention or has an impact on the individual’s treatment or prognosis. Differentiating borderline intellectual functioning and mild intellectual developmental disorder (intellectual disability) requires careful assessment of intellectual and adaptive functions and their discrepancies, particularly in the presence of co-occurring mental disorders that may affect patient compliance with standardized testing procedures (e.g., schizophrenia or attention-deficit/hyperactivity disorder, with severe impulsivity).

SECTION III

Emerging Measures and Models

Assessment Measures

- Cross-Cutting Symptom Measures
  - DSM-5 Self-Rated Level 1 Cross-Cutting Symptom Measure—Adult
  - Parent/Guardian-Rated DSM-5 Level 1 Cross-Cutting Symptom Measure—Child Age 6–17
- Clinician-Rated Dimensions of Psychosis Symptom Severity
- World Health Organization Disability Assessment Schedule 2.0 (WHODAS 2.0)

Culture and Psychiatric Diagnosis

- Key Terms
- Cultural Formulation
  - Core Cultural Formulation Interview (CFI)
  - Cultural Formulation Interview (CFI)—Informant Version
- Cultural Concepts of Distress

Alternative DSM-5 Model for Personality Disorders

Conditions for Further Study

- Attenuated Psychosis Syndrome
- Depressive Episodes With Short-Duration Hypomania
- Caffeine Use Disorder
- Internet Gaming Disorder
- Neurobehavioral Disorder Associated With Prenatal Alcohol Exposure

Suicidal Behavior Disorder

Nonsuicidal Self-Injury Disorder

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This section contains tools and techniques to enhance clinical practice, understand the cultural context of mental disorders, and facilitate further study of proposed emerging diagnoses. The inclusion of this material represents a dynamic DSM-5 that will evolve with advances in the field.

Among the tools in Section III, Assessment Measures provides a Level 1 cross-cutting self/informant-rated measure that serves as a review of systems across mental disorders. A clinician-rated symptom severity scale for schizophrenia and other psychotic disorders also is provided, as well as the World Health Organization Disability Assessment Schedule, Version 2 (WHODAS 2.0). Severity measures for symptoms identified by the Level 1 cross-cutting self/informant-rated measure are available online (www.psychiatry.org/dsm5) and may be used to explore significant responses to the Level 1 screen.

A comprehensive review of the cultural context of mental disorders and the Cultural Formulation Interview (CFI) for clinical use are provided in the chapter “Culture and Psychiatric Diagnosis.” Both clinician and informant versions of the CFI are available online (www.psychiatry.org/dsm5). The chapter also contains a glossary of examples of cultural concepts of distress.

The Alternative DSM-5 Model for Personality Disorders provides an alternative to the extant personality disorders classification in Section II. This hybrid dimensional-categorical model defines personality disorder in terms of impairments in personality functioning and pathological personality traits.

Conditions for Further Study includes proposed criteria sets and descriptive text for new conditions that are the focus of active research, such as attenuated psychosis syndrome and caffeine use disorder.

Assessment Measures

A growing body of scientific evidence favors dimensional concepts in the diagnosis of mental disorders. Limitations of a categorical approach to diagnosis include the failure to find zones of rarity between diagnoses (i.e., delineation of mental disorders from one another by natural boundaries), need for intermediate categories like schizoaffective disorder, high rates of comorbidity, need for frequent use of other or unspecified diagnoses, relative lack of utility in furthering identification of unique antecedent validators for most mental disorders, and lack of treatment specificity for the various diagnostic categories.

From both clinical and research perspectives, there is a need for a more dimensional approach that can be combined with DSM's set of categorical diagnoses to better capture the heterogeneity in the presentation of various mental and substance use disorders. Such an approach allows clinicians or others to better communicate particular variation of features that apply to presentations that meet criteria for a disorder. Such features include differential severity of individual symptoms (including symptoms that are part of the diagnostic features as well as those that are associated with the disorder) as measured by intensity, duration, and impact on functioning. This combined approach also allows clinicians or others to identify conditions that do not meet criteria for a disorder but are severe and disabling and in need of treatment.

It is expected that as the understanding of basic disease mechanisms for mental and substance use disorders based on pathophysiology, neurocircuitry, and gene-environment interactions increases, more objective measures of psychopathology will be incorporated into the diagnostic criteria sets to enhance their accuracy. Until such time, a dimensional approach depending primarily on an individual's subjective reports of symptom experiences along with the clinician's interpretation is highlighted by current psychiatric evaluation guidelines as an important step in enhancing diagnostic practice.

Cross-cutting symptom measures, modeled on general medicine's review of systems, can serve as an approach for reviewing critical psychopathological domains across age groups and diagnoses. The general medical review of systems—a list of questions arranged by organ systems—is crucial to detecting signs and symptoms of dysfunction and disease with which the individual may or may not present that can facilitate diagnosis and treatment. A similar review of various mental systems (or domains), which is the goal of the cross-cutting symptom measures, can aid in a more comprehensive mental status assessment of individuals at the initial evaluation. The review of mental systems can systematically draw attention to signs and symptoms of other domains of mental health and functioning that may be important to the individual's care. The cross-cutting measures have two levels of inquiry: Level 1 uses

1 to 3 questions for each of 13 symptom domains for adults (self-rated) and 12 domains for children (ages 6–17, parent rated) and adolescents (child rated, ages 11–17) to identify emerging signs and symptoms. Level 2 questions provide a more in-depth assessment of certain domains (e.g., depression, anxiety, mania, anger, irritability, somatic symptoms). These measures are developed to be administered both at initial interview and at follow-up visits. Thus, use of these measures can form key aspects of measurement-based care, the process by which standardized assessment tools are

administered and results used to track individuals' progress over time to guide a more precise plan of care. Use of these measures ultimately aims to inform measurement-based care by identifying areas of emerging symptoms and concerns as well as supporting ongoing symptom monitoring, treatment adjustment, and outcomes critical to the provision of quality care for individuals with mental and substance use disorders. As a result, these cross-cutting symptom measures have been identified as important components of psychiatric diagnostic assessment in clinical practice guidelines.

Severity measures are disorder-specific, corresponding closely to the criteria that constitute the disorder definition. They may be administered to individuals who have received a diagnosis or who have a clinically significant syndrome that falls short of meeting full criteria for a diagnosis (e.g., use of the Clinician-Rated Dimensions of Psychosis Symptom Severity in individuals whose symptoms meet criteria for schizophrenia). Some of the assessments are self-rated, while others are rated by the clinician based on observation of the individual. As with the cross-cutting symptom measures, these measures can be administered both at initial interview and over time to track the severity of the individual's disorder and response to treatment. These assessments help operationalize symptom frequency, intensity, or duration; overall symptom severity; or symptom type (e.g., depression, anxiety, sleep disturbance) for many, though not all, DSM-5 diagnoses (e.g., generalized anxiety disorder, social anxiety disorder, psychotic disorders, posttraumatic stress disorder, autism spectrum disorder, and social (pragmatic) communication disorder). Data obtained from use of these disorder-specific measures can assist with diagnosis and inform symptom monitoring and treatment planning.

The World Health Organization Disability Assessment Schedule, Version 2.0 (WHODAS 2.0) was developed by the World Health Organization to assess an individual's ability to perform activities in six areas: understanding and communicating; getting around; self-care; getting along with people; life activities (e.g., household, work/school); and participation in society. This version of the scale is self-administered and was developed for individuals with any medical condition, not just mental disorders. It corresponds to concepts contained in the WHO International Classification of Functioning, Disability and Health. This assessment can also be used over time to track changes in an individual's level of functioning. Assessment of functioning is a key aspect of psychiatric diagnostic assessment given that most DSM-5 criteria sets include a requirement that the disturbance causes clinically significant

distress or impairment in functioning. Individuals with mental disorders are more likely to have severe impairment in functioning (i.e., communicating or understanding; getting along with others; carrying out daily activities at work, home, or school; participating in social activities) compared to individuals with chronic medical conditions. In addition, many individuals seek help for mental disorders because of the direct impact of their disorders on functional impairment across multiple domains and settings. Functional impairment may impact prognosis across diagnoses and, if residual functional impairment remains after symptoms subside, can lead to recurrence or relapse for conditions such as major depressive disorder and anxiety disorders.

This chapter focuses on the DSM-5 Level 1 Cross-Cutting Symptom Measure (adult self-rated and parent/guardian versions); the Clinician-Rated Dimensions of Psychosis Symptom Severity; and the WHODAS 2.0. Clinician instructions, scoring information, and interpretation guidelines are included for each. Description of the child-rated version is not included in print given the overall similarity in items, scoring, and clinician instructions and guidelines with the parent/guardian-rated version. These measures, including the child-rated version, and additional dimensional assessments, such as those for diagnostic severity, can be found online at www.psychiatry.org/dsm5.

Cross-Cutting Symptom Measures

Level 1 Cross-Cutting Symptom Measure

The DSM-5 Level 1 Cross-Cutting Symptom Measure is a self- or informant-rated measure that assesses domains that are important across psychiatric diagnoses. It is intended to help clinicians identify additional areas of inquiry that may have significant impact on the individual's treatment and prognosis. In addition, the measure may be used to track changes in the individual's symptom presentation over time.

The adult version of the measure consists of 23 questions that assess 13 psychiatric domains, including depression, anger, mania, anxiety, somatic symptoms, suicidal ideation, psychosis, sleep problems, memory, repetitive thoughts and behaviors, dissociation, personality functioning, and substance use (Table 1). Each domain consists of one to three questions. Each item inquires about how much (or how often) the individual has been bothered by the specific symptom during the past 2 weeks. If the individual is of impaired capacity and unable to complete the form (e.g., an individual with major neurocognitive disorder), a knowledgeable adult informant may complete this measure.

TABLE 1 Adult DSM-5 Self-Rated Level 1 Cross-Cutting Symptom Measure: 13 domains, thresholds for further inquiry, and associated DSM-5 Level 2 measures

Domain	Domain name	Threshold to guide further inquiry	DSM-5 Level 2 Cross-Cutting Symptom Measure[a]
I.	Depression	Mild or greater	Level 2—Depression—Adult (PROMIS Emotional Distress—Short Form)
II.	Anger	Mild or greater	Level 2—Anger—Adult (PROMIS Emotional Distress—Anger—Short Form)
III.	Mania	Mild or greater	Level 2—Mania—Adult (Altman Self-Rating Mania Scale [ASRM])
IV.	Anxiety	Mild or greater	Level 2—Anxiety—Adult (PROMIS Emotional Distress—Anxiety—Short Form)
V.	Somatic symptoms	Mild or greater	Level 2—Somatic Symptom—Adult (Patient Health Questionnaire–15 [PHQ-15] Somatic Symptom Severity Scale)
VI.	Suicidal ideation	Slight or greater	None
VII.	Psychosis	Slight or greater	None
VIII.	Sleep problems	Mild or greater	Level 2—Sleep Disturbance—Adult (PROMIS Sleep Disturbance—Short Form)
IX.	Memory	Mild or greater	None
X.	Repetitive thoughts and behaviors	Mild or greater	Level 2—Repetitive Thoughts and Behaviors—Adult (Florida Obsessive-Compulsive Inventory [FOCI] Severity Scale)
XI.	Dissociation	Mild or greater	None
XII.	Personality functioning	Mild or greater	None
XIII.	Substance use	Slight or greater	Level 2—Substance Use—Adult (adapted from the NIDA-Modified ASSIST)

Note. NIDA = National Institute on Drug Abuse.
[a]Available at www.psychiatry.org/dsm5.

The measure was found to be clinically useful and to have good reliability in the DSM-5 Field Trials that were conducted in adult clinical samples across the United States and in Canada. In the DSM-5 Field Trials, in which the individual's symptom ratings were shared with the clinician before meeting, individuals reported that the results from the measure helped facilitate communication during the clinical encounter. Similarly, clinicians in both major academic-medical research institutions as well as routine clinical practice settings found the measures clinically useful and feasible for integration into everyday clinical care as well as specialty clinical settings. In addition to results from the DSM-5 Field Trials, several studies have evaluated the psychometric properties of the adult self-rated version of the cross-cutting symptom measure in a variety of populations. For example, findings from a large study of non-treatment-seeking college students across the United States demonstrated acceptable internal consistency and internal validity.

The parent/guardian-rated version of the measure (for children ages 6–17) consists of 25 questions that assess 12 psychiatric domains, including depression, anger, irritability, mania, anxiety, somatic symptoms, inattention, suicidal ideation/attempt,

psychosis, sleep disturbance, repetitive thoughts and behaviors, and substance use (Table 2). Each item asks the parent or guardian to rate how much (or how often) his or her child has been bothered by the specific psychiatric symptom during the past 2 weeks. The measure was also found to be clinically useful and to have good reliability in the DSM-5 Field Trials that were conducted in pediatric clinical samples across the United States. For children ages 11–17, along with the parent/guardian rating of the child's symptoms, the clinician may consider having the child complete the child-rated version of the measure. The child-rated version of the measure can be found online at www.psychiatry.org/dsm5.

TABLE 2 Parent/guardian-rated DSM-5 Level 1 Cross-Cutting Symptom Measure for child age 6–17: 12 domains, thresholds for further inquiry, and associated Level 2 measures

Domain	Domain name	Threshold to guide further inquiry	DSM-5 Level 2 Cross-Cutting Symptom Measure[a]
I.	Somatic symptoms	Mild or greater	Level 2—Somatic Symptoms—Parent/Guardian of Child Age 6–17 (Patient Health Questionnaire–15 [PHQ-15] Somatic Symptom Severity Scale)
II.	Sleep problems	Mild or greater	Level 2—Sleep Disturbance—Parent/Guardian of Child Age 6–17 (PROMIS Sleep Disturbance—Short Form)
III.	Inattention	Slight or greater	Level 2—Inattention—Parent/Guardian of Child Age 6–17 (Swanson, Nolan, and Pelham, Version IV [SNAP-IV])
IV.	Depression	Mild or greater	Level 2—Depression—Parent/Guardian of Child Age 6–17 (PROMIS Emotional Distress—Depression—Parent Item Bank)
V.	Anger	Mild or greater	Level 2—Anger—Parent/Guardian of Child (PROMIS Calibrated Anger Measure—Parent)
VI.	Irritability	Mild or greater	Level 2—Irritability—Parent/Guardian of Child (Affective Reactivity Index [ARI])
VII.	Mania	Mild or greater	Level 2—Mania—Parent/Guardian of Child Age 6–17 (Altman Self-Rating Mania Scale [ASRM])
VIII.	Anxiety	Mild or greater	Level 2—Anxiety—Parent/Guardian of Child Age 6–17 (PROMIS Emotional Distress—Anxiety—Parent Item Bank)
IX.	Psychosis	Slight or greater	None
X.	Repetitive thoughts and behaviors	Mild or greater	None
XI.	Substance use	Yes	Level 2—Substance Use—Parent/Guardian of Child Age 6–17 (adapted from the NIDA-modified ASSIST)
		Don't Know	NIDA-modified ASSIST (adapted)—Child-Rated (age 11–17 years)

Domain	Domain name	Threshold to guide further inquiry	DSM-5 Level 2 Cross-Cutting Symptom Measure[a]
XII.	Suicidal ideation/suicide attempts	Yes	None
		Don't Know	None

Note. NIDA = National Institute on Drug Abuse.
[a]Available at www.psychiatry.org/dsm5.

Scoring and interpretation. On the adult self-rated version of the measure, each item is rated on a 5-point scale (0 = none or not at all; 1 = slight or rare, less than a day or two; 2 = mild or several days; 3 = moderate or more than half the days; and 4 = severe or nearly every day). The score on each item within a multi-item domain should be reviewed by the clinician, especially if a Level 2 cross-cutting symptom assessment is not indicated, to understand which specific symptom within a domain is most problematic (e.g., auditory hallucinations or thought broadcasting for the psychosis domain) to help guide further inquiry. However, a rating of mild (i.e., 2) or greater on any item within a domain, except for substance use, suicidal ideation, and psychosis, strongly suggests the need for

additional inquiry and follow-up to determine if a more detailed assessment is necessary, which may include the Level 2 cross-cutting symptom assessment for the domain (see 1). For substance use, suicidal ideation, and psychosis, a rating of slight (i.e., 1) or greater on any item within the domain may serve as a guide for additional inquiry and follow-up to determine if a more detailed assessment is needed. As such, the rater should indicate the highest score within a domain in the "Highest domain score" column. Table 1 outlines threshold scores that may guide further inquiry for the remaining domains.

On the parent/guardian-rated version of the measure (for children ages 6–17), 19 of the 25 items are each rated on a 5-point scale (0 = none or not at all; 1 = slight or rare, less than a day or two; 2 = mild or several days; 3 = moderate or more than half the days; and 4 = severe or nearly every day). The suicidal ideation, suicide attempt, and substance abuse items are each rated on a "Yes, No, or Don't Know" scale. The score on each item within a domain should be reviewed by the clinician to understand which specific symptom within a domain is most problematic (e.g., visual or auditory hallucination on the psychosis domain) to help guide further inquiry. However, with the exception of inattention and psychosis, a rating of mild (i.e., 2) or greater on any item within a domain that is scored on the 5-point scale may serve as a guide for additional inquiry and follow-up to determine if a more detailed assessment is necessary, which may include the Level 2 cross-cutting symptom assessment for the domain (see Table 2). For inattention or psychosis, a rating of slight or greater (i.e., 1 or greater) may be used as an indicator for additional inquiry. A parent or guardian's rating of "Don't Know" on the suicidal ideation, suicide attempt, and any of the

substance use items, especially for children ages 11–17 years, may result in additional probing of the issues with the child, including using the child-rated Level 2 Cross-Cutting Symptom Measure for the relevant domain. Because additional inquiry is made on the basis of the highest score on any item within a domain, clinicians should indicate that score in the "Highest Domain Score" column. Table 2 outlines threshold scores that may guide further inquiry for the remaining domains.

The clinician instructions and guidelines for the child-rated version are similar to those of the parent/guardian-rated version described above with the exception of the "Don't Know" response categories, which are not present in the child-rated version (see www.psychiatry.org/dsm5).

Level 2 Cross-Cutting Symptom Measures

Any threshold scores on the Level 1 Cross-Cutting Symptom Measure (as noted in Tables 1 and 2 and described in "Scoring and Interpretation") indicate a possible need for detailed clinical inquiry. Level 2 Cross-Cutting Symptom Measures provide one method of obtaining more in-depth information on potentially significant symptoms to inform diagnosis, treatment planning, and follow-up. They are available online at www.psychiatry.org/dsm5. Tables 1 and 2 outline each Level 1 domain and identify the domains for which DSM-5 Level 2 Cross-Cutting Symptom Measures are available for more detailed assessments. Adult and pediatric (parent and child) versions are available online for most Level 1 symptom domains.

Frequency of Use of the Cross-Cutting Symptom Measures

To track change in the individual's symptom presentation over time, the Level 1 and relevant Level 2 cross-cutting symptom measures may be completed at regular intervals as clinically indicated, depending on the stability of the individual's symptoms and treatment status. For individuals with impaired capacity and for children ages 6–17 years, it is preferable for the measures to be completed at follow-up appointments by the same knowledgeable informant and by the same parent or guardian. Consistently high scores on a particular domain may indicate significant and problematic symptoms for the individual that might warrant further assessment, treatment, and follow-up. Clinical judgment should guide decision making.

DSM-5 Self-Rated Level 1 Cross-Cutting Symptom Measure—Adult

Name:_____ **Age:** _____ **Date:**_____

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If the measure is being completed by an informant, **what is your relationship with the individual?:** _____

In a typical week, approximately how much time do you spend with the individual? _____ **hours/week**

Instructions: The questions below ask about things that might have bothered you. For each question, circle the number that best describes how much (or how often) you have been bothered by each problem during the **past TWO (2) WEEKS.**

		During the past **TWO (2) WEEKS,** how much (or how often) have you been bothered by the following problems?	**None** Not at all	**Slight** Rare, less than a day or two	**Mild** Several days	**Moderate** More than half the days	**Severe** Nearly every day	**Highest Domain Score** (clinician)
I.	1.	Little interest or pleasure in doing things?	0	1	2	3	4	
	2.	Feeling down, depressed, or hopeless?	0	1	2	3	4	
II.	3.	Feeling more irritated, grouchy, angry than usual?	0	1	2	3	4	
III.	4.	Sleeping less than usual, but still have a lot of energy?	0	1	2	3	4	
	5.	Starting lots more projects than usual or doing more risky things than usual?	0	1	2	3	4	
IV.	6.	Feeling nervous, anxious, frightened, worried, or on edge?	0	1	2	3	4	
	7.	Feeling panic or being frightened?	0	1	2	3	4	
	8.	Avoiding situations that make you anxious?	0	1	2	3	4	
V.	9.	Unexplained aches and pains (e.g., head, back, joints, abdomen, legs)?	0	1	2	3	4	
	10.	Feeling that your illnesses are not being taken seriously enough?	0	1	2	3	4	
VI.	11.	Thoughts of actually hurting yourself?	0	1	2	3	4	
848 VII.	12.	Hearing things other people couldn't hear, such as voices even when no one was around?	0	1	2	3	4	
	13.	Feeling that someone could hear your thoughts, or that you could hear what another person was thinking?	0	1	2	3	4	
VIII.	14.	Problems with sleep that affected your sleep quality overall?	0	1	2	3	4	
IX.	15.	Problems with memory (e.g., learning new information) or with location (e.g., finding your way home)?	0	1	2	3	4	
X.	16.	Unpleasant thoughts, urges, or images that repeatedly enter your mind?	0	1	2	3	4	
	17.	Feeling driven to perform certain behaviors or mental acts over and over again?	0	1	2	3	4	
XI.	18.	Feeling detached or distant from yourself, your body,	0	1	2	3	4	

		your physical surroundings, or your memories?						
XII.	19.	Not knowing who you really are or what you want out of life?	0	1	2	3	4	
	20.	Not feeling close to other people or enjoying your relationships with them?	0	1	2	3	4	
XIII.	21.	Drink at least 4 drinks of any kind of alcohol in a single day?	0	1	2	3	4	
	22.	Smoke any cigarettes, a cigar, or pipe, or use snuff or chewing tobacco?	0	1	2	3	4	
	23.	Use any of the following medicines ON YOUR OWN, that is, without a doctor's prescription, in greater amounts or longer than prescribed [e.g., painkillers (like Vicodin), stimulants (like Ritalin or Adderall), sedatives or tranquilizers (like sleeping pills or Valium), or drugs like marijuana, cocaine or crack, club drugs (like ecstasy), hallucinogens (like LSD), heroin, inhalants or solvents (like glue), or methamphetamine (like speed)]?	0	1	2	3	4	

Parent/Guardian-Rated DSM-5 Level 1 Cross-Cutting Symptom Measure—Child Age 6–17

Child's Name:_____ **Age:** _____ **Date:**_____

Relationship to the child: _____

Instructions (*to parent or guardian of child*): The questions below ask about things that might have bothered your child. For each question, circle the number that best describes how much (or how often) your child has been bothered by each problem during the **past TWO (2) WEEKS.**

	During the past **TWO (2) WEEKS,** how much (or how often) has your child…		**None** Not at all	**Slight** Rare, less than a day or two	**Mild** Several days	**Moderate** More than half the days	**Severe** Nearly every day	**Highest Domain Score** (clinician)
I.	1.	Complained of stomachaches, headaches, or other aches and pains?	0	1	2	3	4	
	2.	Said he/she was worried about his/her health or about getting sick?	0	1	2	3	4	
II.	3.	Had problems sleeping—	0	1	2	3	4	

		that is, trouble falling asleep, staying asleep, or waking up too early?						
III.	4.	Had problems paying attention when he/she was in class or doing his/her homework or reading a book or playing a game?	0	1	2	3	4	
IV.	5.	Had less fun doing things than he/she used to?	0	1	2	3	4	
	6.	Seemed sad or depressed for several hours?	0	1	2	3	4	
V. andVI.	7.	Seemed more irritated or easily annoyed than usual?	0	1	2	3	4	
	8.	Seemed angry or lost his/her temper?	0	1	2	3	4	
VII.	9.	Starting lots more projects than usual or doing more risky things than usual?	0	1	2	3	4	
	10.	Sleeping less than usual for him/her but still has lots of energy?	0	1	2	3	4	
VIII.	11.	Said he/she felt nervous, anxious, or scared?	0	1	2	3	4	
	12.	Not been able to stop worrying?	0	1	2	3	4	
	13.	Said he/she couldn't do things he/she wanted to or should have done because they made him/her feel nervous?	0	1	2	3	4	
850 IX.	14.	Said that he/she heard voices—when there was no one there—speaking about him/her or telling him/her what to do or saying bad things to him/her?	0	1	2	3	4	
	15.	Said that he/she had a vision when he/she was completely awake—that is, saw something or someone that no one else could see?	0	1	2	3	4	
X.	16.	Said that he/she had thoughts that kept coming into his/her mind that he/she would do something bad or that something bad would happen to him/her or to someone else?	0	1	2	3	4	
	17.	Said he/she felt the need to check on certain	0	1	2	3	4	

		things over and over again, like whether a door was locked or whether the stove was turned off?						
	18.	Seemed to worry a lot about things he/she touched being dirty or having germs or being poisoned?	0	1	2	3	4	
	19.	Said that he/she had to do things in a certain way, like counting or saying special things out loud, in order to keep something bad from happening?	0	1	2	3	4	
	In the past **TWO (2) WEEKS,** has your child …							
XI.	20.	Had an alcoholic beverage (beer, wine, liquor, etc.)?	❑ Yes		❑ No	❑ Don't Know		
	21.	Smoked a cigarette, a cigar, or pipe, or used snuff or chewing tobacco?	❑ Yes		❑ No	❑ Don't Know		
	22.	Used drugs like marijuana, cocaine or crack, club drugs (like ecstasy), hallucinogens (like LSD), heroin, inhalants or solvents (like glue), or methamphetamine (like speed)?	❑ Yes		❑ No	❑ Don't Know		
	23.	Used any medicine without a doctor's prescription (e.g., painkillers [like Vicodin], stimulants [like Ritalin or Adderall], sedatives or tranquilizers [like sleeping pills or Valium], or steroids)?	❑ Yes		❑ No	❑ Don't Know		
XII.	24.	In the **past TWO (2) WEEKS,** has he/she talked about wanting to kill himself/herself or about wanting to commit suicide?	❑ Yes		❑ No	❑ Don't Know		
	25.	Has he/she EVER tried to kill himself/herself?	❑ Yes		❑ No	❑ Don't Know		

Clinician-Rated Dimensions of Psychosis Symptom Severity

As described in the chapter “Schizophrenia Spectrum and Other Psychotic Disorders,” psychotic disorders are heterogeneous, and symptom severity can predict important aspects of the illness, such as the degree of cognitive and/or neurobiological deficits. Dimensional assessments capture meaningful variation in the severity of symptoms, which may help with treatment planning, prognostic decision-making, and research on pathophysiological mechanisms. The Clinician-Rated Dimensions of Psychosis Symptom Severity measure provides scales for the dimensional assessment of the primary symptoms of psychosis, including hallucinations, delusions, disorganized speech, abnormal psychomotor behavior, and negative symptoms. A scale for the dimensional assessment of cognitive impairment is also included. Many individuals with psychotic disorders have impairments in a range of cognitive domains, which predict functional abilities and prognosis. In addition, scales for dimensional assessment of depression and mania are provided, which may alert clinicians to co-occurring mood pathology. The severity of mood symptoms in psychosis has prognostic value and can guide treatment.

The Clinician-Rated Dimensions of Psychosis Symptom Severity is an 8-item measure that may be completed by the clinician at the time of the clinical assessment. Each item asks the clinician to rate the severity of each symptom as experienced by the individual when it was at its most severe during the past 7 days.

Scoring and Interpretation

Each item on the measure is rated on a 5-point scale (0 = none; 1 = equivocal; 2 = present, but mild; 3 = present and moderate; and 4 = present and severe) with a symptom-specific definition of each rating level. The clinician reviews all of the individual’s available information and, based on clinical judgment, selects (with checkmark) the level that most accurately describes the severity of the symptom domain. The clinician then indicates the score for each item in the “Score” column provided.

Frequency of Use

To track changes in the individual’s symptom severity over time, the measure may be completed at regular intervals as clinically indicated, depending on the stability of the individual’s symptoms and treatment status. Consistently high scores on a particular domain may indicate significant and problematic areas for the individual that might warrant further assessment, treatment, and follow-up. Clinical judgment should always guide decision making.

Clinician-Rated Dimensions of Psychosis Symptom Severity

Name:_____ **Age:** _____ **Date:**_____

Instructions: Based on all the information you have on the individual and using your clinical judgment, please rate (with checkmark) the presence and severity of the following symptoms as experienced by the individual, when each symptom was at its most severe, in the past seven (7) days.

Domain	0	1	2	3	4	Score
I. Hallucinations	❑ Not present	❑ Equivocal (severity or duration not sufficient to be considered psychosis)	❑ Present, but mild (little pressure to act upon voices or other types of hallucinations, not very bothered by hallucinations)	❑ Present and moderate (some pressure to respond to voices or other types of hallucinations, or is somewhat bothered by hallucinations)	❑ Present and severe (severe pressure to respond to voices or other types of hallucinations, or is very bothered by hallucinations)	
II. Delusions	❑ Not present	❑ Equivocal (severity or duration not sufficient to be considered psychosis)	❑ Present, but mild (little pressure to act upon delusional beliefs, not very bothered by such beliefs)	❑ Present and moderate (some pressure to act upon delusional beliefs, or is somewhat bothered by such beliefs)	❑ Present and severe (severe pressure to act upon delusional beliefs, or is very bothered by such beliefs)	
III. Disorganized speech	❑ Not present	❑ Equivocal (severity or duration not sufficient to be considered disorganization)	❑ Present, but mild (some difficulty following speech)	❑ Present and moderate (speech often difficult to follow)	❑ Present and severe (speech almost impossible to follow)	
IV. Abnormal psychomotor behavior	❑ Not present	❑ Equivocal (severity or duration not sufficient to be considered abnormal psychomotor behavior)	❑ Present, but mild (occasional abnormal or bizarre motor behavior or catatonia)	❑ Present and moderate (frequent abnormal or bizarre motor behavior or catatonia)	❑ Present and severe (abnormal or bizarre motor behavior or catatonia almost constant)	
853 V. Negative symptoms (restricted emotional expression or avolition)	❑ Not present	❑ Equivocal decrease in facial expressivity, prosody, gestures, or self-initiated behavior	❑ Present, but mild decrease in facial expressivity, prosody, gestures, or self-initiated behavior	❑ Present and moderate decrease in facial expressivity, prosody, gestures, or self-initiated behavior	❑ Present and severe decrease in facial expressivity, prosody, gestures, or self-initiated behavior	
VI. Impaired cognition	❑ Not present	❑ Equivocal (cognitive function not clearly outside the range expected for age or SES; i.e., within 0.5 SD of mean)	❑ Present, but mild (some reduction in cognitive function; below expected for age and SES, 0.5–1 SD from mean)	❑ Present and moderate (clear reduction in cognitive function; below expected for age and SES, 1–2 SD from mean)	❑ Present and severe (severe reduction in cognitive function; below expected for age and SES, > 2 SD from mean)	
VII. Depression	❑ Not present	❑ Equivocal (occasionally feels sad, down, depressed, or hopeless; concerned about having failed someone or at something but not preoccupied)	❑ Present, but mild (frequent periods of feeling very sad, down, moderately depressed, or hopeless;	❑ Present and moderate (frequent periods of deep depression or hopelessness; preoccupation	❑ Present and severe (deeply depressed or hopeless daily; delusional guilt or unreasonable self-reproach grossly out of	

			hopeless, concerned about having failed someone or at something, with some preoccupation)	preoccupation with guilt, having done wrong)	grossly out of proportion to circumstances)	
VIII. Mania	❑ Not present	❑ Equivocal (occasional elevated, expansive, or irritable mood or some restlessness)	❑ Present, but mild (frequent periods of somewhat elevated, expansive, or irritable mood or restlessness)	❑ Present and moderate (frequent periods of extensively elevated, expansive, or irritable mood or restlessness)	❑ Present and severe (daily and extensively elevated, expansive, or irritable mood or restlessness)	

Note. SD = standard deviation; SES = socioeconomic status.

World Health Organization Disability Assessment Schedule 2.0

The adult self-administered version of the World Health Organization Disability Assessment Schedule 2.0 (WHODAS 2.0) is a 36-item measure that assesses disability in adults age 18 years and older. It has been validated across numerous cultures worldwide and demonstrated sensitivity to change. It assesses disability across six domains, including understanding and communicating, getting around, self-care, getting along with people, life activities (i.e., household, work, and/or school activities), and participation in society. If the adult individual is of impaired capacity and unable to complete the form (e.g., a patient with major neurocognitive disorder), a knowledgeable informant may complete the proxy-administered version of the measure, which is available at www.psychiatry.org/dsm5. Each item on the self-administered version of the WHODAS 2.0 asks the individual to rate how much difficulty he or she has had in specific areas of functioning during the past 30 days.

WHODAS 2.0 Scoring Instructions Provided by WHO

WHODAS 2.0 summary scores. There are two basic options for computing the summary scores for the WHODAS 2.0 36-item full version.

Simple: The scores assigned to each of the items—"none" (1), "mild" (2), "moderate" (3), "severe" (4), and "extreme" (5)—are summed for a maximum total raw score of 180. This method is referred to as simple scoring because the scores from each of the items are simply added up without recoding or collapsing of response categories; thus, there is no weighting of individual items. This approach is practical to use as a hand-scoring approach, and may be the method of choice in busy clinical settings or in paper-and-pencil interview situations. As a result, the simple sum of the scores of the items across all domains constitutes a statistic that is sufficient to describe the degree of functional limitations.

Complex: The more complex method of scoring is called "item-response-theory" (IRT)–based scoring. It takes into account multiple levels of difficulty for each WHODAS 2.0 item. It takes the coding for each item response as "none," "mild," "moderate," "severe," and "extreme" separately, and then requires a computer to determine the summary score by differentially weighting the items and the levels of severity. The computer program is available from the WHO Web site. The scoring has three steps:

- Step 1—Summing of recoded item scores within each domain (i.e., for each item, the response options 1–5 are converted to a rate of 0–4, leading to a total raw score of 144).
- Step 2—Summing of all six domain scores.
- Step 3—Converting the summary score into a metric ranging from 0 to 100 (where 0 = no disability; 100 = full disability).

WHODAS 2.0 domain scores. WHODAS 2.0 produces domain-specific scores for six different functioning domains: cognition, mobility, self-care, getting along, life activities (household and work/school), and participation.

WHODAS 2.0 population norms. For the population norms for IRT-based scoring of the WHODAS 2.0 and for the population distribution of IRT-based scores for WHODAS 2.0, please see www.who.int/classifications/icf/Pop_norms_distrib_IRT_scores.pdf.

Additional Scoring and Interpretation Guidance for DSM-5-TR Users

The clinician is asked to review the individual's response on each item on the measure during the clinical interview and to indicate the self-reported score for each item in the section provided for "Clinician Use Only." However, if the clinician determines that the score on an item should be different based on the clinical interview and other information available, he or she may indicate a corrected score in the raw item score box. Based on findings from the DSM-5 Field Trials in adult patient samples across six sites in the United States and one in Canada, *DSM-5-TR recommends calculation and use of average scores for each domain and for general disability.* The average scores are comparable to the WHODAS 5-point scale, which allows the clinician to think of the individual's disability in terms of none (1), mild (2), moderate (3), severe (4), or extreme (5). The average domain and general disability scores were found to be reliable, easy to use, and clinically useful to the clinicians in the DSM-5 Field Trials. The *average domain score* is calculated by dividing the raw domain score by the number of items in the domain (e.g., if all the items within the "understanding and communicating" domain are rated as being moderate, then the average domain score would be 18/6 = 3, indicating moderate disability). The *average general disability score* is calculated by dividing the raw overall score by number of items in the measure (i.e., 36). The individual should be encouraged to complete all of the items on the WHODAS 2.0. If no response is given on 10 or more items of the measure (i.e., more

than 25% of the 36 total items), calculation of the simple and average general disability scores may not be helpful. If 10 or more of the total items on the measure are missing but the items for some of the domains are 75%–100% complete, the simple or average domain scores may be used for those domains.

Frequency of Use

To track change in the individual's level of disability over time, the measure may be completed at regular intervals as clinically indicated, depending on the stability of the individual's symptoms and treatment status. Consistently high scores on a particular domain may indicate significant and problematic areas for the individual that might warrant further assessment and intervention.

WHODAS 2.0

World Health Organization Disability Assessment Schedule 2.0

36-item version, self-administered

Patient Name: _____ **Age:** _____ **Date:** _____

This questionnaire asks about difficulties due to health/mental health conditions. Health conditions include **diseases or illnesses, other health problems that may be short or long lasting, injuries, mental or emotional problems, and problems with alcohol or drugs.** Think back over the **past 30 days** and answer these questions thinking about how much difficulty you had doing the following activities. For each question, please circle only **one** response.

							Clinician Use Only		
Numeric scores assigned to each of the items:		1	2	3	4	5	Raw Item Score	Raw Domain Score	Average Domain Score
In the last 30 days, how much difficulty did you have in:									
Understanding and communicating									
D1.1	Concentrating on doing something for ten minutes?	None	Mild	Moderate	Severe	Extreme or cannot do			
D1.2	Remembering to do important things?	None	Mild	Moderate	Severe	Extreme or cannot do			
D1.3	Analyzing and finding solutions to problems in day-to-day life?	None	Mild	Moderate	Severe	Extreme or cannot do			
D1.4	Learning a new task, for example, learning how to get to a new place?	None	Mild	Moderate	Severe	Extreme or cannot do		30	5
D1.5	Generally understanding what people say?	None	Mild	Moderate	Severe	Extreme or cannot do			
D1.6	Starting and maintaining a conversation?	None	Mild	Moderate	Severe	Extreme or cannot do			
Getting around									
D2.1	Standing for long periods, such as 30 minutes?	None	Mild	Moderate	Severe	Extreme or cannot do			
D2.2	Standing up from sitting down?	None	Mild	Moderate	Severe	Extreme or cannot do			
D2.3	Moving around inside your home?	None	Mild	Moderate	Severe	Extreme or cannot do			
D2.4	Getting out of your home?	None	Mild	Moderate	Severe	Extreme or cannot do		25	5
D2.5	Walking a long distance, such as a kilometer (or equivalent)?	None	Mild	Moderate	Severe	Extreme or cannot do			
Self-care									
D3.1	Washing your whole body?	None	Mild	Moderate	Severe	Extreme or cannot do			
D3.2	Getting dressed?	None	Mild	Moderate	Severe	Extreme or cannot do			
D3.3	Eating?	None	Mild	Moderate	Severe	Extreme or cannot do		20	5
D3.4	Staying by yourself for a few days?	None	Mild	Moderate	Severe	Extreme or cannot do			
Getting along with people									
D4.1	Dealing with people you do not know?	None	Mild	Moderate	Severe	Extreme or cannot do			
D4.2	Maintaining a friendship?	None	Mild	Moderate	Severe	Extreme or cannot do			
D4.3	Getting along with people who are close to you?	None	Mild	Moderate	Severe	Extreme or cannot do			
D4.4	Making new friends?	None	Mild	Moderate	Severe	Extreme or cannot do		25	5
D4.5	Sexual activities?	None	Mild	Moderate	Severe	Extreme or cannot do			

							Clinician Use Only		
Numeric scores assigned to each of the items:		1	2	3	4	5	Raw Item Score	Raw Domain Score	Average Domain Score
In the last 30 days, how much difficulty did you have in:									
Life activities—Household									
D5.1	Taking care of your household responsibilities?	None	Mild	Moderate	Severe	Extreme or cannot do			
D5.2	Doing most important household tasks well?	None	Mild	Moderate	Severe	Extreme or cannot do			
D5.3	Getting all of the household work done that you needed to do?	None	Mild	Moderate	Severe	Extreme or cannot do		20	5
D5.4	Getting your household work done as quickly as needed?	None	Mild	Moderate	Severe	Extreme or cannot do			
Life activities—School/Work									
If you work (paid, non-paid, self-employed) or go to school, complete questions D5.5–D5.8, below. Otherwise, skip to D6.1.									
Because of your health condition, in the past 30 days, how much difficulty did you have in:									
D5.5	Your day-to-day work/school?	None	Mild	Moderate	Severe	Extreme or cannot do			
D5.6	Doing your most important work/school tasks well?	None	Mild	Moderate	Severe	Extreme or cannot do			
D5.7	Getting all of the work done that you need to do?	None	Mild	Moderate	Severe	Extreme or cannot do		20	5
D5.8	Getting your work done as quickly as needed?	None	Mild	Moderate	Severe	Extreme or cannot do			
Participation in society									
In the past 30 days:									
D6.1	How much of a problem did you have in joining in community activities (for example, festivities, religious, or other activities) in the same way as anyone else can?	None	Mild	Moderate	Severe	Extreme or cannot do			
D6.2	How much of a problem did you have because of barriers or hindrances around you?	None	Mild	Moderate	Severe	Extreme or cannot do			
D6.3	How much of a problem did you have living with dignity because of the attitudes and actions of others?	None	Mild	Moderate	Severe	Extreme or cannot do			
D6.4	How much time did you spend on your health condition or its consequences?	None	Some	Moderate	A Lot	Extreme or cannot do		40	5
D6.5	How much have you been emotionally affected by your health condition?	None	Mild	Moderate	Severe	Extreme or cannot do			
D6.6	How much has your health been a drain on the financial resources of you or your family?	None	Mild	Moderate	Severe	Extreme or cannot do			
D6.7	How much of a problem did your family have because of your health problems?	None	Mild	Moderate	Severe	Extreme or cannot do			
D6.8	How much of a problem did you have in doing things by yourself for relaxation or pleasure?	None	Mild	Moderate	Severe	Extreme or cannot do			
						General Disability Score (Total):		180	5

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Culture and Psychiatric Diagnosis

This chapter provides basic information on integrating culture and social context in clinical diagnoses, with sections on key terms, cultural formulation, and cultural concepts of distress.

- The first section defines terms that are essential to the rest of the chapter: *culture*, *race*, and *ethnicity*.
- The Cultural Formulation section presents an outline for a systematic person-centered cultural assessment that is designed to be used by any clinician providing services to any individual in any care setting. This section also includes an interview protocol, the Cultural Formulation Interview, that operationalizes these components. Symptom presentations, interpretations of the illness or predicament that precipitates care, and help-seeking expectations are always influenced by individuals' cultural backgrounds and sociocultural contexts. A person-centered cultural assessment can help improve the care of every individual, regardless of his or her background. Cultural formulation may be especially helpful for individuals who are affected by healthcare disparities driven by systemic disadvantage and discrimination.
- The Cultural Concepts of Distress section describes the ways individuals express, report, and interpret experiences of illness and distress. Cultural concepts of distress include idioms, explanations or perceived causes, and syndromes. Symptoms are expressed and communicated using *cultural idioms of distress*—behaviors or linguistic terms, metaphors, phrases, or ways of talking about symptoms, problems, or suffering that are commonly used by individuals with similar cultural backgrounds to convey a wide range of concerns. Such idioms may be used for a broad spectrum of distress and may not indicate a psychiatric disorder. Common contemporary idioms in the United States include "burnout," "feeling stressed," "nervous breakdown," and "feeling depressed," in the sense of experiencing dissatisfaction or discouragement that does not meet criteria for any psychiatric disorder. Culturally specific explanations and syndromes are also common and distributed widely across populations. This section also provides some illustrative examples of idioms, explanations, and syndromes from diverse geographic regions. The examples were chosen because they have been well studied and their lack of familiarity to

many U.S. clinicians highlights their specific verbal and behavioral expressions and communicative functions.

Key Terms

Understanding the cultural context of illness experience is essential for effective diagnostic assessment and clinical management.

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Culture refers to systems of knowledge, concepts, values, norms, and practices that are learned and transmitted across generations. Culture includes language, religion and spirituality, family structures, life-cycle stages, ceremonial rituals, customs, and ways of understanding health and illness, as well as moral, political, economic, and legal systems. Cultures are open, dynamic systems that undergo continuous change over time; in the contemporary world, most individuals and groups are exposed to multiple cultural contexts, which they use to fashion their own identities and make sense of experience. This process of meaning-making derives from developmental and everyday social experiences in specific contexts, including health care, which may vary for each individual. Much of culture involves background knowledge, values, and assumptions that remain implicit or presumed and so may be difficult for individuals to describe. These features of culture make it crucial not to overgeneralize cultural information or stereotype groups in terms of fixed cultural traits. In relation to diagnosis, it is essential to recognize that all forms of illness and distress, including the DSM disorders, are shaped by cultural contexts. Culture influences how individuals fashion their identities, as well as how they interpret and respond to symptoms and illness.

Race is a social, not a biological, construct that divides humanity into groups based on a variety of superficial physical traits such as skin color that have been falsely viewed as indicating attributes and capacities assumed to be inherent to the group. Racial categories and constructs have varied over history and across societies and have been used to justify systems of oppression, slavery, and genocide. The construct of race is

important for psychiatry because it can lead to racial ideologies, racism, discrimination, and social oppression and exclusion, which have strong negative effects on mental health. There is evidence that racism can exacerbate many psychiatric disorders, contributing to poor outcome, and that racial biases can affect diagnostic assessment.

Ethnicity is a culturally constructed group identity used to define peoples and communities. It may be rooted in a common history, ancestry, geography, language, religion, or other shared characteristics of a group, which distinguish that group from others. Ethnicity may be self-assigned or attributed by outsiders. Increasing mobility, intermarriage, and intermixing of cultural groups have defined new mixed, multiple, or hybrid ethnic identities. These processes may also lead to the dilution of ethnic identification.

Culture, race, and ethnicity may be related to political, economic, and social structural inequities associated with racism and discrimination resulting in health disparities. Cultural, ethnic, and racialized identities can be sources of strength and group support that enhance resilience. They may also lead to psychological, interpersonal, and intergenerational conflict or difficulties in adaptation that require socially and culturally informed diagnosis and clinical assessment. Additional key terms related to racialization and racism are defined in the DSM-5-TR Section I Introduction, under “Cultural and Social Structural Issues,” in the subsection “Impact of Racism and Discrimination on Psychiatric Diagnosis.”

Cultural Formulation

Outline for Cultural Formulation

The Outline for Cultural Formulation introduced in DSM-IV provided a framework for assessing information about cultural features of an individual’s mental health problem and how it relates to a social and cultural context and history. This assessment provides useful information on social context and illness experience relevant to the assessment of every

individual, not only those whose cultural background may be unfamiliar to the clinician.

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Updated from DSM-5, DSM-5-TR includes an expanded version of the Outline and an approach to assessment using the Cultural Formulation Interview (CFI), which has been field-tested among clinicians, patients, and accompanying relatives and found to be a feasible, acceptable, and useful cultural assessment tool.

The Outline for Cultural Formulation calls for systematic assessment of the following categories:

- **Cultural identity of the individual:** Describe the individual's demographic (e.g., age, gender, ethnoracial background) or other socially and culturally defined characteristics that may influence interpersonal relationships, access to resources, and developmental and current challenges, conflicts, or predicaments. Other clinically relevant aspects of identity may include religious affiliation and spirituality, socioeconomic class, caste, personal and family places of birth and growing up, migrant status, occupation, and sexual orientation, among others. Note which aspects of identity are prioritized by the individual and how they interact (intersectionality), which may reflect the influence of clinical setting and health concerns. For migrants, the degree and kinds of involvement with both the cultural contexts of origin and the new cultural contexts should be noted. Similarly, for individuals who identify with racialized and ethnic groups, the degree of interaction and identification with their own group and other segments of society should be noted. Language abilities, preferences, and patterns of use are relevant for identifying difficulties with access to care, social integration, and clinical communication or the need for an interpreter.
- **Cultural concepts of distress:** Describe the cultural constructs that influence how the individual experiences, understands, and communicates his or her symptoms or problems to others. These constructs include cultural idioms of distress, cultural explanations or perceived causes, and cultural syndromes. The level of severity and meaning of the distressing experiences should be assessed in relation to the norms of the individual's cultural background. Priority symptoms, perceived seriousness of the illness, the level of associated stigma, and anticipated outcomes are all relevant. Elicit the individual's and family's or friends' help-seeking expectations and plans, as well as patterns of self-coping and their connection to the individual's cultural concepts of distress, including past help-seeking experiences. Assessment of coping and

help-seeking patterns should consider the use of professional as well as traditional, alternative, or complementary sources of care.

- **Psychosocial stressors and cultural features of vulnerability and resilience:** Identify key stressors, challenges, and supports in the individual's social environment (which may include both local and distant events). These include social determinants of the individual's mental health such as access to resources (e.g., housing, transportation) and opportunities (e.g., education, employment); exposure to racism, discrimination, and systemic institutional stigmatization; and social marginalization or exclusion (structural violence). Also assess the role of religion, family, and other interpersonal relationships and social networks (e.g., friends, neighbors, coworkers, online forums or groups) in causing stress or providing emotional, instrumental, and informational support. Social stressors and social supports vary with social context, family structure, developmental tasks, and the cultural meaning of events. Levels of functioning, disability, and resilience should be assessed in light of the individual's cultural background.
- **Cultural features of the relationship between the individual and the clinician, treatment team, and institution:** Identify differences in cultural background, language, education, and social status among other aspects of identity between an individual and clinician (or the treatment team and institution) that may cause difficulties in communication and may influence diagnosis and treatment. Considering the ways that individuals

and clinicians are positioned socially and perceive each other in terms of social categories may influence the assessment process. Experiences of racism and discrimination in the larger society may impede establishing trust and safety in the clinical diagnostic encounter. Effects may include problems eliciting symptoms, misunderstanding of the cultural and clinical significance of symptoms and behaviors, and difficulty establishing or maintaining the rapport needed for accurate assessment and an effective clinical alliance.

- **Overall cultural assessment:** Summarize the implications of the components of the cultural formulation identified in earlier sections of the Outline for the differential diagnosis of mental disorders and other clinically relevant issues or problems, as well as appropriate management and treatment intervention.

Cultural Formulation Interview (CFI)

The Cultural Formulation Interview (CFI) is a set of protocols that clinicians may use to obtain information during a mental health assessment about the impact of culture on key aspects of an individual's clinical presentation and care. The CFI consists of three components: the core CFI, a set of 16 questions that can be used to obtain an initial assessment from any individual; an Informant version of the core CFI to obtain collateral information; and a set of Supplementary modules to expand the evaluation as needed. In the CFI, the term *culture* includes:

- The processes through which individuals assign meaning to experience, drawing from the values, orientations, knowledge, and practices of the diverse social groups (e.g., ethnic groups, faith groups, occupational groups, veterans' groups) and communities in which they participate.
- Aspects of individuals' background, developmental experiences, and current social contexts and position that affect their perspective, such as age, gender, social class, geographic origin, migration, language, religion, sexual orientation, disability, or ethnic or racialized background.
- The influence of family, friends, and other community members (particularly, the individual's *social network*) on the individual's illness experience.
- The cultural background of the health care providers and the values and assumptions embedded in the organization and practices of health care systems and institutions that may affect the clinical interaction.

Cultural processes involve interactions of the individual with local and larger social contexts. A cultural assessment thus evaluates processes both within the individual and in the social world, assessing the context as much as the person.

The CFI is a brief semistructured interview for systematically assessing cultural factors relevant to the care of any individual. The CFI focuses on the individual's experience and the social contexts of the clinical problem, symptoms, or concerns. The CFI follows a person-centered approach to cultural assessment by eliciting information from the individual about his or her own views and those of others in his or her social network. This approach is designed to avoid stereotyping, in that each individual's cultural knowledge affects how he or she interprets illness experience and guides how he or she seeks help. Because the CFI concerns the individual's personal views, there are no right or wrong answers to these questions. The

core CFI (and informant version) is included later in this chapter and is available online at www.psychiatry.org/dsm5; the Supplementary modules are also available online.

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The core CFI (and informant version) is formatted as two text columns. The left-hand column contains the instructions for administering the CFI and describes the goals for each interview domain. The questions in the right-hand column illustrate how to explore these domains, but they are not meant to be exhaustive. Follow-up questions may be needed to clarify individuals' answers. Questions may be rephrased as needed. The CFI is intended as a guide to cultural assessment and should be used flexibly to maintain a natural flow of the interview and rapport with the individual.

The CFI is best used in conjunction with demographic information obtained before the interview in order to tailor the CFI questions to address the individual's background and current situation. Specific demographic domains to be explored with the CFI will vary across individuals and settings. A comprehensive assessment may include place of birth, age, gender, ethnic or racialized background, marital status, family composition, education, language fluencies, sexual orientation, religious or spiritual affiliation, occupation, employment, income, and migration history.

The CFI can be used in the initial assessment of individuals at any age, in any clinical setting, regardless of the cultural background of the individual or of the clinician. Individuals and clinicians who appear to share the same cultural background may nevertheless differ in ways that are relevant to care. The CFI may be used in its entirety, or components may be incorporated into a clinical evaluation as needed. The CFI may be especially helpful in clinical practice when any of the following occur:

- Difficulty in diagnostic assessment owing to significant differences in the cultural, religious, or socioeconomic backgrounds of clinician and the individual.
- Uncertainty about the fit between culturally distinctive symptoms and diagnostic criteria.
- Difficulty in judging illness severity or impairment.

- Divergent views of symptoms or expectations of care based on previous experience with other cultural systems of healing and health care.
- Disagreement between the individual and clinician on the course of care.
- Potential mistrust of mainstream services and institutions by individuals with collective histories of trauma and oppression.
- Limited engagement in and adherence to treatment by the individual.

The core CFI emphasizes four domains of assessment: Cultural Definition of the Problem (questions 1–3); Cultural Perceptions of Cause, Context, and Support (questions 4–10); Cultural Factors Affecting Self-Coping and Past Help Seeking (questions 11–13); and Cultural Factors Affecting Current Help Seeking (questions 14–16). Both the person-centered process of conducting the CFI and the information it elicits are intended to enhance the cultural validity of diagnostic assessment, facilitate treatment planning, and promote the individual's engagement and satisfaction. To achieve these goals, the clinician should integrate the information obtained from the CFI with all other available clinical material into a comprehensive clinical and contextual evaluation. An Informant version of the CFI can be used to collect collateral information on the CFI domains from family members or caregivers.

Supplementary modules have been developed that expand on each domain of the core CFI and guide clinicians who wish to explore these domains in greater depth. Supplementary modules have also been developed for specific populations, such as children and adolescents, elderly individuals, caregivers, and immigrants and refugees. These supplementary modules are referenced in the core CFI under the pertinent subheadings and are available online at www.psychiatry.org/dsm5.

Core Cultural Formulation Interview (CFI)

Supplementary modules used to expand each CFI subtopic are noted in parentheses.

GUIDE TO INTERVIEWER	INSTRUCTIONS TO THE INTERVIEWER ARE ***ITALICIZED.***

Core Cultural Formulation Interview (CFI)

The following questions aim to clarify key aspects of the presenting clinical problem from the point of view of the individual and other members of the individual's social network (i.e., family, friends, or others involved in current problem). This includes the problem's meaning, potential sources of help, and expectations for services.

INTRODUCTION FOR THE INDIVIDUAL:

I would like to understand the problems that bring you here so that I can help you more effectively. I want to know about ***your*** experience and ideas. I will ask some questions about what is going on and how you are dealing with it. Please remember there are no right or wrong answers.

CULTURAL DEFINITION OF THE PROBLEM

Cultural Definition of the Problem

(Explanatory Model, Level of Functioning)

Elicit the individual's view of core problems and key concerns.

Focus on the individual's own way of understanding the problem.

Use the term, expression, or brief description elicited in question 1 to identify the problem in subsequent questions (e.g., "your conflict with your son").

1. What brings you here today?
   IF INDIVIDUAL GIVES FEW DETAILS OR ONLY MENTIONS SYMPTOMS OR A MEDICAL DIAGNOSIS, PROBE:
   People often understand their problems in their own way, which may be similar to or different from how doctors describe the problem. How would *you* describe your problem?

Ask how individual frames the problem for members of the social network.

2. Sometimes people have different ways of describing their problem to their family, friends, or others in their community. How would you describe your problem to them?

Focus on the aspects of the problem that matter most to the individual.

3. What troubles you most about your problem?

CULTURAL PERCEPTIONS OF CAUSE, CONTEXT, AND SUPPORT

Causes

(Explanatory Model, Social Network, Older Adults)

This question indicates the meaning of the condition for the individual, which may be relevant for clinical care.

Note that individuals may identify multiple causes, depending on the facet of the problem they are considering.

4. Why do you think this is happening to you? What do you think are the causes of your [PROBLEM]?
   PROMPT FURTHER IF REQUIRED:
   Some people may explain their problem as the result of bad things that happen in their life, problems with others, a physical illness, a spiritual reason, or many other causes.

Focus on the views of members of the individual's social network. These may be diverse and vary from the individual's.

5. What do others in your family, your friends, or others in your community think is causing your [PROBLEM]?

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Stressors and Supports

(Social Network, Caregivers, Psychosocial Stressors, Religion and Spirituality, Immigrants and Refugees, Cultural Identity, Older Adults, Coping and Help Seeking)

Elicit information on the individual's life context, focusing on resources, social supports, and resilience. May also probe other supports (e.g., from co-workers, from participation in religion or spirituality).

6. Are there any kinds of support that make your [PROBLEM] better, such as support from family, friends, or others?

Focus on stressful aspects of the individual's environment. Can also probe, e.g., relationship problems, difficulties at work or school, or discrimination.

7. Are there any kinds of stresses that make your [PROBLEM] worse, such as difficulties with money, or family problems?

Role of Cultural Identity

(Cultural Identity, Psychosocial Stressors, Religion and Spirituality, Immigrants and Refugees, Older Adults, Children and Adolescents)

Sometimes, aspects of people's background or identity can make their [PROBLEM] better or worse. By ***background*** or ***identity,*** I mean, for example, the communities you belong to, the languages you speak, where you or your family are from, your race or ethnic background, your gender or sexual orientation, or your faith or religion.

Ask the individual to reflect on the most salient elements of his or her cultural identity. Use this information to tailor questions 9–10 as needed.

8. For you, what are the most important aspects of your background or identity?

Elicit aspects of identity that make the problem better or worse.

9. Are there any aspects of your background or identity that make a difference to your [PROBLEM]?

Probe as needed (e.g., clinical worsening as a result of discrimination due to migration status, race/ethnicity, or sexual orientation).

Probe as needed (e.g., migration-related problems; conflict across generations or due to gender roles).

10. Are there any aspects of your background or identity that are causing other concerns or difficulties for you?

CULTURAL FACTORS AFFECTING SELF-COPING AND PAST HELP SEEKING

Self-Coping

(Coping and Help Seeking, Religion and Spirituality, Older Adults, Caregivers, Psychosocial Stressors)

Clarify self-coping for the problem.

11. Sometimes people have various ways of dealing with problems like [PROBLEM]. What have you done on your own to cope with your [PROBLEM]?

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Past Help Seeking

(Coping and Help Seeking, Religion and Spirituality, Older Adults, Caregivers, Psychosocial Stressors, Immigrants and Refugees, Social Network, Clinician-Patient Relationship)

Elicit various sources of help (e.g., medical care, mental health treatment, support groups, work-based counseling, folk healing, religious or spiritual counseling, other forms of traditional or alternative healing).

12. Often, people look for help from many different sources, including different kinds of doctors, helpers, or healers. In the past, what kinds of treatment, help, advice, or healing have you sought for your [PROBLEM]?

Probe as needed (e.g., "What other sources of help have you used?").

PROBE IF DOES NOT DESCRIBE USEFULNESS OF HELP RECEIVED:

Clarify the individual's experience and regard for previous help.

What types of help or treatment were most useful? Not useful?

Barriers

(Coping and Help Seeking, Religion and Spirituality, Older Adults, Psychosocial Stressors, Immigrants and Refugees, Social Network, Clinician-Patient Relationship)

Clarify the role of social barriers to help seeking, access to care, and problems engaging in previous treatment.

13. Has anything prevented you from getting the help you need?

PROBE AS NEEDED:

Probe details as needed (e.g., "What got in the way?").

For example, money, work or family commitments, stigma or discrimination, or lack of services that understand your language or background?

CULTURAL FACTORS AFFECTING CURRENT HELP SEEKING

Preferences

(Social Network, Caregivers, Religion and Spirituality, Older Adults, Coping and Help Seeking)

Clarify individual's current perceived needs and expectations of help, broadly defined.

Now let's talk some more about the help you need.

Probe if individual lists only one source of help (e.g., "What other kinds of help would be useful to you at this time?").

14. What kinds of help do you think would be most useful to you at this time for your [PROBLEM]?

Core Cultural Formulation Interview (CFI)

Focus on the views of the social network regarding help seeking.	15. Are there other kinds of help that your family, friends, or other people have suggested would be helpful for you now?

CLINICIAN-PATIENT RELATIONSHIP

(Clinician-Patient Relationship, Older Adults)	
Elicit possible concerns about the clinic or the clinician-patient relationship, including perceived racism, language barriers, or cultural differences that may undermine goodwill, communication, or care delivery.	Sometimes doctors and patients misunderstand each other because they come from different backgrounds or have different expectations.
Probe details as needed (e.g., "In what way?"). *Address possible barriers to care or concerns about the clinic and the clinician-patient relationship raised previously.*	16. Have you been concerned about this and is there anything that we can do to provide you with the care you need?

Cultural Formulation Interview (CFI)—Informant Version

The CFI Informant Version collects collateral information from an informant who is knowledgeable about the clinical problems and life circumstances of the identified individual. This version can be used to supplement information obtained from the core CFI or can be used instead of the core CFI when the individual is unable to provide information (e.g., children or adolescents, individuals with florid psychosis, individuals with cognitive impairment).

Cultural Formulation Interview (CFI)—Informant Version

GUIDE TO INTERVIEWER	INSTRUCTIONS TO THE INTERVIEWER ARE ***ITALICIZED.***

Cultural Formulation Interview (CFI)—Informant Version

The following questions aim to clarify key aspects of the presenting clinical problem from the informant's point of view. This includes the problem's meaning, potential sources of help, and expectations for services.

INTRODUCTION FOR THE INFORMANT:

I would like to understand the problems that bring your family member/friend here so that I can help you and him/her more effectively. I want to know about ***your*** experience and ideas. I will ask some questions about what is going on and how you and your family member/friend are dealing with it. There are no right or wrong answers.

RELATIONSHIP WITH THE PATIENT

Clarify the informant's relationship with the individual and/or the individual's family.

1. How would you describe your relationship to [INDIVIDUAL OR TO FAMILY]?

   PROBE IF NOT CLEAR:

   How often do you see [INDIVIDUAL]?

CULTURAL DEFINITION OF THE PROBLEM

Elicit the informant's view of core problems and key concerns.

Focus on the informant's way of understanding the individual's problem.

Use the term, expression, or brief description elicited in question 1 to identify the problem in subsequent questions (e.g., "her conflict with her son").

2. What brings your family member/friend here today?

   IF INFORMANT GIVES FEW DETAILS OR ONLY MENTIONS SYMPTOMS OR A MEDICAL DIAGNOSIS, PROBE:

   People often understand problems in their own way, which may be similar or different from how doctors describe the problem. How would ***you*** describe [INDIVIDUAL'S] problem?

Ask how informant frames the problem for members of the social network.

3. Sometimes people have different ways of describing the problem to family, friends, or others in their community. How would ***you*** describe [INDIVIDUAL'S] problem to them?

Focus on the aspects of the problem that matter most to the informant.

4. What troubles you most about [INDIVIDUAL'S] problem?

CULTURAL PERCEPTIONS OF CAUSE, CONTEXT, AND SUPPORT

CAUSES

This question indicates the meaning of the condition for the informant, which may be relevant for clinical care.

Note that informants may identify multiple causes depending on the facet of the problem they are considering.

5. Why do you think this is happening to [INDIVIDUAL]? What do you think are the causes of his/her [PROBLEM]?

   PROMPT FURTHER IF REQUIRED:

   Some people may explain the problem as the result of bad things that happen in their life, problems with others, a physical illness, a spiritual reason, or many other causes.

Focus on the views of members of the individual's social network. These may be diverse and vary from the informant's.

6. What do others in [INDIVIDUAL'S] family, his/her friends, or others in the community think is causing [INDIVIDUAL'S] [PROBLEM]?

STRESSORS AND SUPPORTS

Elicit information on the individual's life context, focusing on resources, social supports, and resilience. May also probe other supports (e.g., from co-workers, from participation in religion or spirituality).

7. Are there any kinds of supports that make his/her [PROBLEM] better, such as from family, friends, or others?

Focus on stressful aspects of the individual's environment. Can also probe, e.g., relationship problems, difficulties at work or school, or discrimination.

8. Are there any kinds of stresses that make his/her [PROBLEM] worse, such as difficulties with money, or family problems?

ROLE OF CULTURAL IDENTITY

Sometimes, aspects of people's background or identity can make the [PROBLEM] better or worse. By ***background*** or ***identity,*** I mean, for example, the communities you belong to, the languages you speak, where you or your family are from, your race or ethnic background, your gender or sexual orientation, and your faith or religion.

Ask the informant to reflect on the most salient elements of the individual's cultural identity. Use this information to tailor questions 10–11 as needed.

9. For you, what are the most important aspects of [INDIVIDUAL'S] background or identity?

Elicit aspects of identity that make the problem better or worse.

Probe as needed (e.g., clinical worsening as a result of discrimination due to migration status, race/ethnicity, or sexual orientation).

10. Are there any aspects of [INDIVIDUAL'S] background or identity that make a difference to his/her [PROBLEM]?

Probe as needed (e.g., migration-related problems; conflict across generations or due to gender roles).

11. Are there any aspects of [INDIVIDUAL'S] background or identity that are causing other concerns or difficulties for him/her?

CULTURAL FACTORS AFFECTING SELF-COPING AND PAST HELP SEEKING

SELF-COPING

Clarify individual's self-coping for the problem.

12. Sometimes people have various ways of dealing with problems like [PROBLEM]. What has [INDIVIDUAL] done on his/her own to cope with his/her [PROBLEM]?

PAST HELP SEEKING

Elicit various sources of help (e.g., medical care, mental health treatment, support groups, work-based counseling, folk healing, religious or spiritual counseling, other alternative healing).

Probe as needed (e.g., "What other sources of help has he/she used?").

Clarify the individual's experience and regard for previous help.

13. Often, people also look for help from many different sources, including different kinds of doctors, helpers, or healers. In the past, what kinds of treatment, help, advice, or healing has [INDIVIDUAL] sought for his/her [PROBLEM]?

PROBE IF DOES NOT DESCRIBE USEFULNESS OF HELP RECEIVED:

What types of help or treatment were most useful? Not useful?

BARRIERS

Clarify the role of social barriers to help seeking, access to care, and problems engaging in previous treatment.

Probe details as needed (e.g., "What got in the way?").

14. Has anything prevented [INDIVIDUAL] from getting the help he/she needs?

PROBE AS NEEDED:

For example, money, work or family commitments, stigma or discrimination, or lack of services that understand his/her language or background?

CULTURAL FACTORS AFFECTING CURRENT HELP SEEKING

PREFERENCES

Clarify individual's current perceived needs and expectations of help, broadly defined, from the point of view of the informant.

Now let's talk about the help [INDIVIDUAL] needs.

Probe if informant lists only one source of help (e.g., "What other kinds of help would be useful to [INDIVIDUAL] at this time?").

15. What kinds of help would be most useful to him/her at this time for his/her [PROBLEM]?

Focus on the views of the social network regarding help seeking.

16. Are there other kinds of help that [INDIVIDUAL'S] family, friends, or other people have suggested would be helpful for him/her now?

Cultural Formulation Interview (CFI)—Informant Version	
Elicit possible concerns about the clinic or the clinician-patient relationship, including perceived racism, language barriers, or cultural differences that may undermine goodwill, communication, or care delivery.	Sometimes doctors and patients misunderstand each other because they come from different backgrounds or have different expectations.
Probe details as needed (e.g., "In what way?"). *Address possible barriers to care or concerns about the clinic and the clinician-patient relationship raised previously.*	17. Have you been concerned about this, and is there anything that we can do to provide [INDIVIDUAL] with the care he/she needs?

Cultural Concepts of Distress

Relevance for Diagnostic Assessment

The term *cultural concepts of distress* refers to ways that individuals experience, understand, and communicate suffering, behavioral problems, or troubling thoughts and emotions. Three main types of cultural concepts of distress may be distinguished. *Cultural idioms of distress* are ways of expressing distress that may not involve specific symptoms or syndromes, but that provide collective, shared ways of experiencing and talking about personal or social concerns. For example, everyday talk about "nerves" or "depression" may refer to widely varying forms of suffering without mapping onto a discrete set of symptoms, syndrome, or disorder. *Cultural explanations* or perceived causes are labels, attributions, or features of an explanatory model that indicate culturally recognized meaning or etiology for symptoms, illness, or distress. *Cultural syndromes* are clusters of symptoms and attributions that tend to co-occur among individuals in specific cultural groups, communities, or contexts and that are recognized locally as coherent patterns of experience.

These three cultural concepts of distress—cultural idioms of distress, cultural explanations, and cultural syndromes—are more relevant to clinical practice than the older formulation *culture-bound syndrome*. Specifically, the term *culture-bound syndrome* ignores the fact that clinically important

cultural differences often involve explanations or experience of distress rather than culturally distinctive configurations of symptoms. Furthermore, the term *culture bound* overemphasizes the extent to which cultural concepts of distress are characterized by highly idiosyncratic experiences that are restricted to specific geographic regions. The current formulation acknowledges that all forms of distress are locally shaped, including the DSM disorders. From this perspective, many DSM diagnoses can be understood as operationalized prototypes that started out as cultural syndromes and became widely accepted as a result of their clinical and research utility. Across groups there remain culturally patterned differences in symptoms, ways of talking about distress, and locally perceived causes, which in turn are associated with coping strategies and patterns of help seeking.

Cultural concepts of distress arise from local "folk" or professional diagnostic systems for mental and emotional distress, and they may also reflect the influence of

biomedical concepts. Cultural concepts of distress have four key features in relation to the DSM-5 nosology:

- There is seldom a one-to-one correspondence of any cultural concept of distress with a DSM diagnostic entity; the correspondence is more likely to be one-to-many in either direction. Symptoms or behaviors that might be sorted by DSM-5 into several disorders may be included in a single cultural concept of distress, and diverse presentations that might be classified by DSM-5 as variants of a single disorder may be sorted into several distinct concepts by an indigenous diagnostic system.
- Cultural concepts of distress may apply to a wide range of symptom and functional severity, including presentations that do not meet DSM criteria for any mental disorder. For example, an individual with acute grief or a social predicament may use the same idiom of distress or display the same cultural syndrome as another individual with more severe psychopathology.
- In common usage, the same cultural term frequently denotes more than one type of cultural concept of distress. A familiar example may be the concept of "depression," which may be used to describe a syndrome (e.g., major depressive disorder), an idiom of distress (e.g., as in the common expression "I feel depressed"), or an explanation or perceived cause (e.g., "the baby

was born with emotional problems because his mother suffered from depression during her pregnancy").

- Like culture and DSM itself, cultural concepts of distress may change over time in response to both local and global influences.

Cultural concepts of distress are important to psychiatric diagnosis for several reasons:

- **To enhance identification of individuals' concerns and detection of psychopathology:** Referring to cultural concepts of distress in screening instruments or in reviews of systems may facilitate identification of individuals' concerns and enhance detection of psychopathology, as individuals may be more familiar with these cultural concepts of distress than with professional terminology.
- **To avoid misdiagnosis:** Cultural variation in symptoms and in explanatory models associated with these cultural concepts of distress may lead clinicians to misjudge the severity of a problem or assign the wrong diagnosis (e.g., socially warranted suspicion may be misunderstood as paranoia; unfamiliar symptom presentations may be misdiagnosed as psychosis).
- **To obtain useful clinical information:** Cultural variations in symptoms and attributions may be associated with particular features of risk, resilience, and outcome. Clinical exploration of cultural concepts of distress can elicit information on the role that specific contexts play in symptom development and course and in their response to coping strategies.
- **To improve clinical rapport and engagement:** "Speaking the language of the patient," both linguistically and in terms of his or her dominant cultural concepts of distress and metaphors, can result in greater communication and satisfaction, facilitate treatment negotiation, and lead to higher retention and adherence.
- **To improve therapeutic efficacy:** Culture influences the psychological mechanisms of a disorder, which need to be understood and addressed to improve clinical efficacy. For example, culturally specific catastrophic cognitions can contribute to symptom escalation into panic attacks.
- **To guide clinical research:** Locally perceived connections between cultural concepts of distress may help identify patterns of comorbidity and underlying biological substrates. Cultural concepts of distress, particularly cultural syndromes, may also point to previously unrecognized disorders or variants that could be included in future

nosological revisions (e.g., in a change from DSM-IV, the concept of possession was added to the DSM-5 criteria for dissociative identity disorder).

- **To clarify cultural epidemiology:** Cultural concepts of distress are not endorsed uniformly by everyone in a given cultural context. Distinguishing cultural idioms of distress, cultural explanations, and cultural syndromes provides an approach for studying the distribution of cultural features of illness across settings and regions, and over time. It also suggests questions about cultural determinants of risk, course, and outcome in clinical and community settings to enhance the evidence base of cultural research.

DSM-5 includes information on cultural concepts of distress in order to improve the accuracy of diagnosis and the comprehensiveness of clinical assessment. Clinical assessment of individuals presenting with these cultural concepts of distress should determine whether their presentation meets DSM-5 criteria for a specified disorder or instead is best classified as an *other specified* diagnosis. Once the disorder is diagnosed, the cultural terms and explanations should be included in case formulations; they may help clarify symptoms and etiological attributions that could otherwise be confusing. Individuals whose symptoms do not meet DSM criteria for a specific mental disorder may still expect and require treatment; this should be assessed on a case-by-case basis. In addition to the CFI and its informant and supplementary modules, DSM-5-TR contains the following information and tools that may be useful when integrating cultural information in clinical practice:

- **Data in updated DSM-5-TR text for specific disorders:** The text includes information on cultural variations in symptom expression; attributions for disorder causes or precipitants; factors associated with differential prevalence across demographic groups; cultural norms that may affect the threshold for pathology and the perceived severity of the condition; risk for misdiagnosis when evaluating individuals from socially oppressed ethnoracial or marginalized groups; associated cultural concepts of distress; and other material relevant to culturally informed diagnosis. It is important to emphasize that there is no one-to-one correspondence at the categorical level between DSM disorders and cultural concepts of distress. Differential diagnosis for individuals must therefore incorporate information on cultural variation with information elicited by the CFI.

- **Other Conditions That May Be a Focus of Clinical Attention:** Some of the clinical concerns identified by the CFI may correspond to one of the conditions or problems listed in the Section II chapter "Other Conditions That May Be a Focus of Clinical Attention" (e.g., acculturation problems, parent-child relational problems, religious or spiritual problems), along with the associated ICD-10-CM code.

Examples of Cultural Concepts of Distress

Clinicians need to familiarize themselves with individuals' cultural concepts of distress to understand individuals' concerns and facilitate accurate diagnostic assessment; use of the Cultural Formulation Interview may help in this regard. The following ten examples were selected to illustrate some of the ways in which cultural concepts of distress may affect the process of diagnosis. The principles illustrated with these examples can be applied to the myriad other cultural concepts of distress found in specific cultural contexts.

The same term may be used for multiple types of cultural concepts of distress and clinical presentations, depending on context. Potentially, cultural concepts of distress can occur on their own or coexist with any psychiatric disorder and influence clinical presentation, course, and outcome. For example, in U.S. Latinx communities, *ataque de nervios* can be comorbid with nearly all psychiatric disorders.

Each of the following examples of cultural concepts of distress includes a description of "Related conditions in DSM-5-TR" to highlight 1) the DSM-5 disorders that overlap

phenomenologically with the cultural concept of distress (e.g., panic disorder and *ataque de nervios*, due to their paroxysmic nature and symptom similarity) and 2) the DSM-5 disorders that are frequently attributed to the causal explanation or idiom (e.g., PTSD and *kufungisisa*).

Ataque de nervios

Ataque de nervios ("attack of nerves") is a syndrome found in Latinx cultural contexts, characterized by symptoms of intense emotional upset,

including acute anxiety, anger, or grief; screaming and shouting uncontrollably; attacks of crying; trembling; heat in the chest rising into the head; and becoming verbally and physically aggressive. Dissociative experiences (e.g., depersonalization, derealization, amnesia), seizure-like or fainting episodes, and suicidal behavior are prominent in some *ataques* but absent in others. A general feature of an *ataque de nervios* is a sense of being out of control. Attacks frequently occur as a direct result of a stressful event relating to the family, such as news of the death of a close relative, conflicts with a spouse or children, or witnessing an accident involving a family member. For a minority of individuals, no particular social or interpersonal event triggers their *ataques*; instead, their vulnerability to losing control comes from the accumulated experience of suffering.

No one-to-one relationship has been found between *ataque* and any specific psychiatric disorder, although several disorders, including panic disorder, other specified or unspecified dissociative disorder, and functional neurological symptom disorder (conversion disorder), have symptomatic overlap with *ataque*.

In community samples, *ataque* is reported among U.S. Latinx by 7%–15% of adults and 4%–9% of youth, depending on region and Latinx subgroup. It is associated with suicidal thoughts, disability, and outpatient psychiatric utilization, after adjustment for psychiatric diagnoses, traumatic exposure, and other covariates. However, some *ataques* represent normative expressions of acute distress (e.g., at a funeral) without clinical sequelae. The term *ataque de nervios* may also refer to an idiom of distress that includes any “fit”-like paroxysm of emotionality (e.g., hysterical laughing) and may be used to indicate an episode of loss of control in response to an intense stressor.

Related conditions in other cultural contexts. Indisposition in Haiti, blacking out in several West Indies and Caribbean countries, and falling out in the Southern United States. This use of the terms blacking out or falling out should not be confused with alcohol- or other substance-induced blackouts or amnesia.

Related conditions in DSM-5-TR. Panic attack, panic disorder, other specified or unspecified dissociative disorder, functional neurological symptom disorder, intermittent explosive disorder, other specified or unspecified

anxiety disorder, other specified or unspecified trauma- and stressor-related disorder.

Dhat syndrome

Dhat syndrome is a term that was coined in South Asia little more than half a century ago to account for common clinical presentations of young men who attributed their various symptoms to semen loss. Despite the name, it is not a discrete syndrome but rather a cultural explanation of distress for individuals who refer to diverse symptoms, such as anxiety, fatigue, weakness, weight loss, erectile dysfunction, other multiple somatic complaints, and depressed mood. The cardinal feature is anxiety and distress about the loss of *dhat* in the absence of any identifiable physiological dysfunction. *Dhat* was identified by individuals as a white discharge that was noted on defecation or urination. Ideas about this substance are related to the concept of *dhatu* (semen) described in the Hindu system of medicine, Ayurveda, as one of seven essential bodily fluids whose balance is necessary to maintain health.

Although *dhat syndrome* was formulated as a clinical category to help inform local clinical practice, related ideas about the harmful effects of semen loss have been shown to be widespread in the general population, suggesting a cultural disposition for explaining health problems and symptoms with reference to *dhat*-related concepts. Research in health care settings has yielded diverse estimates of the prevalence of *dhat syndrome* (e.g., 64% of men attending psychiatric clinics in India for sexual complaints; 30% of men attending general medical clinics in Pakistan). Although *dhat syndrome* is most commonly identified with young men from lower socioeconomic backgrounds, middle-age men may also be affected. Comparable concerns about white vaginal discharge (leukorrhea) have been associated with a variant of the concept for women. The term *dhat* may also be used as an idiom and causal explanation for sexually transmitted infections (e.g., gonorrhea, chlamydia), in the absence of psychological distress.

Related conditions in other cultural contexts. *Koro* in Southeast Asia, particularly Singapore, and *shen-k'uei* ("kidney deficiency") in China.

Related conditions in DSM-5-TR. Major depressive disorder, persistent depressive disorder, generalized anxiety disorder, somatic symptom disorder, illness anxiety disorder, erectile disorder, early (premature) ejaculation, other specified or unspecified sexual dysfunction, educational problems.

Hikikomori

Hikikomori (a Japanese term composed of *hiku* [to pull back] and *moru* [to seclude oneself]) is a syndrome of protracted and severe social withdrawal observed in Japan that may result in complete cessation of in-person interactions with others. The typical picture in *hikikomori* is an adolescent or young adult male who does not leave his room within his parents' home and has no in-person social interactions. This behavior may initially be ego-syntonic but usually leads to distress over time; it is often associated with high intensity of Internet use and virtual social exchanges. Other features include no interest or willingness to attend school or work. The 2010 guideline of the Japan Ministry of Health, Labor, and Welfare requires 6 months of social withdrawal for a diagnosis of *hikikomori*. The extreme social withdrawal seen in *hikikomori* may occur in the context of an established DSM-5 disorder ("secondary") or manifest independently ("primary").

Related conditions in other cultural contexts. Protracted social withdrawal among adolescents and young adults has been reported in many settings, including Australia, Bangladesh, Brazil, China, France, India, Iran, Italy, Oman, South Korea, Spain, Taiwan, Thailand, and the United States. Individuals with *hikikomori*-type behaviors in Japan, India, South Korea, and the United States tend to display high levels of loneliness, limited social networks, and moderate functional impairment.

Related conditions in DSM-5-TR. Social anxiety disorder, major depressive disorder, generalized anxiety disorder, posttraumatic stress disorder, autism spectrum disorder, schizoid personality disorder, avoidant personality disorder, schizophrenia or other psychotic disorder. The condition may also

be associated with Internet gaming disorder and, in adolescents, with school refusal.

Khyâl cap

"*Khyâl* attacks" (*khyâl* cap), or "wind attacks," is a syndrome found in Cambodian cultural contexts. Common symptoms include those of panic attacks, such as dizziness, palpitations, shortness of breath, and cold extremities, as well as other symptoms of anxiety and autonomic arousal (e.g., tinnitus and neck soreness). *Khyâl* attacks include catastrophic cognitions centered on the concern that *khyâl* (a windlike substance) may rise in the body—along with blood—and cause a range of serious effects (e.g., compressing the lungs to cause

shortness of breath and asphyxia; entering the cranium to cause tinnitus, dizziness, blurry vision, and a fatal syncope). *Khyâl* attacks may occur without warning but are frequently brought about by triggers such as worrisome thoughts, standing up (i.e., orthostasis), specific odors with negative associations, and agoraphobic-type cues like going to crowded spaces or riding in a car. *Khyâl* attacks usually meet panic attack criteria and may shape the experience of other anxiety and trauma- and stressor-related disorders. *Khyâl* attacks may be associated with considerable disability.

Related conditions in other cultural contexts. *Pen lom* in Laos, *srog rlung gi nad* in Tibet, *vata* in Sri Lanka, and *hwa byung* in Korea.

Related conditions in DSM-5-TR. Panic attack, panic disorder, generalized anxiety disorder, agoraphobia, posttraumatic stress disorder, illness anxiety disorder.

Kufungisisa

Kufungisisa ("thinking too much" in Shona) is an idiom of distress and a cultural explanation among the Shona of Zimbabwe. As an explanation, it is considered to be causative of anxiety, depression, and somatic problems (e.g., "My heart is painful because I think too much"). As an idiom of

psychosocial distress, it is indicative of interpersonal and social difficulties (e.g., marital problems, having no money to take care of children, unemployment). *Kufungisisa* involves ruminating on upsetting thoughts, particularly worries, including concerns about chronic physical illness, such as HIV-related disorders.

Kufungisisa is associated with a range of psychopathology, including anxiety symptoms, excessive worry, panic attacks, depressive symptoms, irritability, and posttraumatic stress disorder. In a study of a random community sample, two-thirds of the cases identified by a general psychopathology measure included this complaint.

Related conditions in other cultural contexts. "Thinking too much" is a common idiom of distress and cultural explanation across many countries and ethnic groups; despite some commonalities across global regions, "thinking too much" shows important heterogeneity across and within cultural contexts. It has been described in Africa, Asia, the Caribbean and Latin America, the Middle East, and among indigenous groups. "Thinking too much" may also be a key component of cultural syndromes such as "brain fag" in Nigeria. In the case of "brain fag," "thinking too much" is primarily attributed to excessive study, which is considered to damage the brain in particular, with symptoms including feelings of heat or crawling sensations in the head.

Cross-culturally, "thinking too much" typically references ruminative, intrusive, and/or anxious thoughts—sometimes focused on a singular concern or past trauma and other times based on numerous current worries. In some contexts, it is thought to lead to more severe disorder-like psychosis, suicidal thoughts, or even death.

Related conditions in DSM-5-TR. Major depressive disorder, persistent depressive disorder, generalized anxiety disorder, posttraumatic stress disorder, obsessive-compulsive disorder, prolonged grief disorder.

Maladi dyab

Maladi dyab or *maladi satan* (literally "devil/Satan illness," also referred to as "sent sickness") is a cultural explanation in Haitian communities for diverse medical and psychiatric disorders, or other negative experiences and problems in functioning. In this explanatory model, interpersonal envy and

malice cause people to harm their enemies by having sorcerers send illnesses such as psychosis, depression, social or academic failure, and inability to perform activities of daily living. These sicknesses have various names (e.g., *ekspedisyon*, *mòvè zespri*, *kout poud*) based on how they are "sent". This etiological explanation assumes that illness may be caused by others' envy and hatred, provoked by the

victim's economic success as evidenced by a new job or expensive purchase. One person's gain is assumed to produce another person's loss, so visible success makes an individual vulnerable to attack. Assigning the label of "sent sickness" depends more on mode of onset, social status, and form of treatment that proves successful than on presenting symptoms. A wide range of psychiatric disorders can be attributed to this cultural explanation. The acute onset of new symptoms or an abrupt behavioral change raises suspicions of a spiritual attack. An individual who is attractive, intelligent, or wealthy is perceived as especially vulnerable, and even young healthy children are at risk.

Related conditions in other cultural contexts. Concerns about illness (typically, physical illness) caused by envy or social conflict are common across cultural contexts and often expressed in the form of "evil eye" (e.g., in Spanish, *mal de ojo*; in Italian, *mal'occhiu*).

Related conditions in DSM-5-TR. Subsyndromal affliction (e.g., problems related to the social environment, educational problems), in addition to a wide range of psychiatric disorders; the cultural explanation of supernatural forces may lead to misdiagnosis of delusional disorder, persecutory type; or schizophrenia.

Nervios

Nervios ("nerves") is a common cultural idiom of distress and causal explanation in Latinx cultural contexts in the United States and Latin America. *Nervios* refers to a general state of vulnerability to stressful life experiences and to difficult life circumstances. The term *nervios* includes a wide range of symptoms of emotional distress, somatic disturbance, and

inability to function. The most common symptoms attributed to *nervios* include headaches and “brain aches” (occipital neck tension), irritability, gastrointestinal disturbances, sleep difficulties, nervousness, easy tearfulness, inability to concentrate, trembling, tingling sensations, and *mareos* (dizziness with occasional vertigo-like exacerbations). *Nervios* is a broad cultural idiom of distress that spans the range of severity from cases with no mental disorder to presentations resembling adjustment, anxiety, depressive, dissociative, somatic symptom, or psychotic disorders. The term can also refer to a cultural explanation for multiple forms of psychological distress, especially those involving weakness, enervation, and anxiety. *Nervios* may indicate a range of conditions, which show regional variation, related to the nervous system (literally, the anatomical nerves). In Puerto Rican communities, for example, *nervios* includes conditions such as “being nervous since childhood,” which appears to be more of a trait and may precede social anxiety disorder, and “being ill with nerves,” which is more related than other forms of *nervios* to psychiatric problems, especially dissociation and depression.

Related conditions in other cultural contexts. *Nevra* among Greeks in North America, *nierbi* among Sicilians in North America, and “nerves” among Whites in Appalachia and Newfoundland. “Tension” is a related idiom and causal explanation among South Asian popul ations.

Related conditions in DSM-5-TR. Major depressive disorder, persistent depressive disorder, generalized anxiety disorder, social anxiety disorder, other specified or unspecified dissociative disorder, somatic symptom disorder, schizophrenia.

Shenjing shuairuo

Shenjing shuairuo (“weakness of the nervous system” in Mandarin Chinese) is a cultural syndrome that integrates conceptual categories of Traditional Chinese Medicine with the Western construct of neurasthenia. In the second, revised edition of the *Chinese Classification of Mental Disorders* (CCMD-2-R), *shenjing shuairuo* was defined as a syndrome composed of three out of five symptom clusters: weakness (e.g., mental fatigue), emotions (e.g., feeling vexed), excitement (e.g., increased recollections), nervous pain (e.g., headache),

and sleep (e.g., insomnia). *Fan nao* (feeling vexed) is a form of irritability mixed with worry and distress over conflicting thoughts and unfulfilled desires. The third edition of the CCMD retained *shenjing shuairuo* as a somatoform diagnosis of exclusion. However, China adopted the ICD-10 as its official classification system in 2011, displacing the CCMD; although ICD-10 included neurasthenia as a diagnostic category, ICD-11 does not. The use of *shenjing shuairuo* has decreased substantially in recent years and appears to have been replaced by idioms of depression and anxiety, at least in urban areas; among mental health clinicians, *shenjing shuairuo* may largely be invoked in interactions with traditional patients to facilitate communication and limit the stigma associated with psychiatric diagnoses.

Salient precipitants of *shenjing shuairuo* include work or family-related stressors, loss of face (*mianzi*, *lianzi*), and an acute sense of failure (e.g., in academic performance). *Shenjing shuairuo* is related to traditional concepts of weakness (*xu*) and health imbalances related to deficiencies of a vital essence (e.g., the depletion of *qi* [vital energy] following overstraining or stagnation of *qi* due to excessive worry). In the traditional interpretation, *shenjing shuairuo* results when bodily channels (*jing*) conveying vital forces (*shen*) become dysregulated as a result of various social and interpersonal stressors, such as the inability to change a chronically frustrating and distressing situation. Various psychiatric disorders are associated with *shenjing shuairuo*, notably mood, anxiety, and somatic symptom disorders. In medical clinics in China, however, up to 45% of patients with *shenjing shuairuo* do not have symptoms that meet criteria for any DSM-IV disorder.

Related conditions in other cultural contexts. Neurasthenia-spectrum idioms and syndromes are present in many cultural contexts, including India (*ashaktapanna*), Mongolia (*yadargaa*), and Japan (*shinkei-suijaku*), among other settings. Other conditions, such as brain fag syndrome, burnout syndrome, and chronic fatigue syndrome, are also closely related.

Related conditions in DSM-5-TR.

Major depressive disorder, persistent depressive disorder, generalized anxiety disorder, somatic symptom disorder, social anxiety disorder, specific phobia, posttraumatic stress disorder.

Susto

Susto ("fright") is a cultural explanation for distress and misfortune prevalent in some Latinx cultural contexts in North, Central, and South America. It is not recognized as an illness category among Latinx from the Caribbean. *Susto* is an illness attributed to a frightening event that causes the soul to leave the body and results in unhappiness and sickness, as well as difficulties functioning in key social roles. Symptoms may appear any time from days to years after the fright is experienced. In extreme cases, *susto* may result in death. There are no specific defining symptoms for *susto*; however, symptoms that are often reported by individuals with *susto* include appetite disturbances; inadequate or excessive sleep; troubled sleep or dreams; feelings of sadness, low self-worth, or dirtiness; interpersonal sensitivity; and lack of motivation to do anything. Somatic symptoms accompanying *susto* may include muscle aches and pains, cold in the extremities, pallor, headache, stomachache, and diarrhea. Precipitating events are diverse and include natural phenomena, animals, interpersonal situations, and supernatural agents, among others.

Three syndromic types of *susto* (referred to as *cibih* in the Zapotec language) have been identified, each having different relationships with psychiatric diagnoses. An interpersonal *susto* characterized by feelings of loss, abandonment, and not being loved by family, with accompanying symptoms of sadness, poor self-image, and suicidal thoughts, seems to be closely related to major depressive disorder. When *susto* results from a traumatic event that plays a major role in shaping symptoms and in emotional processing of the experience, the diagnosis of posttraumatic stress disorder appears more appropriate. *Susto* characterized by various recurrent somatic symptoms—for which the individual seeks health care from several practitioners—is thought to resemble a somatic symptom disorder.

Related conditions in other cultural contexts. Similar etiological concepts and symptom configurations are found globally. In the Andean region, *susto* is

referred to as *espanto*. Soul loss conditions in South Asia and Southeast Asia also share features with *susto*. In soul loss, individuals experiencing a fright are thought to temporarily lose their soul, a piece of their soul, or one of many souls. This makes the individual vulnerable to other physical and psychological forms of distress.

Related conditions in DSM-5-TR. Major depressive disorder, posttraumatic stress disorder, other specified or unspecified trauma and stressor-related disorder, somatic symptom disorder.

Taijin kyofusho

Taijin kyofusho ("interpersonal fear disorder" in Japanese) is a syndrome found in Japanese cultural contexts characterized by anxiety about and avoidance of interpersonal situations due to the thought, feeling, or conviction that the individual's appearance and actions in social interactions are inadequate or offensive to others. *Taijin kyofusho* includes two culture-related forms: a "sensitive type," with extreme social sensitivity and anxiety about interpersonal interactions, and an "offensive type," in which the major concern is offending others. Variants include major concerns about facial blushing (*sekimen-kyofu*), having an offensive body odor (*jiko-shu-kyofu*), inappropriate gaze (too much or too little eye contact, *jiko-shisen-kyofu*), and stiff or awkward facial expression or bodily movements (e.g., stiffening, trembling) or body deformity (*shubo-kyofu*).

Taijin kyofusho is a broader construct than social anxiety disorder in DSM-5. *Taijin kyofusho* also includes syndromes with features of body dysmorphic disorder, olfactory reference syndrome, and delusional disorder; delusional disorder should be considered when concerns have a delusional quality, responding poorly to simple reassurance or counterexample.

Related conditions in other cultural contexts. The distinctive symptoms of *taijin kyofusho* occur in specific cultural contexts and, to some extent, with more severe social anxiety cross-culturally. Similar syndromes are found in Korea (*taein kong po*) and other societies that place a strong emphasis on the self-conscious maintenance of appropriate social behavior in hierarchical interpersonal relationships. An interdependent self-construal, which emphasizes the relatedness of self to a collective and the

identification of self in terms of social roles and relationships, may be a risk factor for *taijin kyofusho* symptoms across diverse cultures. The concern with offending others through inappropriate social behavior, characteristic of offensive-type *taijin kyofusho*, has also been described in several societies, including the United States, Australia, Indonesia, and New Zealand.

Related conditions in DSM-5-TR. Social anxiety disorder, body dysmorphic disorder, delusional disorder, obsessive-compulsive disorder, olfactory reference syndrome (a type of other specified obsessive-compulsive and related disorder). Olfactory reference syndrome is related specifically to the *jikoshu-kyofu* variant of *taijin kyofusho*; this presentation is seen in various cultures outside Japan.

Alternative DSM-5 Model for Personality Disorders

Provided as an alternative to the extant personality disorders classification in Section II, this hybrid dimensional-categorical model in Section III defines personality disorder in terms of impairments in personality functioning and pathological personality traits. The inclusion of both models of personality disorder diagnosis in DSM-5 reflects the decision of the APA Board of Trustees to preserve continuity with current clinical practice, while also introducing an alternative approach that aims to address numerous shortcomings of the approach in Section II to personality disorders. For example, in the approach in Section II, symptoms meeting criteria for a specific personality disorder frequently also meet criteria for other personality disorders, and other specified or unspecified personality disorder is often the correct (but mostly uninformative) diagnosis, in the sense that individuals do not tend to present with patterns of symptoms that correspond with one and only one personality disorder.

In the following alternative DSM-5 model, personality disorders are characterized by impairments in personality *functioning* and pathological personality *traits*. The specific personality disorder diagnoses that may be derived from this model include antisocial, avoidant, borderline, narcissistic, obsessive-compulsive, and schizotypal personality disorders. This approach also includes a diagnosis of personality disorder—trait specified (PD-TS) that can be made when a personality disorder is considered present but the criteria for a specific disorder are not met.

General Criteria for Personality Disorder

General Criteria for Personality Disorder

The essential features of a personality disorder are

A. Moderate or greater impairment in personality (self/interpersonal) functioning.
B. One or more pathological personality traits.
C. The impairments in personality functioning and the individual's personality trait expression are relatively inflexible and pervasive across a broad range of personal and social situations.
D. The impairments in personality functioning and the individual's personality trait expression are relatively stable across time, with onsets that can be traced back to at least adolescence or early adulthood.
E. The impairments in personality functioning and the individual's personality trait expression are not better explained by another mental disorder.
F. The impairments in personality functioning and the individual's personality trait expression are not solely attributable to the physiological effects of a substance or another medical condition (e.g., severe head trauma).
G. The impairments in personality functioning and the individual's personality trait expression are not better understood as normal for an individual's developmental stage or sociocultural environment.

A diagnosis of a personality disorder requires two determinations: 1) an assessment of the level of impairment in personality functioning, which is needed for Criterion A, and 2) an evaluation of pathological personality traits, which is required for Criterion B. The impairments in personality functioning and personality trait expression are relatively inflexible and

pervasive across a broad range of personal and social situations (Criterion C); relatively stable across time, with onsets that can be traced back to at least adolescence or early adulthood (Criterion D); not better explained by another mental disorder (Criterion E); not attributable to the physiological effects of a substance or another medical condition (Criterion F); and not better understood as normal for an individual's developmental stage or sociocultural environment (Criterion G). All Section III personality disorders described by criteria sets, as well as PD-TS, meet these general criteria, by definition.

Criterion A: Level of Personality Functioning

Disturbances in **self** and **interpersonal** functioning constitute the core of personality psychopathology, and in this alternative diagnostic model they are evaluated on a continuum. Self functioning involves identity and self-direction; interpersonal functioning involves empathy and intimacy (see Table 1). The Level of Personality Functioning Scale (LPFS; see Table 2, pp. 895–898) uses each of these elements to differentiate five levels of impairment, ranging from little or no impairment (i.e., healthy, adaptive functioning; Level 0) to some (Level 1), moderate (Level 2), severe (Level 3), and extreme (Level 4) impairment.

Impairment in personality functioning predicts the presence of a personality disorder, and the severity of impairment predicts whether an individual has more than one personality disorder or one of the more typically severe personality disorders. A moderate level of impairment in personality functioning is required for the diagnosis of a personality disorder; this threshold is based on empirical evidence that the moderate level of impairment maximizes the ability of clinicians to accurately and efficiently identify personality disorder pathology.

Criterion B: Pathological Personality Traits

Pathological personality traits are organized into five broad domains: Negative Affectivity, Detachment, Antagonism, Disinhibition, and Psychoticism. Within the five broad **trait domains** are 25 specific **trait facets** that were developed initially from a review of existing trait models and subsequently through iterative research with samples of persons who

sought mental health services. The full trait taxonomy is presented in Table 3 (see pp. 899–901). The B criteria for the specific personality disorders comprise subsets of the 25 trait facets, based on meta-analytic reviews and empirical data on the relationships of the traits to DSM-IV personality disorder diagnoses.

Criteria C and D: Pervasiveness and Stability

Impairments in personality functioning and pathological personality traits are *relatively* pervasive across a range of personal and social contexts, as personality is defined as a pattern of perceiving, relating to, and thinking about the environment and oneself. The term *relatively* reflects the fact that all except the most extremely pathological personalities show some degree of adaptability. The pattern in personality disorders is maladaptive and relatively inflexible, which leads to disabilities in social, occupational, or other important pursuits, as individuals are unable to modify their thinking or behavior, even in the face of evidence that their approach is not working. The impairments in functioning and personality traits are also *relatively* stable. Personality traits—the dispositions to behave or feel in certain ways—are more stable than the symptomatic expressions of these dispositions, but personality traits can also change. Impairments in personality functioning are more stable than symptoms.

TABLE 1 Elements of personality functioning

Self:

1. ***Identity:*** Experience of oneself as unique, with clear boundaries between self and others; stability of self-esteem and accuracy of self-appraisal; capacity for, and ability to regulate, a range of emotional experience.
2. ***Self-direction:*** Pursuit of coherent and meaningful short-term and life goals; utilization of constructive and prosocial internal standards of behavior; ability to self-reflect productively.

Interpersonal:

1. ***Empathy:*** Comprehension and appreciation of others' experiences and motivations; tolerance of differing perspectives; understanding of the effects of one's own behavior on others.
2. ***Intimacy:*** Depth and duration of connection with others; desire and capacity for closeness; mutuality of regard reflected in interpersonal behavior.

Criteria E, F, and G: Alternative Explanations for Personality Pathology (Differential Diagnosis)

On some occasions, what appears to be a personality disorder may be better explained by another mental disorder, the physiological effects of a substance or another medical condition, or a normal developmental stage (e.g., adolescence, late life) or the individual's sociocultural environment. When another mental disorder is present, the diagnosis of a personality disorder is not made if the manifestations of the personality disorder clearly are an expression of the other mental disorder (e.g., if features of schizotypal personality disorder are present only in the context of schizophrenia). On the other hand, personality disorders can be accurately diagnosed in the presence of another mental disorder, such as major depressive disorder, and patients with other mental disorders should be assessed for comorbid personality disorders because personality disorders often impact the course of other mental disorders. Therefore, it is always appropriate to assess personality functioning and pathological personality traits to provide a context for other psychopathology.

Specific Personality Disorders

Section III includes diagnostic criteria for antisocial, avoidant, borderline, narcissistic, obsessive-compulsive, and schizotypal personality disorders. Each personality disorder is defined by typical impairments in personality functioning (Criterion A) and characteristic pathological personality traits (Criterion B):

- Typical features of **antisocial personality disorder** are a failure to conform to lawful and ethical behavior, and an egocentric, callous lack of concern for others, accompanied by deceitfulness, irresponsibility, manipulativeness, and/or risk taking.
- Typical features of **avoidant personality disorder** are avoidance of social situations and inhibition in interpersonal relationships related to feelings of ineptitude and inadequacy, anxious preoccupation with negative evaluation and rejection, and fears of ridicule or embarrassment.
- Typical features of **borderline personality disorder** are instability of self-image, personal goals, interpersonal relationships, and affects, accompanied by impulsivity, risk taking, and/or hostility.

- Typical features of **narcissistic personality disorder** are variable and vulnerable self-esteem, with attempts at regulation through attention and approval seeking, and either overt or covert grandiosity.

- Typical features of **obsessive-compulsive personality disorder** are difficulties in establishing and sustaining close relationships, associated with rigid perfectionism, inflexibility, and restricted emotional expression.
- Typical features of **schizotypal personality disorder** are impairments in the capacity for social and close relationships, and eccentricities in cognition, perception, and behavior that are associated with distorted self-image and incoherent personal goals and accompanied by suspiciousness and restricted emotional expression.

The A and B criteria for the six specific personality disorders and for PD-TS follow. All personality disorders also meet criteria C through G of the General Criteria for Personality Disorder.

Antisocial Personality Disorder

Typical features of antisocial personality disorder are a failure to conform to lawful and ethical behavior, and an egocentric, callous lack of concern for others, accompanied by deceitfulness, irresponsibility, manipulativeness, and/or risk taking. Characteristic difficulties are apparent in identity, self-direction, empathy, and/or intimacy, as described below, along with specific maladaptive traits in the domains of Antagonism and Disinhibition.

Proposed Diagnostic Criteria

A. Moderate or greater impairment in personality functioning, manifested by characteristic difficulties in two or more of the following four areas:

   1. ***Identity:*** Egocentrism; self-esteem derived from personal gain, power, or pleasure.

2. ***Self-direction:*** Goal setting based on personal gratification; absence of prosocial internal standards, associated with failure to conform to lawful or culturally normative ethical behavior.
3. ***Empathy:*** Lack of concern for feelings, needs, or suffering of others; lack of remorse after hurting or mistreating another.
4. ***Intimacy:*** Incapacity for mutually intimate relationships, as exploitation is a primary means of relating to others, including by deceit and coercion; use of dominance or intimidation to control others.

B. Six or more of the following seven pathological personality traits:

1. ***Manipulativeness*** (an aspect of **Antagonism**): Frequent use of subterfuge to influence or control others; use of seduction, charm, glibness, or ingratiation to achieve one's ends.
2. ***Callousness*** (an aspect of **Antagonism**): Lack of concern for feelings or problems of others; lack of guilt or remorse about the negative or harmful effects of one's actions on others; aggression; sadism.
3. ***Deceitfulness*** (an aspect of **Antagonism**): Dishonesty and fraudulence; misrepresentation of self; embellishment or fabrication when relating events.
4. ***Hostility*** (an aspect of **Antagonism**): Persistent or frequent angry feelings; anger or irritability in response to minor slights and insults; mean, nasty, or vengeful behavior.
5. ***Risk taking*** (an aspect of **Disinhibition**): Engagement in dangerous, risky, and potentially self-damaging activities, unnecessarily and without regard for consequences; boredom proneness and thoughtless initiation of activities to counter boredom; lack of concern for one's limitations and denial of the reality of personal danger.

6. ***Impulsivity*** (an aspect of **Disinhibition**): Acting on the spur of the moment in response to immediate stimuli; acting on a momentary basis without a plan or consideration of outcomes; difficulty establishing and following plans.

7. ***Irresponsibility*** (an aspect of **Disinhibition**): Disregard for—and failure to honor—financial and other obligations or commitments; lack of respect for—and lack of follow-through on—agreements and promises.

Note. The individual is at least 18 years of age.

Specify if:

With psychopathic features

Specifiers. A distinct variant often termed *psychopathy* (or "primary" psychopathy) is marked by a lack of anxiety or fear and by a bold interpersonal style that may mask maladaptive behaviors (e.g., fraudulence). This psychopathic variant is characterized by low levels of anxiousness (Negative Affectivity domain) and withdrawal (Detachment domain) and high levels of attention seeking (Antagonism domain). High attention seeking and low withdrawal capture the social potency (assertive/dominant) component of psychopathy, whereas low anxiousness captures the stress immunity (emotional stability/resilience) component.

In addition to psychopathic features, trait and personality functioning specifiers may be used to record other personality features that may be present in antisocial personality disorder but are not required for the diagnosis. For example, traits of Negative Affectivity (e.g., anxiousness) are not diagnostic criteria for antisocial personality disorder (see Criterion B) but can be specified when appropriate. Furthermore, although moderate or greater impairment in personality functioning is required for the diagnosis of antisocial personality disorder (Criterion A), the level of personality functioning can also be specified.

Avoidant Personality Disorder

Typical features of avoidant personality disorder are avoidance of social situations and inhibition in interpersonal relationships related to feelings of ineptitude and inadequacy, anxious preoccupation with negative evaluation and rejection, and fears of ridicule or embarrassment. Characteristic difficulties are apparent in identity, self-direction, empathy, and/or intimacy, as described below, along with specific maladaptive traits in the domains of Negative Affectivity and Detachment.

Proposed Diagnostic Criteria

A. Moderate or greater impairment in personality functioning, manifested by characteristic difficulties in two or more of the following four areas:

   1. ***Identity:*** Low self-esteem associated with self-appraisal as socially inept, personally unappealing, or inferior; excessive feelings of shame.
   2. ***Self-direction:*** Unrealistic standards for behavior associated with reluctance to pursue goals, take personal risks, or engage in new activities involving interpersonal contact.
   3. ***Empathy:*** Preoccupation with, and sensitivity to, criticism or rejection, associated with distorted inference of others' perspectives as negative.
   4. ***Intimacy:*** Reluctance to get involved with people unless being certain of being liked; diminished mutuality within intimate relationships because of fear of being shamed or ridiculed.

B. Three or more of the following four pathological personality traits, one of which must be (1) Anxiousness:

   1. ***Anxiousness*** (an aspect of **Negative Affectivity**): Intense feelings of nervousness, tenseness, or panic, often in reaction to social situations; worry about the

negative effects of past unpleasant experiences and future negative possibilities; feeling fearful, apprehensive, or threatened by uncertainty; fears of embarrassment.

2. ***Withdrawal*** (an aspect of **Detachment**): Reticence in social situations; avoidance of social contacts and activity; lack of initiation of social contact.

3. ***Anhedonia*** (an aspect of **Detachment**): Lack of enjoyment from, engagement in, or energy for life's experiences; deficits in the capacity to feel pleasure or take interest in things.
4. ***Intimacy avoidance*** (an aspect of **Detachment**): Avoidance of close or romantic relationships, interpersonal attachments, and intimate sexual relationships.

Specifiers. Considerable heterogeneity in the form of additional personality traits is found among individuals diagnosed with avoidant personality disorder. Trait and level of personality functioning specifiers can be used to record additional personality features that may be present in avoidant personality disorder. For example, other Negative Affectivity traits (e.g., depressivity, separation insecurity, submissiveness, suspiciousness, hostility) are not diagnostic criteria for avoidant personality disorder (see Criterion B) but can be specified when appropriate. Furthermore, although moderate or greater impairment in personality functioning is required for the diagnosis of avoidant personality disorder (Criterion A), the level of personality functioning also can be specified.

Borderline Personality Disorder

Typical features of borderline personality disorder are instability of self-image, personal goals, interpersonal relationships, and affects, accompanied by impulsivity, risk taking, and/or hostility. Characteristic difficulties are apparent in identity, self-direction, empathy, and/or intimacy, as described

below, along with specific maladaptive traits in the domain of Negative Affectivity, and also Antagonism and/or Disinhibition.

Proposed Diagnostic Criteria

A. Moderate or greater impairment in personality functioning, manifested by characteristic difficulties in two or more of the following four areas:

   1. ***Identity:*** Markedly impoverished, poorly developed, or unstable self-image, often associated with excessive self-criticism; chronic feelings of emptiness; dissociative states under stress.
   2. ***Self-direction:*** Instability in goals, aspirations, values, or career plans.
   3. ***Empathy:*** Compromised ability to recognize the feelings and needs of others associated with interpersonal hypersensitivity (i.e., prone to feel slighted or insulted); perceptions of others selectively biased toward negative attributes or vulnerabilities.
   4. ***Intimacy:*** Intense, unstable, and conflicted close relationships, marked by mistrust, neediness, and anxious preoccupation with real or imagined abandonment; close relationships often viewed in extremes of idealization and devaluation and alternating between overinvolvement and withdrawal.

B. Four or more of the following seven pathological personality traits, at least one of which must be (5) Impulsivity, (6) Risk taking, or (7) Hostility:

   1. ***Emotional lability*** (an aspect of **Negative Affectivity**): Unstable emotional experiences and frequent mood changes; emotions that are easily aroused, intense, and/or out of proportion to events and circumstances.

2. ***Anxiousness*** (an aspect of **Negative Affectivity**): Intense feelings of nervousness, tenseness, or panic, often in reaction to interpersonal stresses; worry about the negative effects of past unpleasant experiences and future negative possibilities; feeling fearful, apprehensive, or threatened by uncertainty; fears of falling apart or losing control.
3. ***Separation insecurity*** (an aspect of **Negative Affectivity**): Fears of rejection by—and/or separation from—significant others, associated with fears of excessive dependency and complete loss of autonomy.

4. ***Depressivity*** (an aspect of **Negative Affectivity**): Frequent feelings of being down, miserable, and/or hopeless; difficulty recovering from such moods; pessimism about the future; pervasive shame; feelings of inferior self-worth; thoughts of suicide and suicidal behavior.
5. ***Impulsivity*** (an aspect of **Disinhibition**): Acting on the spur of the moment in response to immediate stimuli; acting on a momentary basis without a plan or consideration of outcomes; difficulty establishing or following plans; a sense of urgency and self-harming behavior under emotional distress.
6. ***Risk taking*** (an aspect of **Disinhibition**): Engagement in dangerous, risky, and potentially self-damaging activities, unnecessarily and without regard to consequences; lack of concern for one's limitations and denial of the reality of personal danger.
7. ***Hostility*** (an aspect of **Antagonism**): Persistent or frequent angry feelings; anger or irritability in response to minor slights and insults.

Specifiers. Trait and level of personality functioning specifiers may be used to record additional personality features that may be present in borderline personality disorder but are not required for the diagnosis. For example, traits of Psychoticism (e.g., cognitive and perceptual dysregulation) are not diagnostic criteria for borderline personality disorder (see Criterion B) but can be specified when appropriate. Furthermore, although moderate or greater impairment in personality functioning is required for the diagnosis of borderline personality disorder (Criterion A), the level of personality functioning can also be specified.

Narcissistic Personality Disorder

Typical features of narcissistic personality disorder are variable and vulnerable self-esteem, with attempts at regulation through attention and approval seeking, and either overt or covert grandiosity. Characteristic difficulties are apparent in identity, self-direction, empathy, and/or intimacy, as described below, along with specific maladaptive traits in the domain of Antagonism.

Proposed Diagnostic Criteria

A. Moderate or greater impairment in personality functioning, manifested by characteristic difficulties in two or more of the following four areas:

   1. ***Identity:*** Excessive reference to others for self-definition and self-esteem regulation; exaggerated self-appraisal inflated or deflated, or vacillating between extremes; emotional regulation mirrors fluctuations in self-esteem.
   2. ***Self-direction:*** Goal setting based on gaining approval from others; personal standards unreasonably high in order to see oneself as exceptional, or too low based on a sense of entitlement; often unaware of own motivations.
   3. ***Empathy:*** Impaired ability to recognize or identify with the feelings and needs of others; excessively attuned to

reactions of others, but only if perceived as relevant to self; over- or underestimation of own effect on others.

4. ***Intimacy:*** Relationships largely superficial and exist to serve self-esteem regulation; mutuality constrained by little genuine interest in others' experiences and predominance of a need for personal gain.

B. Both of the following pathological personality traits:

1. ***Grandiosity*** (an aspect of **Antagonism**): Feelings of entitlement, either overt or covert; self-centeredness; firmly holding to the belief that one is better than others; condescension toward others.

2. ***Attention seeking*** (an aspect of **Antagonism**): Excessive attempts to attract and be the focus of the attention of others; admiration seeking.

Specifiers. Trait and personality functioning specifiers may be used to record additional personality features that may be present in narcissistic personality disorder but are not required for the diagnosis. For example, other traits of Antagonism (e.g., manipulativeness, deceitfulness, callousness) are not diagnostic criteria for narcissistic personality disorder (see Criterion B) but can be specified when more pervasive antagonistic features (e.g., "malignant narcissism") are present. Other traits of Negative Affectivity (e.g., depressivity, anxiousness) can be specified to record more "vulnerable" presentations. Furthermore, although moderate or greater impairment in personality functioning is required for the diagnosis of narcissistic personality disorder (Criterion A), the level of personality functioning can also be specified.

Obsessive-Compulsive Personality Disorder

Typical features of obsessive-compulsive personality disorder are difficulties in establishing and sustaining close relationships, associated

with rigid perfectionism, inflexibility, and restricted emotional expression. Characteristic difficulties are apparent in identity, self-direction, empathy, and/or intimacy, as described below, along with specific maladaptive traits in the domains of Negative Affectivity and/or Detachment.

Proposed Diagnostic Criteria

A. Moderate or greater impairment in personality functioning, manifested by characteristic difficulties in two or more of the following four areas:
    1. ***Identity:*** Sense of self derived predominantly from work or productivity; constricted experience and expression of strong emotions.
    2. ***Self-direction:*** Difficulty completing tasks and realizing goals, associated with rigid and unreasonably high and inflexible internal standards of behavior; overly conscientious and moralistic attitudes.
    3. ***Empathy:*** Difficulty understanding and appreciating the ideas, feelings, or behaviors of others.
    4. ***Intimacy:*** Relationships seen as secondary to work and productivity; rigidity and stubbornness negatively affect relationships with others.

B. Three or more of the following four pathological personality traits, one of which must be (1) Rigid perfectionism:
    1. ***Rigid perfectionism*** (an aspect of extreme Conscientiousness [the opposite pole of Disinhibition]): Rigid insistence on everything being flawless, perfect, and without errors or faults, including one's own and others' performance; sacrificing of timeliness to ensure correctness in every detail; believing that there is only one right way to do things; difficulty changing ideas and/or viewpoint; preoccupation with details, organization, and order.

2. ***Perseveration*** (an aspect of **Negative Affectivity**): Persistence at tasks long after the behavior has ceased to be functional or effective; continuance of the same behavior despite repeated failures.
3. ***Intimacy avoidance*** (an aspect of **Detachment**): Avoidance of close or romantic relationships, interpersonal attachments, and intimate sexual relationships.
4. ***Restricted affectivity*** (an aspect of **Detachment**): Little reaction to emotionally arousing situations; constricted emotional experience and expression; indifference or coldness.

Specifiers. Trait and personality functioning specifiers may be used to record additional personality features that may be present in obsessive-compulsive personality disorder but are not required for the diagnosis. For example, other traits of Negative Affectivity (e.g., anxiousness) are not diagnostic criteria for obsessive-compulsive personality disorder (see Criterion B) but can be specified when appropriate. Furthermore, although moderate or greater impairment in personality functioning is required for the diagnosis of obsessive-compulsive personality disorder (Criterion A), the level of personality functioning can also be specified.

Schizotypal Personality Disorder

Typical features of schizotypal personality disorder are impairments in the capacity for social and close relationships and eccentricities in cognition, perception, and behavior that are associated with distorted self-image and incoherent personal goals and accompanied by suspiciousness and restricted emotional expression. Characteristic difficulties are apparent in identity, self-direction, empathy, and/or intimacy, along with specific maladaptive traits in the domains of Psychoticism and Detachment.

Proposed Diagnostic Criteria

A. Moderate or greater impairment in personality functioning, manifested by characteristic difficulties in two or more of the following four areas:
    1. ***Identity:*** Confused boundaries between self and others; distorted self-concept; emotional expression often not congruent with context or internal experience.
    2. ***Self-direction:*** Unrealistic or incoherent goals; no clear set of internal standards.
    3. ***Empathy:*** Pronounced difficulty understanding impact of own behaviors on others; frequent misinterpretations of others' motivations and behaviors.
    4. ***Intimacy:*** Marked impairments in developing close relationships, associated with mistrust and anxiety.

B. Four or more of the following six pathological personality traits:
    1. ***Cognitive and perceptual dysregulation*** (an aspect of **Psychoticism**): Odd or unusual thought processes; vague, circumstantial, metaphorical, overelaborate, or stereotyped thought or speech; odd sensations in various sensory modalities.
    2. ***Unusual beliefs and experiences*** (an aspect of **Psychoticism**): Thought content and views of reality that are viewed by others as bizarre or idiosyncratic; unusual experiences of reality.
    3. ***Eccentricity*** (an aspect of **Psychoticism**): Odd, unusual, or bizarre behavior or appearance; saying unusual or inappropriate things.
    4. ***Restricted affectivity*** (an aspect of **Detachment**): Little reaction to emotionally arousing situations; constricted emotional experience and expression; indifference or coldness.

5. ***Withdrawal*** (an aspect of **Detachment**): Preference for being alone to being with others; reticence in social situations; avoidance of social contacts and activity; lack of initiation of social contact.
6. ***Suspiciousness*** (an aspect of **Detachment**): Expectations of—and heightened sensitivity to—signs of interpersonal ill-intent or harm; doubts about loyalty and fidelity of others; feelings of persecution.

Specifiers. Trait and personality functioning specifiers may be used to record additional personality features that may be present in schizotypal personality disorder but are not required for the diagnosis. For example, traits of Negative Affectivity (e.g., depressivity, anxiousness) are not diagnostic criteria for schizotypal personality disorder (see Criterion B)

but can be specified when appropriate. Furthermore, although moderate or greater impairment in personality functioning is required for the diagnosis of schizotypal personality disorder (Criterion A), the level of personality functioning can also be specified.

Personality Disorder—Trait Specified

Proposed Diagnostic Criteria

A. Moderate or greater impairment in personality functioning, manifested by difficulties in two or more of the following four areas:
   1. ***Identity***
   2. ***Self-direction***
   3. ***Empathy***
   4. ***Intimacy***

B. One or more pathological personality trait domains OR specific trait facets within domains, considering ALL of the following domains:

1. **Negative Affectivity** (vs. Emotional Stability): Frequent and intense experiences of high levels of a wide range of negative emotions (e.g., anxiety, depression, guilt/shame, worry, anger), and their behavioral (e.g., self-harm) and interpersonal (e.g., dependency) manifestations.
2. **Detachment** (vs. Extraversion): Avoidance of socioemotional experience, including both withdrawal from interpersonal interactions, ranging from casual, daily interactions to friendships to intimate relationships, as well as restricted affective experience and expression, particularly limited hedonic capacity.
3. **Antagonism** (vs. Agreeableness): Behaviors that put the individual at odds with other people, including an exaggerated sense of self-importance and a concomitant expectation of special treatment, as well as a callous antipathy toward others, encompassing both unawareness of others' needs and feelings and a readiness to use others in the service of self-enhancement.
4. **Disinhibition** (vs. Conscientiousness): Orientation toward immediate gratification, leading to impulsive behavior driven by current thoughts, feelings, and external stimuli, without regard for past learning or consideration of future consequences.
5. **Psychoticism** (vs. Lucidity): Exhibiting a wide range of culturally incongruent odd, eccentric, or unusual behaviors and cognitions, including both process (e.g., perception, dissociation) and content (e.g., beliefs).

Subtypes. Because personality features vary continuously along multiple trait dimensions, a comprehensive set of potential expressions of PD-TS can be represented by DSM-5's dimensional model of maladaptive personality

trait variants (see Table 3, pp. 899–901). Thus, subtypes are unnecessary for PD-TS, and instead, the descriptive elements that constitute personality are provided, arranged in an empirically based model. This arrangement allows clinicians to tailor the description of each individual's personality disorder profile, considering all five broad domains of personality trait variation and drawing on the descriptive features of these domains as needed to characterize the individual.

Specifiers. The specific personality features of individuals are always recorded in evaluating Criterion B, so the combination of personality features characterizing an individual directly constitutes the specifiers in each case. For example, two individuals who are both characterized by emotional lability, hostility, and depressivity may differ such that the first individual is characterized additionally by callousness, whereas the second is not.

Personality Disorder Scoring Algorithms

The requirement for any two of the four A criteria for each of the six personality disorders was based on maximizing the relationship of these criteria to their corresponding personality disorder. Diagnostic thresholds for the B criteria were also set empirically to minimize change in prevalence of the disorders from DSM-IV and overlap with other personality disorders, and to maximize relationships with functional impairment. The resulting diagnostic criteria sets represent clinically useful personality disorders with high fidelity, in terms of core impairments in personality functioning of varying degrees of severity and constellations of pathological personality traits.

Personality Disorder Diagnosis

Individuals who have a pattern of impairment in personality functioning and maladaptive traits that matches one of the six defined personality disorders should be diagnosed with that personality disorder. If an individual also has one or even several prominent traits that may have

clinical relevance in addition to those required for the diagnosis (e.g., see narcissistic personality disorder), the option exists for these to be noted as specifiers. Individuals whose personality functioning or trait pattern is substantially different from that of any of the six specific personality disorders should be diagnosed with PD-TS. The individual may not meet the required number of A or B criteria and, thus, have a subthreshold presentation of a personality disorder. The individual may have a mix of features of personality disorder types or some features that are less characteristic of a type and more accurately considered a mixed or atypical presentation. The specific level of impairment in personality functioning and the pathological personality traits that characterize the individual's personality can be specified for PD-TS, using the Level of Personality Functioning Scale (Table 2) and the pathological trait taxonomy (Table 3). The current diagnoses of paranoid, schizoid, histrionic, and dependent personality disorders are represented also by the diagnosis of PD-TS; these are defined by moderate or greater impairment in personality functioning and can be specified by the relevant pathological personality trait combinations.

Level of Personality Functioning

Like most human tendencies, personality functioning is distributed on a continuum. Central to functioning and adaptation are individuals' characteristic ways of thinking about and understanding themselves and their interactions with others. An optimally functioning individual has a complex, fully elaborated, and well-integrated psychological world that includes a mostly positive, volitional, and adaptive self-concept; a rich, broad, and appropriately regulated emotional life; and the capacity to behave as a productive member of society with reciprocal and fulfilling interpersonal relationships. At the opposite end of the continuum, an individual with severe personality pathology has an impoverished, disorganized, and/or conflicted psychological world that includes a weak, unclear, and maladaptive self-concept; a propensity to negative, dysregulated emotions; and a deficient capacity for adaptive interpersonal functioning and social behavior.

Self and *Interpersonal* Functioning Dimensional Definition

Generalized severity may be the most important single predictor of concurrent and prospective dysfunction in assessing personality psychopathology. Personality disorders are optimally characterized by a generalized personality severity continuum with additional

specification of stylistic elements, derived from personality disorder symptom constellations and personality traits. At the same time, the core of personality psychopathology is impairment in ideas and feelings regarding self and interpersonal relationships; this notion is consistent with multiple theories of personality disorder and their research bases. The components of the Level of Personality Functioning Scale—identity, self-direction, empathy, and intimacy (see Table 1)—are particularly central in describing a personality functioning continuum.

Mental representations of the self and interpersonal relationships are reciprocally influential and inextricably tied, affect the nature of interaction with mental health professionals, and can have a significant impact on both treatment efficacy and outcome, underscoring the importance of assessing an individual's characteristic self-concept as well as views of other people and relationships. Although the degree of disturbance in the self and interpersonal functioning is continuously distributed, it is useful to consider the level of impairment in functioning for clinical characterization and for treatment planning and prognosis.

Rating Level of Personality Functioning

To use the Level of Personality Functioning Scale (LPFS), the clinician selects the level that most closely captures the individual's *current overall* level of impairment in personality functioning. The rating is necessary for the diagnosis of a personality disorder (moderate or greater impairment) and can be used to specify the severity of impairment present for an individual with any personality disorder at a given point in time. The LPFS may also be used as a global indicator of personality functioning without

specification of a personality disorder diagnosis, or in the event that personality impairment is subthreshold for a disorder diagnosis.

Personality Traits

Definition and Description

Criterion B in the alternative model involves assessments of personality traits that are grouped into five domains. A *personality trait* is a tendency to feel, perceive, behave, and think in relatively consistent ways across time and across situations in which the trait may manifest. For example, individuals with a high level of the personality trait of *anxiousness* would tend to *feel* anxious readily, including in circumstances in which most people would be calm and relaxed. Individuals high in trait anxiousness also would *perceive* situations to be anxiety-provoking more frequently than would individuals with lower levels of this trait, and those high in the trait would tend to *behave* so as to avoid situations that they *think* would make them anxious. They would thereby tend to *think* about the world as more anxiety provoking than other people.

Importantly, individuals high in trait anxiousness would not necessarily be anxious at all times and in all situations. Individuals' trait levels also can and do change throughout life. Some changes are very general and reflect maturation (e.g., teenagers generally are higher on trait impulsivity than are older adults), whereas other changes reflect individuals' life experiences.

Dimensionality of personality traits. All individuals can be located on the spectrum of trait dimensions; that is, personality traits apply to everyone in different degrees rather than being present versus absent. Moreover, personality traits, including those identified specifically in the Section III model, exist on a spectrum with two opposing poles. For example, the opposite of the trait of *callousness* is the tendency to be empathic and kind-hearted, even in circumstances in which most persons would not feel that way. Hence,

although in Section III this trait is labeled *callousness,* because that pole of the dimension is the primary focus, it could be described in full as *callousness versus kind-heartedness.* Moreover, its opposite pole can be recognized and may not be adaptive in all circumstances (e.g., individuals who, due to extreme kind-heartedness, repeatedly allow themselves to be taken advantage of by unscrupulous others).

Hierarchical structure of personality. Some trait terms are quite specific (e.g., "talkative") and describe a narrow range of behaviors, whereas others are quite broad (e.g., Detachment) and characterize a wide range of behavioral propensities. Broad trait dimensions are called *domains,* and specific trait dimensions are called *facets.* Personality trait *domains* comprise a spectrum of more specific personality *facets* that tend to occur together. For example, withdrawal and anhedonia are specific trait *facets* in the trait *domain* of Detachment. Despite some cross-cultural variation in personality trait facets, the broad domains they collectively comprise are relatively consistent across cultures.

The Personality Trait Model

The Section III personality trait system includes five broad domains of personality trait variation—Negative Affectivity (vs. Emotional Stability), Detachment (vs. Extraversion), Antagonism (vs. Agreeableness), Disinhibition (vs. Conscientiousness), and Psychoticism (vs. Lucidity)—comprising 25 specific personality trait facets. Table 3 provides definitions of all personality domains and facets. These five broad domains are maladaptive variants of the five domains of the extensively validated and replicated personality model known as the "Big Five," or Five Factor Model of personality (FFM), and are also similar to the domains of the Personality Psychopathology Five (PSY-5). The specific 25 facets represent a list of personality facets chosen for their clinical relevance.

Although the Trait Model focuses on personality traits associated with psychopathology, there are healthy, adaptive, and resilient personality traits identified as the polar opposites of these traits, as noted in the parentheses above (i.e., Emotional Stability, Extraversion, Agreeableness, Conscientiousness, and Lucidity). Their presence can greatly mitigate the

effects of mental disorders and facilitate coping and recovery from traumatic injuries and other medical illness.

Distinguishing Traits, Symptoms, and Specific Behaviors

Although traits are by no means immutable and do change throughout the life span, they show relative consistency compared with symptoms and specific behaviors. For example, a person may behave impulsively at a specific time for a specific reason (e.g., a person who is rarely impulsive suddenly decides to spend a great deal of money on a particular item because of an unusual opportunity to purchase something of unique value), but it is only when behaviors aggregate across time and circumstance, such that a pattern of behavior distinguishes between individuals, that they reflect traits. Nevertheless, it is important to recognize, for example, that even people who are impulsive are not acting impulsively all of the time. A trait is a tendency or disposition toward specific behaviors; a specific behavior is an instance or manifestation of a trait.

Similarly, traits are distinguished from most symptoms because symptoms tend to wax and wane, whereas traits are relatively more stable. For example, individuals with higher levels of *depressivity* have a greater likelihood of experiencing discrete episodes of a depressive disorder and of showing the symptoms of these disorders, such as difficulty concentrating. However, even patients who have a trait propensity to *depressivity* typically cycle through distinguishable episodes of mood disturbance, and specific symptoms such as difficulty concentrating tend to wax and wane in concert with specific episodes, so they do

not form part of the trait definition. Importantly, however, symptoms and traits are both amenable to intervention, and many interventions targeted at symptoms can affect the longer term patterns of personality functioning that are captured by personality traits.

Assessment of the DSM-5 Section III Personality Trait Model

The clinical utility of the Section III multidimensional personality trait model lies in its ability to focus attention on multiple relevant areas of personality variation in each individual patient. Rather than focusing attention on the identification of one and only one optimal diagnostic label, clinical application of the Section III personality trait model involves reviewing all five broad personality domains portrayed in Table 3. The clinical approach to personality is similar to the well-known review of systems in clinical medicine. For example, an individual's presenting complaint may focus on a specific neurological symptom, yet during an initial evaluation clinicians still systematically review functioning in all relevant systems (e.g., cardiovascular, respiratory, gastrointestinal), lest an important area of diminished functioning and corresponding opportunity for effective intervention be missed.

Clinical use of the Section III personality trait model proceeds similarly. An initial inquiry reviews all five broad domains of personality. This systematic review is facilitated by the use of formal psychometric instruments designed to measure specific facets and domains of personality. For example, the personality trait model is operationalized in the Personality Inventory for DSM-5 (PID-5), which can be completed in its self-report form by patients and in its informant-report form by those who know the patient well (e.g., a spouse). A detailed clinical assessment would involve collection of both patient- and informant-report data on all 25 facets of the personality trait model. However, if this is not possible, due to time or other constraints, assessment focused at the five-domain level is an acceptable clinical option when only a general (vs. detailed) portrait of a patient's personality is needed (see Criterion B of PD-TS). However, if personality-based problems are the focus of treatment, then it will be important to assess individuals' trait facets as well as domains.

Because personality traits are continuously distributed in the population, an approach to making the judgment that a specific trait is elevated (and therefore is present for diagnostic purposes) could involve comparing individuals' personality trait levels with population norms and/or clinical judgment. If a trait is elevated—that is, formal psychometric testing and/or interview data support the clinical judgment of elevation—then it is considered as contributing to meeting Criterion B of Section III personality disorders.

Clinical Utility of the Multidimensional Personality Functioning and Trait Model

Disorder and trait constructs each add value to the other in predicting important antecedent (e.g., family history, history of child abuse), concurrent (e.g., functional impairment, medication use), and predictive (e.g., hospitalization, suicide attempts) variables. DSM-5 impairments in personality functioning and pathological personality traits each contribute independently to clinical decisions about degree of disability; risks for self-harm, violence, and criminality; recommended treatment type and intensity; and prognosis—all important aspects of the utility of psychiatric diagnoses. Notably, knowing the level of an individual's personality functioning and his or her pathological trait profile also provides the clinician with a rich base of information and is valuable in treatment planning and in predicting the course and outcome of many mental disorders in addition to personality disorders. Therefore, assessment of personality functioning and pathological personality traits may be relevant whether an individual has a personality disorder or not.

TABLE 2 Level of Personality Functioning Scale

	SELF		INTERPERSONAL	
Level of impairment	**Identity**	**Self-direction**	**Empathy**	**Intimacy**
0—Little or no impairment	Has ongoing awareness of a unique self; maintains role-appropriate boundaries. Has consistent and self-regulated positive self-esteem, with accurate self-appraisal. Is capable of experiencing, tolerating, and	Sets and aspires to reasonable goals based on a realistic assessment of personal capacities. Utilizes appropriate standards of behavior, attaining fulfillment in multiple realms.	Is capable of accurately understanding others' experiences and motivations in most situations. Comprehends and appreciates others' perspectives, even if disagreeing.	Maintains multiple satisfying and enduring relationships in personal and community life. Desires and engages in a number of caring, close, and reciprocal relationships. Strives for cooperation and mutual benefit and flexibly responds to

	regulating a full range of emotions.	Can reflect on, and make constructive meaning of, internal experience.	Is aware of the effect of own actions on others.	a range of others' ideas, emotions, and behaviors.
1—Some impairment	Has relatively intact sense of self, with some decrease in clarity of boundaries when strong emotions and mental distress are experienced. Self-esteem diminished at times, with overly critical or somewhat distorted self-appraisal. Strong emotions may be distressing, associated with a restriction in range of emotional experience.	Is excessively goal-directed, somewhat goal-inhibited, or conflicted about goals. May have an unrealistic or socially inappropriate set of personal standards, limiting some aspects of fulfillment. Is able to reflect on internal experiences, but may overemphasize a single (e.g., intellectual, emotional) type of self-knowledge.	Is somewhat compromised in ability to appreciate and understand others' experiences; may tend to see others as having unreasonable expectations or a wish for control. Although capable of considering and understanding different perspectives, resists doing so. Has inconsistent awareness of effect of own behavior on others.	Is able to establish enduring relationships in personal and community life, with some limitations on degree of depth and satisfaction. Is capable of forming and desires to form intimate and reciprocal relationships, but may be inhibited in meaningful expression and sometimes constrained if intense emotions or conflicts arise. Cooperation may be inhibited by unrealistic standards; somewhat limited in ability to respect or respond to others' ideas, emotions, and behaviors.
2—Moderate impairment	Depends excessively on others for identity definition, with compromised boundary delineation. Has vulnerable self-esteem controlled by exaggerated concern about external evaluation, with a wish for approval.	Goals are more often a means of gaining external approval than self-generated, and thus may lack coherence and/or stability. Personal standards may be	Is hyperattuned to the experience of others, but only with respect to perceived relevance to self. Is excessively self-referential; significantly compromised	Is capable of forming and desires to form relationships in personal and community life, but connections may be largely superficial. Intimate relationships are predominantly based on meeting self-regulatory and self-esteem needs, with an unrealistic

	Has sense of incompleteness or inferiority, with compensatory inflated, or deflated, self-appraisal. Emotional regulation depends on positive external appraisal. Threats to self-esteem may engender strong emotions such as rage or shame.	unreasonably high (e.g., a need to be special or please others) or low (e.g., not consonant with prevailing social values). Fulfillment is compromised by a sense of lack of authenticity. Has impaired capacity to reflect on internal experience.	ability to appreciate and understand others' experiences and to consider alternative perspectives. Is generally unaware of or unconcerned about effect of own behavior on others, or unrealistic appraisal of own effect.	expectation of being perfectly understood by others. Tends not to view relationships in reciprocal terms, and cooperates predominantly for personal gain.
897 3—Severe impairment	Has a weak sense of autonomy/agency; experience of a lack of identity, or emptiness. Boundary definition is poor or rigid: may show overidentification with others, overemphasis on independence from others, or vacillation between these. Fragile self-esteem is easily influenced by events, and self-image lacks coherence. Self-appraisal is un-nuanced: self-loathing, self-aggrandizing, or an illogical, unrealistic combination. Emotions may be rapidly shifting or a chronic,	Has difficulty establishing and/or achieving personal goals. Internal standards for behavior are unclear or contradictory. Life is experienced as meaningless or dangerous. Has significantly compromised ability to reflect on and understand own mental processes.	Ability to consider and understand the thoughts, feelings, and behavior of other people is significantly limited; may discern very specific aspects of others' experience, particularly vulnerabilities and suffering. Is generally unable to consider alternative perspectives; highly threatened by differences of opinion or alternative viewpoints. Is confused about or unaware of	Has some desire to form relationships in community and personal life, but capacity for positive and enduring connections is significantly impaired. Relationships are based on a strong belief in the absolute need for the intimate other(s), and/or expectations of abandonment or abuse. Feelings about intimate involvement with others alternate between fear/rejection and desperate desire for connection. Little mutuality: others are conceptualized primarily in terms of how they affect the self (negatively

	unwavering feeling of despair.		impact of own actions on others; often bewildered about people's thoughts and actions, with destructive motivations frequently misattributed to others.	or positively); cooperative efforts are often disrupted due to the perception of slights from others.
898 4—Extreme impairment	Experience of a unique self and sense of agency/autonomy are virtually absent, or are organized around perceived external persecution. Boundaries with others are confused or lacking. Has weak or distorted self-image easily threatened by interactions with others; significant distortions and confusion around self-appraisal. Emotions not congruent with context or internal experience. Hatred and aggression may be dominant affects, although they may be disavowed and attributed to others.	Has poor differentiation of thoughts from actions, so goal-setting ability is severely compromised, with unrealistic or incoherent goals. Internal standards for behavior are virtually lacking. Genuine fulfillment is virtually inconceivable. Is profoundly unable to constructively reflect on own experience. Personal motivations may be unrecognized and/or experienced as external to self.	Has pronounced inability to consider and understand others' experience and motivation. Attention to others' perspectives is virtually absent (attention is hypervigilant, focused on need fulfillment and harm avoidance). Social interactions can be confusing and disorienting.	Desire for affiliation is limited because of profound disinterest or expectation of harm. Engagement with others is detached, disorganized, or consistently negative. Relationships are conceptualized almost exclusively in terms of their ability to provide comfort or inflict pain and suffering. Social/interpersonal behavior is not reciprocal; rather, it seeks fulfillment of basic needs or escape from pain.

TABLE 3 Definitions of DSM-5 personality disorder trait domains and facets

DOMAINS (Polar Opposites) and Facets	Definitions
NEGATIVE AFFECTIVITY (vs. Emotional Stability)	Frequent and intense experiences of high levels of a wide range of negative emotions (e.g., anxiety, depression, guilt/shame, worry, anger) and their behavioral (e.g., self-harm) and interpersonal (e.g., dependency) manifestations.
Emotional lability	Instability of emotional experiences and mood; emotions that are easily aroused, intense, and/or out of proportion to events and circumstances.
Anxiousness	Feelings of nervousness, tenseness, or panic in reaction to diverse situations; frequent worry about the negative effects of past unpleasant experiences and future negative possibilities; feeling fearful and apprehensive about uncertainty; expecting the worst to happen.
Separation insecurity	Fears of being alone due to rejection by—and/or separation from—significant others, based in a lack of confidence in one's ability to care for oneself, both physically and emotionally.
Submissiveness	Adaptation of one's behavior to the actual or perceived interests and desires of others even when doing so is antithetical to one's own interests, needs, or desires.
Hostility	Persistent or frequent angry feelings; anger or irritability in response to minor slights and insults; mean, nasty, or vengeful behavior. *See also* Antagonism.
Perseveration	Persistence at tasks or in a particular way of doing things long after the behavior has ceased to be functional or effective; continuance of the same behavior despite repeated failures or clear reasons for stopping.
Depressivity	*See* Detachment.
Suspiciousness	*See* Detachment.
Restricted affectivity (lack of)	The ***lack of*** this facet characterizes ***low levels*** of Negative Affectivity. *See* Detachment for definition of this facet.
DETACHMENT (vs. Extraversion)	Avoidance of socioemotional experience, including both withdrawal from interpersonal interactions (ranging from casual, daily interactions to friendships to intimate relationships) and restricted affective experience and expression, particularly limited hedonic capacity.
Withdrawal	Preference for being alone to being with others; reticence in social situations; avoidance of social contacts and activity; lack of initiation of social contact.
Intimacy avoidance	Avoidance of close or romantic relationships, interpersonal attachments, and intimate sexual relationships.
Anhedonia	Lack of enjoyment from, engagement in, or energy for life's experiences; deficits in the capacity to feel pleasure and take interest in things.

DOMAINS (Polar Opposites) and Facets	Definitions
Depressivity	Feelings of being down, miserable, and/or hopeless; difficulty recovering from such moods; pessimism about the future; pervasive shame and/or guilt; feelings of inferior self-worth; thoughts of suicide and suicidal behavior.
Restricted affectivity	Little reaction to emotionally arousing situations; constricted emotional experience and expression; indifference and aloofness in normatively engaging situations.
Suspiciousness	Expectations of—and sensitivity to signs of—interpersonal ill-intent or harm; doubts about loyalty and fidelity of others; feelings of being mistreated, used, and/or persecuted by others.
900 ANTAGONISM (vs. Agreeableness)	Behaviors that put the individual at odds with other people, including an exaggerated sense of self-importance and a concomitant expectation of special treatment, as well as a callous antipathy toward others, encompassing both an unawareness of others' needs and feelings and a readiness to use others in the service of self-enhancement.
Manipulativeness	Use of subterfuge to influence or control others; use of seduction, charm, glibness, or ingratiation to achieve one's ends.
Deceitfulness	Dishonesty and fraudulence; misrepresentation of self; embellishment or fabrication when relating events.
Grandiosity	Believing that one is superior to others and deserves special treatment; self-centeredness; feelings of entitlement; condescension toward others.
Attention seeking	Engaging in behavior designed to attract notice and to make oneself the focus of others' attention and admiration.
Callousness	Lack of concern for the feelings or problems of others; lack of guilt or remorse about the negative or harmful effects of one's actions on others.
Hostility	*See* Negative Affectivity.
DISINHIBITION (vs. Conscientiousness)	Orientation toward immediate gratification, leading to impulsive behavior driven by current thoughts, feelings, and external stimuli, without regard for past learning or consideration of future consequences.
Irresponsibility	Disregard for—and failure to honor—financial and other obligations or commitments; lack of respect for—and lack of follow-through on—agreements and promises; carelessness with others' property.
Impulsivity	Acting on the spur of the moment in response to immediate stimuli; acting on a momentary basis without a plan or consideration of outcomes; difficulty establishing and following plans; a sense of urgency and self-harming behavior under emotional distress.
Distractibility	Difficulty concentrating and focusing on tasks; attention is easily diverted by extraneous stimuli; difficulty maintaining goal-focused behavior, including both planning and completing tasks.

DOMAINS (Polar Opposites) and Facets	Definitions
Risk taking	Engagement in dangerous, risky, and potentially self-damaging activities, unnecessarily and without regard to consequences; lack of concern for one's limitations and denial of the reality of personal danger; reckless pursuit of goals regardless of the level of risk involved.
Rigid perfectionism (lack of)	Rigid insistence on everything being flawless, perfect, and without errors or faults, including one's own and others' performance; sacrificing of timeliness to ensure correctness in every detail; believing that there is only one right way to do things; difficulty changing ideas and/or viewpoint; preoccupation with details, organization, and order. The ***lack of*** this facet characterizes ***low levels*** of Disinhibition.
901 PSYCHOTICISM (vs. Lucidity)	Exhibiting a wide range of culturally incongruent odd, eccentric, or unusual behaviors and cognitions, including both process (e.g., perception, dissociation) and content (e.g., beliefs).
Unusual beliefs and experiences	Belief that one has unusual abilities, such as mind reading, telekinesis, thought-action fusion; unusual experiences of reality, including hallucination-like experiences.
Eccentricity	Odd, unusual, or bizarre behavior, appearance, and/or speech; having strange and unpredictable thoughts; saying unusual or inappropriate things.
Cognitive and perceptual dysregulation	Odd or unusual thought processes and experiences, including depersonalization, derealization, and dissociative experiences; mixed sleep-wake state experiences; thought-control experiences.

Conditions for Further Study

Proposed criteria sets are presented for conditions on which future research is encouraged. It is hoped that such research will allow the field to better understand these conditions and inform future decisions about possible placement in forthcoming editions of DSM. Notably, persistent complex bereavement disorder, originally located in this section, has been moved to the chapter "Trauma- and Stressor-Related Disorders" as an official diagnosis in Section II. On the basis of thorough reviews finding sufficient evidence of validity, reliability, and clinical utility to justify its new placement, it is now named "prolonged grief disorder" and the criteria have been appropriately reformulated.

The specific items, thresholds, and minimum durations contained in these research criteria sets were set by expert consensus—informed by literature review, data reanalysis, and field trial results, where available—and are intended to provide a common language for researchers and clinicians who are interested in studying these disorders. The DSM-5 Task Force and Work Groups subjected each of these proposed criteria sets to a careful empirical review and invited wide commentary from the field as well as from the general public. The Task Force ultimately determined that there was insufficient evidence to warrant inclusion of these proposals as official mental disorder diagnoses in Section II of DSM-5. ***These proposed criteria sets are therefore not intended for clinical use; only the criteria sets and disorders in Section II of DSM-5 are officially recognized and should be used for clinical purposes.***

Attenuated Psychosis Syndrome

Proposed Criteria

A. At least one of the following symptoms is present and is of sufficient severity or frequency to warrant clinical attention:
    1. Attenuated delusions.
    2. Attenuated hallucinations.
    3. Attenuated disorganized speech.
B. Symptom(s) must have been present at least once per week for the past month.
C. Symptom(s) must have begun or worsened in the past year.
D. Symptom(s) is sufficiently distressing and disabling to the individual to warrant clinical attention.
E. Symptom(s) is not better explained by another mental disorder, including a depressive or bipolar disorder with psychotic features, and is not attributable to the physiological effects of a substance or another medical condition.
F. Criteria for any psychotic disorder have never been met.

Diagnostic Features

Attenuated psychotic symptoms, as defined in Criterion A, are psychosis-like but below the threshold to be considered a psychotic symptom that would count toward the diagnosis of a psychotic disorder. Compared with full psychotic disorders, the symptoms are less severe and more transient. Moreover, the individual maintains reasonable insight into the psychotic-like experiences and generally appreciates that perceptions are altered, and magical ideation is not compelling. Attenuated psychosis does not have the fixed nature that is necessary for the diagnosis of a full-blown psychotic disorder. In attenuated psychosis, doubt about beliefs can be elicited, skepticism about perceptions can be induced, and insight can be tested using open-ended questions, such as “I see that this is how you experience the world—could there be a different explanation?” A diagnosis of attenuated psychosis syndrome requires state psychopathology associated

with functional impairment rather than long-standing trait pathology. The psychopathology has not progressed to full psychotic severity. Changes in experiences and behaviors are noted by the individual or others, suggesting a clinically significant change in mental state (i.e., the symptoms are of sufficient severity or frequency to warrant clinical attention) (Criterion A).

Attenuated delusions (Criterion A1) may have suspiciousness/persecutory ideational content, including persecutory ideas of reference. The individual may have a guarded, distrustful attitude. When this type of attenuated delusion is moderate in severity, the individual views others as untrustworthy and may be hypervigilant or sense ill will in others. When the attenuated delusions are severe but below the threshold to be considered psychotic, the individual entertains loosely organized beliefs about danger or hostile intention. Guarded behavior in the interview can interfere with the ability to gather information, and the propensity for viewing the world as hostile and dangerous is strong. On the other hand, attenuated delusions may have grandiose content presenting as an unrealistic sense of superior capacity. When this type of attenuated delusion is moderate in severity, the individual harbors notions of being gifted, influential, or special. When the attenuated delusions are severe, the individual has beliefs of superiority that often alienate friends and worry relatives. Thoughts of being special may lead to unrealistic plans and investments.

Attenuated hallucinations (Criterion A2) include alterations in sensory perceptions, usually auditory and/or visual. When the attenuated hallucinations are moderate, the sounds and images are often unformed (e.g., shadows, trails, halos, murmurs, rumbling), and they are experienced as unusual or puzzling. When the attenuated hallucinations are severe, these experiences become more vivid and frequent (i.e., recurring illusions or hallucinations that capture attention and affect thinking and concentration). These perceptual abnormalities may disrupt behavior, but skepticism about their reality can still be induced.

Attenuated disorganized communication (Criterion A3) may manifest as odd speech (vague, metaphorical, overelaborate, stereotyped), unfocused speech (confused, muddled, too fast or too slow, wrong words, irrelevant context, off track), or meandering speech (circumstantial, tangential). When the disorganization is moderately severe, the individual frequently gets into

irrelevant topics but responds easily to clarifying questions. Speech becomes meandering and circumstantial and may be odd but understandable. When the disorganization is severe, the individual fails to get to the point without external guidance (tangential). At a more severe level, some thought blocking or loose associations may occur infrequently, especially when the individual is under pressure, but reorienting questions quickly return structure and organization to the conversation.

The individual must experience distress and/or impaired performance in social or role functioning (Criterion D), and the individual or responsible others must note the changes and express concern, such that clinical care is indicated (Criterion A).

Measures are available to determine whether Criteria A–E are met or to broadly identify a clinical high-risk state for psychosis.

Associated Features

The individual may experience magical thinking, difficulty in concentration, some disorganization in thought or behavior, excessive suspiciousness, anxiety, social withdrawal, and disruption in sleep-wake cycle. Impaired cognitive function and negative symptoms are often observed.

Neuroimaging variables distinguish cohorts with attenuated psychosis syndrome from normal control cohorts with patterns similar to, but less severe than, that observed in schizophrenia. However, neuroimaging data are not diagnostic at the individual level.

Prevalence

Very little information is available about prevalence. However, in Switzerland, where one of the few relevant studies was conducted, the prevalence of attenuated psychosis syndrome in non-help-seeking individuals ages 16–40 years was found to be only 0.3%. Another 2.3% have attenuated symptoms that meet Criterion A, but these symptoms either began prior to the past year or had not worsened in the past year, as required

by Criterion C. In up to 7% of the general population across a broad range of countries, individuals acknowledge experiencing attenuated delusions or hallucinations. While the prevalence of Criterion A symptoms can be higher or lower across countries or ethnonational groups, the prevalence of attenuated psychosis symptoms tends to be higher among migrant groups than among native populations, possibly due to higher exposure to trauma and discrimination.

Development and Course

Onset of attenuated psychosis syndrome is usually in mid-to-late adolescence or early adulthood. It may be preceded by normal development or evidence for impaired cognition, negative symptoms, or impaired social development. In help-seeking cohorts, those whose presentations met criteria for attenuated psychosis syndrome had an increased probability of developing psychosis compared with those whose presentations did not meet the criteria. In the group whose presentations met criteria, the 3-year cumulative risk was up to 22%, and in the group whose presentations did not meet criteria, the 3-year cumulative risk of psychosis was 1.54%. Factors predicting progression to a full psychotic disorder (most frequently schizophrenia spectrum disorder) include male sex, lifetime stress/trauma, unemployment, living alone, severity of attenuated positive psychotic symptoms, severity of negative symptoms, disorganized and cognitive symptoms, and poor functioning. Eleven percent of those attenuated psychosis syndrome cases that progress to full psychosis develop affective psychosis (depressive or bipolar disorder with psychotic features), whereas 73% of attenuated psychosis syndrome cases that progress to full psychosis develop a schizophrenia spectrum disorder. Most evidence has validated attenuated psychotic symptom criteria in individuals ages 12–35 years, but there is only limited evidence in the youngest. Although the highest risk for transition to psychosis is within the first 2 years, individuals continue to be at risk for up to 10 years after initial referral, with an overall risk of transition of 34.9% over a 10-year period. Individuals presenting with attenuated psychosis syndrome may display other poor clinical outcomes beyond the development of psychosis, such as persistent attenuated psychotic symptoms, persistent or recurrent comorbid mental disorders,

disability, and low functioning. Clinical remission is present in only one-third of individuals with attenuated psychosis syndrome. Overall, about one-third of these individuals would develop psychosis, one-third would remit, and one-third would present persistent disability.

Risk and Prognostic Factors

Temperamental. Factors predicting prognosis of attenuated psychosis syndrome have not been definitively characterized.

Genetic and physiological. In individuals whose symptoms meet criteria for attenuated psychosis syndrome, there is no evidence that a family history of psychosis increases the risk of psychosis compared with control subjects over a 4-year period. Structural, functional, electrophysiological, and neurochemical imaging data are associated with increased risk of transition to psychosis. However, these predictors have not yet been validated for clinical use.

Culture-Related Diagnostic Issues

Assessing the presence of attenuated symptoms without considering the impact of sociocultural context can be difficult. Some perceptual experiences (e.g., hearing noises, seeing shadows) and religious or supernatural beliefs (e.g., evil eye, causing illness through curses, influence of spirits) may be considered odd in some cultural contexts and accepted in others. In addition, populations that experience trauma or persecution (e.g., torture, political violence, racism, discrimination) can report symptoms and fears that may be misjudged as attenuated or frank paranoid delusions, because of the impact of trauma on the individual's mood and communication (e.g., some fears may be appropriate to avoid threats, and may commingle with fears of recurrence of trauma or posttraumatic symptoms). Groups at higher risk of misdiagnosis include migrants, socially oppressed ethnic and racialized populations, and other groups facing social adversity and discrimination. The distress and impairment criterion helps to distinguish socioculturally normative experiences from symptoms of

attenuated psychosis syndrome (e.g., adaptive wariness toward authority figures by discriminated groups, which may be confused with paranoia).

Functional Consequences of Attenuated Psychosis Syndrome

Many individuals may experience functional impairments at presentation. Modest-to-moderate impairment in social and role functioning may persist even with abatement of symptoms.

Differential Diagnosis

Brief psychotic disorder. When symptoms of attenuated psychosis syndrome initially manifest, they may resemble symptoms of brief psychotic disorder. However, in attenuated psychosis syndrome, the attenuated symptoms (delusions, hallucinations, or disorganized speech) do not cross the psychosis threshold.

Schizotypal personality disorder. Symptomatic features of schizotypal personality disorder, particularly during early stages of presentation, are similar to those of attenuated psychosis syndrome. However, schizotypal personality disorder is a relatively stable trait disorder not meeting the state-dependent aspects (Criterion C) of attenuated psychosis syndrome. In addition, a broader array of symptoms is required for the diagnosis of schizotypal personality disorder.

Reality distortions occurring in other mental disorders. Reality distortions that can resemble attenuated delusions can occur in the context of other mental disorders (e.g., feelings of low self-esteem or attributions of low regard from others in the context of major depressive disorder, a feeling of being the focus of undesired attention in the context of social anxiety disorder, inflated self-esteem in the context of pressured speech and reduced need for sleep in bipolar I or bipolar II disorder, a sense of being unable to experience feelings in the context of an intense fear of real or imagined abandonment and recurrent self-mutilation in borderline personality disorder). If these reality distortions occur only during

the course of another mental disorder, an additional diagnosis of attenuated psychosis syndrome would not be made.

Adjustment reaction of adolescence. Mild, transient symptoms typical of normal development and consistent with the degree of stress experienced do not qualify for attenuated psychosis syndrome.

Extreme end of perceptual aberration and magical thinking in the non-ill population. This diagnostic possibility should be strongly entertained when reality distortions are not associated with distress and functional impairment and need for care.

Substance/medication-induced psychotic disorder. Attenuated delusions and attenuated hallucinations can occur in the context of intoxication with cannabis, hallucinogens, phencyclidine, inhalants, and stimulants, or during withdrawal from alcohol and sedatives, hypnotics, or anxiolytics. Attenuated psychosis syndrome should not be diagnosed if the attenuated psychotic symptoms occur only during substance use, in which case a diagnosis of substance/medication-induced psychotic disorder may be preferred.

Attention-deficit/hyperactivity disorder. A history of attentional impairment does not exclude a current attenuated psychosis syndrome diagnosis. Earlier attentional impairment may be a prodromal condition or comorbid attention-deficit/hyperactivity disorder.

Comorbidity

Most individuals with attenuated psychosis syndrome experience some comorbid mental disorder, mostly depression (41%) and/or anxiety (15%). A little more than half of individuals have at least one comorbid disorder at follow-up, most of which were present when the individual was first assessed; the persistence of comorbid disorders at follow-up is associated with poor clinical and functional outcomes. Although some individuals with an attenuated psychosis syndrome diagnosis will progress to developing a new diagnosis, including anxiety, depressive, bipolar, and personality disorders, individuals with attenuated psychosis syndrome are not at increased risk of developing new nonpsychotic disorders compared with help-seeking control subjects.

Depressive Episodes With Short-Duration Hypomania

Proposed Criteria

Lifetime experience of at least one major depressive episode meeting the following criteria:

A. Five (or more) of the following criteria have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either (1) depressed mood or (2) loss of interest or pleasure. (**Note:** Do not include symptoms that are clearly attributable to a medical condition.)

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad, empty, or hopeless) or observation made by others (e.g., appears tearful). (**Note:** In children and adolescents, can be irritable mood.)
2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation).
3. Significant weight loss when not dieting or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. (**Note:** In children, consider failure to make expected weight gain.)

4. Insomnia or hypersomnia nearly every day.
5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).

6. Fatigue or loss of energy nearly every day.
7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).
8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).
9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

B. The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The disturbance is not attributable to the physiological effects of a substance or another medical condition.

D. The disturbance is not better explained by schizoaffective disorder and is not superimposed on schizophrenia, schizophreniform disorder, delusional disorder, or other specified or unspecified schizophrenia spectrum and other psychotic disorder.

At least two lifetime episodes of hypomanic periods that involve the required criterion symptoms below but are of insufficient duration (at least 2 days but less than 4 consecutive days) to meet criteria for a hypomanic episode. The criterion symptoms are as follows:

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood and abnormally and persistently increased activity or energy.

B. During the period of mood disturbance and increased energy and activity, three (or more) of the following symptoms have persisted (four if the mood is only irritable), represent a noticeable change from usual behavior, and have been present to a significant degree:

1. Inflated self-esteem or grandiosity.
2. Decreased need for sleep (e.g., feels rested after only 3 hours of sleep).
3. More talkative than usual or pressured to keep talking.
4. Flight of ideas or subjective experience that thoughts are racing.
5. Distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli), as reported or observed.
6. Increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation.
7. Excessive involvement in activities that have a high potential for painful consequences (e.g., the individual engages in unrestrained buying sprees, sexual indiscretions, or foolish business investments).

C. The episode is associated with an unequivocal change in functioning that is uncharacteristic of the individual when not symptomatic.

D. The disturbance in mood and the change in functioning are observable by others.

E. The episode is not severe enough to cause marked impairment in social or occupational functioning or to necessitate hospitalization. If there are psychotic features, the episode is, by definition, manic.

F. The episode is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication or other treatment).

Diagnostic Features

Individuals with depressive episodes with short-duration hypomania have experienced at least one major depressive episode as well as at least two

episodes of 2–3 days' duration in which criteria for a hypomanic episode were met (except for symptom duration). These episodes are of sufficient intensity to be categorized as a hypomanic episode but do not meet the 4-day duration requirement. Symptoms are present to a significant degree, such that they represent a noticeable change from the individual's normal behavior.

An individual with a history of a syndromal hypomanic episode and a major depressive episode by definition has bipolar II disorder, regardless of current duration of hypomanic symptoms.

Associated Features

Individuals who have experienced both short-duration hypomania and a major depressive episode, with their increased psychiatric comorbidity, greater family history of bipolar disorder, earlier onset, more recurrent major depressive episodes, and higher rate of suicide attempts, more closely resemble individuals with bipolar disorder than those with major depressive disorder.

Prevalence

The prevalence of depressive episodes with short-duration hypomania is unclear, as epidemiological studies have yet to be published using the DSM-5 definition. Using somewhat different criteria (subthreshold hypomania defined by either of the following: duration shorter than 4 days or having fewer than three Criterion B symptoms), major depressive disorder with subthreshold hypomania occurs in up to 6.7% of the U.S. population, making it more common than bipolar I or II disorder. In clinical settings studied across diverse countries, however, depressive episodes with short-duration hypomania is about one-fourth as common as depressive episodes with full-duration hypomania. Depressive episodes with short-duration hypomania may be more common in women, who may present with more features of atypical depression.

Risk and Prognostic Factors

Genetic and physiological.

A family history of bipolar disorder is three to four times more common among individuals with depressive episodes with short-duration hypomania than among those with major depressive disorder, whereas family history of bipolar disorder is similar among individuals with depressive episodes and short- versus full-duration hypomania.

Association With Suicidal Thoughts or Behavior

Individuals with depressive episodes with short-duration hypomania have higher rates of suicide attempts than individuals with major depressive disorder and similar rates of suicide attempts compared with individuals with depressive episodes and full-duration hypomania (bipolar II disorder).

Functional Consequences of Short-Duration Hypomania

Functional impairments associated specifically with depressive episodes with short-duration hypomania are as yet not fully determined. However, research suggests that individuals with this disorder have similar global assessment of functioning scores as compared to those with depressive episodes with full-duration hypomania.

Differential Diagnosis

Bipolar II disorder. Bipolar II disorder is characterized by major depressive episodes and hypomanic episodes, whereas depressive episodes with short-duration hypomania are characterized by depressive episodes with periods of 2–3 days of hypomanic symptoms. Once an individual has experienced a full-blown hypomanic episode lasting 4 days or more in addition to lifetime major depressive episodes, the diagnosis changes to and remains bipolar II disorder regardless of the duration of future hypomanic symptom periods.

Major depressive disorder. Major depressive disorder is also characterized by at least one lifetime major depressive episode. However, the additional presence of at least two lifetime periods of 2–3 days of hypomanic symptoms leads to a diagnosis of depressive episodes with short-duration hypomania rather than to major depressive disorder.

Major depressive disorder with mixed features. Both major depressive disorder with mixed features and depressive episodes with short-duration hypomania are characterized by the presence of some hypomanic symptoms and a major depressive episode. However, major depressive disorder with mixed features is characterized by hypomanic features that manifest *concurrently* with a major depressive episode, whereas individuals with depressive episodes with short-duration hypomania experience subsyndromal hypomania and fully syndromal major depression at different times.

Bipolar I disorder. Bipolar I disorder is differentiated from depressive episodes with short-duration hypomania by at least one lifetime manic episode, which is longer (at least 1 week) and more severe (causing marked impairment in social or occupational functioning or necessitating hospitalization to prevent harm to self and others) than a hypomanic episode. An episode (of any duration) that involves psychotic symptoms or necessitates hospitalization is by definition a manic episode rather than a hypomanic one.

Cyclothymic disorder. While cyclothymic disorder is characterized by periods of depressive symptoms and periods of hypomanic symptoms, the lifetime presence of a major depressive episode precludes the diagnosis of cyclothymic disorder.

Caffeine Use Disorder

Proposed Criteria

A problematic pattern of caffeine use leading to clinically significant impairment or distress, as manifested by at least the first three of the following criteria occurring within a 12-month period:

1. A persistent desire or unsuccessful efforts to cut down or control caffeine use.
2. Continued caffeine use despite knowledge of having a persistent or recurrent physical or psychological problem that

is likely to have been caused or exacerbated by caffeine.

3. Withdrawal, as manifested by either of the following:
   a. The characteristic withdrawal syndrome for caffeine.
   b. Caffeine (or a closely related substance) is taken to relieve or avoid withdrawal symptoms.
4. Caffeine is often taken in larger amounts or over a longer period than was intended.
5. Recurrent caffeine use resulting in a failure to fulfill major role obligations at work, school, or home (e.g., repeated tardiness or absences from work or school related to caffeine use or withdrawal).
6. Continued caffeine use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of caffeine (e.g., arguments with spouse about consequences of use, medical problems, cost).

7. Tolerance, as defined by either of the following:
   a. A need for markedly increased amounts of caffeine to achieve desired effect.
   b. Markedly diminished effect with continued use of the same amount of caffeine.
8. A great deal of time is spent in activities necessary to obtain caffeine, use caffeine, or recover from its effects.
9. Craving or a strong desire or urge to use caffeine.

Various research studies have provided documentation and characterization of individuals with problematic caffeine use, and several reviews provide an analysis of this literature. The working diagnostic algorithm proposed for the study of caffeine use disorder differs from that of the other substance use disorders, reflecting the need to identify only cases that have sufficient clinical importance to warrant the labeling of a mental disorder. A key goal of including caffeine use disorder in this section of DSM-5 is to stimulate research that will determine the reliability,

validity, and prevalence of caffeine use disorder based on the proposed diagnostic schema, with particular attention to the association of the diagnosis with functional impairments as part of validity testing.

The proposed criteria for caffeine use disorder reflect the need for a diagnostic threshold higher than that used for the other substance use disorders. Such a threshold is intended to prevent overdiagnosis of caffeine use disorder due to the high rate of habitual nonproblematic daily caffeine use in the general population.

Diagnostic Features

Caffeine use disorder is characterized by the continued use of caffeine and failure to control use despite negative physical and/or psychological consequences. In two U.S. population surveys, 14%–17% of caffeine users endorsed caffeine use despite physical or psychological problems, 34%–45% reported a persistent desire or unsuccessful efforts to control caffeine use, and 18%–27% reported withdrawal or using caffeine to relieve or avoid withdrawal. In these same surveys, some caffeine users reported using more caffeine than intended, spending a great deal of time using or obtaining caffeine (e.g., drinking coffee all day and until the evening), tolerance, a strong desire or craving for caffeine, failure to fulfill major role obligations due to caffeine (e.g., spending family vacation time searching for caffeinated beverages, resulting in relationship distress; repeatedly late for work due to need to get coffee), and, to a much lesser extent, caffeine use despite social or interpersonal problems. Medical and psychological problems attributed to caffeine included heart, stomach, and urinary problems, and complaints of anxiety, depression, insomnia, irritability, and difficulty thinking.

In a study of 2,259 Hungarian caffeine consumers, factor analysis of the nine caffeine use disorder criteria resulted in a one-factor solution, suggesting that caffeine use disorder is a unitary construct. In two Baltimore-area caffeine treatment studies, the most commonly endorsed criteria were withdrawal (97%), persistent desire or unsuccessful efforts to control use (91%–94%), and use despite knowledge of physical or psychological problems caused by caffeine (75%–91%).

Among individuals seeking treatment for problematic caffeine use, 88% reported having made prior serious attempts to modify caffeine use, and 43%–47% reported having been advised by a medical professional to reduce or eliminate caffeine. Common reported reasons for modifying caffeine use were health-related (59%) and a desire to not be dependent on caffeine (35%).

The text for caffeine withdrawal in the Section II chapter "Substance-Related and Addictive Disorders" provides information on the features of the withdrawal criterion. It is well documented that habitual caffeine users can experience a well-defined withdrawal syndrome upon acute abstinence from caffeine, and many caffeine-dependent individuals report continued use of caffeine to avoid experiencing withdrawal symptoms.

Prevalence

The prevalence of caffeine use disorder in the general population is unclear. One population-based study in Vermont reported that 9% of individuals endorsed the three proposed DSM-5 caffeine use disorder criteria plus tolerance. In a sample of 1,006 caffeine-consuming adults recruited using demographic quotas to reflect the U.S. population, 8% endorsed all three criteria required for a caffeine use disorder diagnosis.

In a sample of caffeine-consuming adolescents presenting for routine medical care in a Boston hospital, 3.9% endorsed all three criteria required for a caffeine use disorder diagnosis. Among a convenience sample of caffeine consumers in Hungary, 13.9% endorsed all three criteria, with 4.3% of those reporting that the symptoms caused significant distress in their everyday life.

Development and Course

Individuals whose pattern of use meets criteria for a caffeine use disorder have shown a wide range of daily caffeine intake and have been consumers of various types of caffeinated products (e.g., coffee, soft drinks, tea, energy drinks) and medications. A diagnosis of caffeine use disorder has been

shown to prospectively predict a greater incidence of caffeine reinforcement and more severe withdrawal.

There has been no longitudinal or cross-sectional lifespan research on caffeine use disorder. Caffeine use disorder has been identified in both adolescents and adults. Rates of caffeine consumption and overall level of caffeine consumption in the United States tend to increase with age. Age-related factors for caffeine use disorder are unknown, although concern is growing related to excessive caffeine consumption among adolescents and young adults through use of caffeinated energy drinks.

Risk and Prognostic Factors

Genetic and physiological. Heritabilities of heavy caffeine use, caffeine tolerance, and caffeine withdrawal range from 35% to 77%. For caffeine use, alcohol use, and cigarette smoking, a common genetic factor (polysubstance use) underlies the use of these three substances, with 28%–41% of the heritable effects of caffeine use (or heavy use) shared with alcohol and smoking. Caffeine and tobacco use and use disorders are associated with and substantially influenced by genetic factors unique to these licit drugs. The magnitude of heritability for caffeine use disorder markers appears to be similar to that for alcohol and tobacco use disorder markers.

Culture-Related Diagnostic Issues

Consumption of caffeine is affected by geographic origin, cultural context, lifestyle, social behavior, and economic status. The type of caffeinated beverage preferred (e.g., tea; coffee; carbonated sodas containing caffeine; *mate* [a beverage made from the herb *yerba mate*]) and the mode of preparation vary globally, leading to marked differences in the amounts and types of compounds in a “cup” of coffee, tea, or *mate*. These differences must be considered when assessing the quantity of caffeine ingested.

Association With Suicidal Thoughts or Behavior

No research specifically addresses the relationship between caffeine use disorder and suicidal thoughts or behavior. There is contradictory evidence

regarding caffeine consumption; namely, that high levels of caffeine consumption either may be associated with increased risk for suicidal thoughts or behavior or may be protective for suicidal thoughts or behavior.

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Functional Consequences of Caffeine Use Disorder

One U.S. population survey found that those who fulfilled the criteria for caffeine use disorder were more likely to report greater caffeine-related distress, feeling bad or guilty about caffeine use, sleep problems, anxiety, depression, and stress. A greater number of total symptoms endorsed also predicted these negative outcomes. Caffeine use disorder may predict greater use of caffeine during pregnancy.

Differential Diagnosis

Nonproblematic use of caffeine. The distinction between nonproblematic use of caffeine and caffeine use disorder can be difficult to make because social, behavioral, or psychological problems may be difficult to attribute to the substance, especially in the context of use of other substances. Regular, heavy caffeine use that can result in tolerance and withdrawal is relatively common, which by itself should not be sufficient for making a diagnosis.

Other stimulant use disorder. Problems related to use of other stimulant medications or substances may approximate the features of caffeine use disorder.

Anxiety disorders. Chronic heavy caffeine use may mimic generalized anxiety disorder, and acute caffeine consumption may produce and mimic panic attacks.

Comorbidity

Comorbidities associated with caffeine use disorder include daily cigarette smoking, cannabis use disorder, and a family or personal history of alcohol use disorder. Compared with individuals in the general population, rates of caffeine use disorder are higher among those seeking treatment for problematic caffeine use; individuals who use tobacco; high school and

college students; and those with histories of alcohol or illicit drug misuse. Features of caffeine use disorder may be positively associated with several diagnoses: major depression, generalized anxiety disorder, panic disorder, antisocial personality disorder, and alcohol, cannabis, and cocaine use disorders.

Internet Gaming Disorder

Proposed Criteria

Persistent and recurrent use of the Internet to engage in games, often with other players, leading to clinically significant impairment or distress as indicated by five (or more) of the following in a 12-month period:

1. Preoccupation with Internet games. (The individual thinks about previous gaming activity or anticipates playing the next game; Internet gaming becomes the dominant activity in daily life.)

   Note: This disorder is distinct from Internet gambling, which is included under gambling disorder.
2. Withdrawal symptoms when Internet gaming is taken away. (These symptoms are typically described as irritability, anxiety, or sadness, but there are no physical signs of pharmacological withdrawal.)
3. Tolerance—the need to spend increasing amounts of time engaged in Internet games.
4. Unsuccessful attempts to control the participation in Internet games.
5. Loss of interests in previous hobbies and entertainment as a result of, and with the exception of, Internet games.

6. Continued excessive use of Internet games despite knowledge of psychosocial problems.
7. Has deceived family members, therapists, or others regarding the amount of Internet gaming.
8. Use of Internet games to escape or relieve a negative mood (e.g., feelings of helplessness, guilt, anxiety).
9. Has jeopardized or lost a significant relationship, job, or educational or career opportunity because of participation in Internet games.

Note: Only nongambling Internet games are included in this disorder. Use of the Internet for required activities in a business or profession is not included; nor is the disorder intended to include other recreational or social Internet use. Similarly, sexual Internet sites are excluded.

Specify current severity:

Internet gaming disorder can be mild, moderate, or severe depending on the degree of disruption of normal activities. Individuals with less severe Internet gaming disorder may exhibit fewer symptoms and less disruption of their lives. Those with severe Internet gaming disorder will have more hours spent on the computer and more severe loss of relationships or career or school opportunities.

Gambling disorder is currently the only non-substance-related disorder included in the DSM-5 Section II chapter "Substance-Related and Addictive Disorders." However, there are other behavioral disorders that show some similarities to substance use disorders and gambling disorder for which the word *addiction* is commonly used in nonmedical settings, and the one condition with a considerable literature is the compulsive playing of Internet games. Internet gaming has been reportedly defined as an "addiction" by the Chinese government and is considered a public health threat in South Korea, where treatment and prevention systems have been set up. Reports of treatment of this condition have appeared in medical journals, mostly from Asian countries, but also in the United States and other high-income countries.

The DSM-5 work group reviewed more than 240 articles and found some behavioral similarities of Internet gaming to gambling disorder and to substance use disorders. The literature suffers, however, from lack of a standard definition from which to derive prevalence data. An understanding of the natural histories of cases, with or without treatment, is also missing. The literature does describe many underlying similarities to substance addictions, including aspects of tolerance, withdrawal, repeated unsuccessful attempts to cut back or quit, and impairment in normal functioning. Further, the seemingly high prevalence rates, both in Asian countries and in the West, justified inclusion of this disorder in Section III of DSM-5 and in the Mental, Behavioural, and Neurodevelopmental Disorders chapter in ICD-11. Note that since the publication of DSM-5, the number of clinical reports has continued to accumulate, but many of the issues remain unresolved.

Internet gaming disorder has achieved significant public health importance, and additional research may eventually lead to evidence that Internet gaming disorder (also commonly referred to as *Internet use disorder*, *Internet addiction*, or *gaming addiction*) has merit as an independent disorder. As with gambling disorder, there should be epidemiological studies to determine prevalence, clinical course, possible genetic influence, and potential biological factors based on, for example, brain imaging data.

Diagnostic Features

The essential feature of Internet gaming disorder is a pattern of excessive and prolonged participation in Internet gaming that results in a cluster of cognitive and behavioral symptoms, including progressive loss of control over gaming, tolerance, and withdrawal

symptoms, analogous to the symptoms of substance use disorders. These Internet-based games typically involve competition between groups of players who are often in different global regions, so that extended duration of play is encouraged by time-zone independence. Although Internet

gaming disorder most often involves specific Internet games with multiplayer competition, it can include non-Internet computerized off-line games as well, although these have been less researched. The Internet gaming often includes a significant aspect of social interactions during play, and the team aspects of play appear to be a key motivation. Attempts to direct the individual toward schoolwork or interpersonal activities are strongly resisted.

Individuals with Internet gaming disorder continue to sit at a computer and engage in gaming activities despite neglect of other activities. They typically devote 8–10 hours or more per day to this activity and at least 30 hours per week. If they are prevented from using a computer and returning to the game, they become agitated and angry. They often go for long periods without food or sleep. Normal obligations, such as school or work, or family obligations are neglected.

Until the optimal criteria and threshold for diagnosis are determined empirically, conservative definitions ought to be used, such that diagnoses are considered for endorsement of five or more of nine criteria.

Associated Features

Although no consistent personality types associated with Internet gaming disorder have been identified, negative affectivity, detachment, antagonism, disinhibition, and psychoticism have been associated with the disorder. Individuals with compulsive Internet gaming have demonstrated brain activation in specific regions triggered by exposure to the Internet game but not limited to reward system structures.

Prevalence

The mean prevalence of 12-month Internet gaming disorder is estimated as 4.7% across multiple countries, with a range of 0.7% to 15.6% across studies. Research using the DSM-5 proposed criteria suggests that prevalence is similar in Asian and Western countries. In the United States, based on large Internet-based surveys, the prevalence of DSM-5 Internet gaming disorder is 1% or lower. An international meta-analysis of 16 studies found a pooled prevalence of Internet gaming disorder among

adolescents of 4.6%, with adolescent boys/men generally reporting a higher prevalence rate (6.8%) than adolescent girls/women (1.3%).

Risk and Prognostic Factors

Environmental. Computer availability with Internet connection allows access to the types of games with which Internet gaming disorder is most often associated.

Genetic and physiological. Adolescent men seem to be at greatest risk of developing Internet gaming disorder.

Sex- and Gender-Related Diagnostic Issues

Internet gaming disorder appears to be more common in adolescent and young adult men than adolescent and young adult women. Adolescent boys ages 12–15 years also may be at greater risk of adverse effects of disordered gaming (e.g., lower school grades, loneliness). There may also be gender differences in the types of games played, in that adolescent girls ages 12–15 tend to choose games that include puzzles, music, and social and educational themes, whereas adolescent boys of the same age more often choose action, fighting, strategy, and role-playing games that may have greater addictive potential.

Association With Suicidal Thoughts or Behavior

Few studies specifically address suicide in individuals diagnosed with Internet gaming disorder, but studies on a broader phenotype of problematic Internet and online gaming behaviors are available. A nationally representative household survey of Australian youth ages 11–17 years (Young Minds Matter) found that problem Internet and online gaming behavior was associated with higher risk of suicide attempt in the prior year. After controlling for demographics, depression, family support, and self-esteem, a survey study of 9,510 Taiwanese students ages 12–18 years found that Internet addiction, including online gaming, was associated with suicidal thoughts and suicide attempt. In a representative sample of 8,807

students from randomly selected European schools, 3.62% had Internet gaming disorder (using DSM-5 criteria), and 3.11% of the students were considered to have pathological Internet use but were not gamers. Both groups showed similarly increased risks for emotional symptoms, conduct disorder, hyperactivity/inattention, self-injurious behaviors, and suicidal thoughts and behavior. The mental health effects of problematic Internet use, including suicidal thoughts or behavior, appear to be related to and perhaps mediated by the impact of problematic Internet use on sleep.

Functional Consequences of Internet Gaming Disorder

Internet gaming disorder may lead to school failure, job loss, or marriage failure. The compulsive gaming behavior tends to crowd out normal social, scholastic, and family activities. Students may show declining grades and eventually failure in school. Family responsibilities may be neglected.

Differential Diagnosis

Excessive use of the Internet not involving playing of online games (e.g., excessive use of social media, such as Facebook; viewing pornography online) is not considered analogous to Internet gaming disorder, and future research on other excessive uses of the Internet would need to follow similar guidelines as suggested herein. Excessive gambling online may qualify for a separate diagnosis of gambling disorder.

Comorbidity

Health may be neglected due to compulsive gaming. Other diagnoses that may be associated with Internet gaming disorder include major depressive disorder, ADHD, and obsessive-compulsive disorder.

Neurobehavioral Disorder Associated With Prenatal Alcohol Exposure

Proposed Criteria

A. More than minimal exposure to alcohol during gestation, including prior to pregnancy recognition. Confirmation of gestational exposure to alcohol may be obtained from maternal self-report of alcohol use in pregnancy, medical or other records, or clinical observation.

B. Impaired neurocognitive functioning as manifested by one or more of the following:

   1. Impairment in global intellectual performance (i.e., IQ of 70 or below, or a standard score of 70 or below on a comprehensive developmental assessment).
   2. Impairment in executive functioning (e.g., poor planning and organization; inflexibility; difficulty with behavioral inhibition).

   3. Impairment in learning (e.g., lower academic achievement than expected for intellectual level; specific learning disability).
   4. Memory impairment (e.g., problems remembering information learned recently; repeatedly making the same mistakes; difficulty remembering lengthy verbal instructions).
   5. Impairment in visual-spatial reasoning (e.g., disorganized or poorly planned drawings or constructions; problems differentiating left from right).

C. Impaired self-regulation as manifested by one or more of the following:

   1. Impairment in mood or behavioral regulation (e.g., mood lability; negative affect or irritability; frequent behavioral outbursts).
   2. Attention deficit (e.g., difficulty shifting attention; difficulty sustaining mental effort).

3. Impairment in impulse control (e.g., difficulty waiting turn; difficulty complying with rules).

D. Impairment in adaptive functioning as manifested by two or more of the following, one of which must be (1) or (2):
   1. Communication deficit (e.g., delayed acquisition of language; difficulty understanding spoken language).
   2. Impairment in social communication and interaction (e.g., overly friendly with strangers; difficulty reading social cues; difficulty understanding social consequences).
   3. Impairment in daily living skills (e.g., delayed toileting, feeding, or bathing; difficulty managing daily schedule).
   4. Impairment in motor skills (e.g., poor fine motor development; delayed attainment of gross motor milestones or ongoing deficits in gross motor function; deficits in coordination and balance).

E. Onset of the disorder (symptoms in Criteria B, C, and D) occurs in childhood.

F. The disturbance causes clinically significant distress or impairment in social, academic, occupational, or other important areas of functioning.

G. The disorder is not better explained by the direct physiological effects associated with postnatal use of a substance (e.g., a medication, alcohol or other drugs), a general medical condition (e.g., traumatic brain injury, delirium, dementia), another known teratogen (e.g., fetal hydantoin syndrome), a genetic condition (e.g., Williams syndrome, Down syndrome, Cornelia de Lange syndrome), or environmental neglect.

Alcohol is a neurobehavioral teratogen, and prenatal alcohol exposure has teratogenic effects on central nervous system (CNS) development and subsequent function. *Neurobehavioral disorder associated with prenatal alcohol exposure* (ND-PAE) is a new clarifying term, intended to

encompass the full range of developmental disabilities associated with exposure to alcohol in utero. ND-PAE may be diagnosed both in the absence and in the presence of the physical effects of prenatal alcohol exposure (e.g., facial dysmorphology required for a diagnosis of fetal alcohol syndrome).

Diagnostic Features

The essential features of ND-PAE are the manifestation of impairment in neurocognitive, behavioral, and adaptive functioning associated with prenatal alcohol exposure. Impairment can be documented based on past diagnostic evaluations (e.g., psychological or educational assessments) or medical records, reports by the individual or informants, and/or observation by a clinician.

A clinical diagnosis of fetal alcohol syndrome, including specific prenatal alcohol-related facial dysmorphology and growth retardation, can be used as evidence of

significant levels of prenatal alcohol exposure; specific guidelines for facial dysmorphology have been developed for diverse ethnoracial physiognomies. Although both animal and human studies have documented adverse effects of lower levels of drinking, identifying how much prenatal exposure is needed to significantly impact neurodevelopmental outcome remains challenging. Data suggest that a history of more than minimal gestational exposure prior to pregnancy recognition and/or following pregnancy recognition may be required. More than minimal exposure is defined as greater than 13 drinks per month during pregnancy or more than 2 drinks on any one occasion. Identifying a minimal threshold of drinking during pregnancy will require consideration of a variety of factors known to affect exposure and/or interact to influence developmental outcomes, including stage of prenatal development, gestational smoking, maternal and fetal genetics, and maternal physical status (i.e., age, health, and certain obstetric problems).

Symptoms of ND-PAE include marked impairment in global intellectual performance (IQ) or neurocognitive impairments in any of the following areas: executive functioning, learning, memory, and/or visual-spatial reasoning. Impairments in self-regulation are present and may include impairment in mood or behavioral regulation, attention deficit, or impairment in impulse control. Finally, impairments in adaptive functioning include communication deficits and impairment in social communication and interaction. Impairment in daily living (self-help) skills and impairment in motor skills may be present. As it may be difficult to obtain an accurate assessment of the neurocognitive abilities of very young children, it is appropriate to defer a diagnosis for children 3 years of age and younger.

Associated Features

Associated features vary depending on age, degree of alcohol exposure, and the individual's environment. An individual can be diagnosed with this disorder regardless of socioeconomic or cultural background. However, ongoing parental alcohol/substance misuse, parental mental illness, exposure to domestic or community violence, neglect or abuse, disrupted caregiving relationships, multiple out-of-home placements, and lack of continuity in medical or mental health care are often present.

Prevalence

In the United States, the prevalence of ND-PAE (encompassing fetal alcohol spectrum disorders) has been estimated as 15.2/1,000 (range: 11.3–50.0/1,000), with higher estimates derived when only children with full evaluations were included (31.1–98.5/1,000). When vulnerable subpopulations are considered, rates of ND-PAE can be much higher (e.g., among children in care settings, 251.5/1,000), according to a meta-analysis of data from multiple countries. In 2012, the mean global prevalence of fetal alcohol spectrum disorder in the general population was 7.7 per 1,000 individuals, with a prevalence of 8.8 per 1,000 in the region of the Americas (including the United States).

Development and Course

Among individuals with prenatal alcohol exposure, evidence of CNS dysfunction varies according to developmental stage. Although about one-half of young children prenatally exposed to alcohol show marked developmental delay in the first 3 years of life, other children affected by prenatal alcohol exposure may not exhibit signs of CNS dysfunction until they are preschool- or school-age. Additionally, impairments in higher order cognitive processes (i.e., executive functioning), which are often associated with prenatal alcohol exposure, may be more easily assessed in older children. When children reach school age, learning difficulties, impairment in executive function, and problems with integrative language functions usually emerge more clearly, and both social skills deficits and challenging behavior may become more evident. In particular, as school and other requirements

become more complex, greater deficits are noted. Because of this, the school years represent the ages at which a diagnosis of ND-PAE would be most likely.

Risk and Prognostic Factors

Environmental. Low socioeconomic status and low educational level in the mother are risk factors for fetal alcohol syndrome. This association is related to social, structural, and psychological factors that may increase the risk of maternal drinking or worsen its impact, including social determinants of health, such as the high concentration of liquor stores in low-income, ethnoracially segregated communities.

Culture-Related Diagnostic Issues

Socioeconomic and cultural factors affect the consumption of alcohol during pregnancy, which ranges globally from 0.2% in the Eastern Mediterranean region to 25.2% in the European region. Individuals belonging to ethnic groups that have higher proportions of certain alleles of alcohol-metabolizing enzymes (e.g., of aldehyde dehydrogenase 2) may be less likely to exhibit the effects of prenatal alcohol exposure.

Association With Suicidal Thoughts or Behavior

Suicide is a high-risk outcome, with rates increasing significantly in late adolescence and early adulthood. Analyses of the Canadian national fetal alcohol spectrum disorder (FASD) database show that among individuals with FASD who have impaired affect regulation, there is a markedly higher risk of suicidal thoughts or behavior. In an Alberta-based registry, it was found that individuals with fetal alcohol syndrome are at markedly increased risk for premature death, with 15% dying from suicide. In California, a study of 54 adolescents ages 13–18 years with FASD also demonstrated markedly higher rates of suicidal thoughts and serious attempts (all by boys) compared with the general U.S. adolescent population. In a Canadian survey, the mothers of individuals with FASD were over six times as likely to die by suicide and almost five times more likely to attempt suicide after giving birth to a child with FASD compared with mothers whose child did not have FASD, suggesting that the increased rates of suicidal ideation and suicide attempts among youth with FASD may be mediated by family factors (genetic and/or environmental), in addition to any risk conferred by the FASD condition itself.

Functional Consequences of Neurobehavioral Disorder Associated With Prenatal Alcohol Exposure

The CNS dysfunction seen in individuals with ND-PAE often leads to decrements in adaptive behavior and to maladaptive behavior with lifelong consequences. Abnormalities have been associated with ND-PAE in multiple organ systems, including the heart, kidney, liver, gastrointestinal tract, and endocrine systems. Individuals affected by prenatal alcohol exposure have a higher prevalence of disrupted school experiences, poor employment records, trouble with the law, confinement (legal or psychiatric), and dependent living conditions.

Differential Diagnosis

Other considerations include maternal exposure to other substances during the prenatal period; poor prenatal care; the physiological effects of postnatal substance use, such as a medication, alcohol, or other substances; disorders

due to another medical condition, such as traumatic brain injury or other neurocognitive disorders (e.g., delirium, major neurocognitive disorder [dementia]); and environmental neglect.

Genetic conditions such as Williams syndrome, Down syndrome, or Cornelia de Lange syndrome and other teratogenic conditions such as fetal hydantoin syndrome and

maternal phenylketonuria may have similar physical and behavioral characteristics. A careful review of prenatal exposure history is needed to clarify the teratogenic agent, and an evaluation by a clinical geneticist may be needed to distinguish physical characteristics associated with these and other genetic conditions.

Comorbidity

Mental health problems have been identified in more than 90% of individuals with histories of significant prenatal alcohol exposure. The most common co-occurring diagnosis is attention-deficit/hyperactivity disorder, but research has shown that individuals with ND-PAE differ in neuropsychological characteristics and in their responsiveness to pharmacological interventions. Other high- probability co-occurring disorders include oppositional defiant disorder and conduct disorder, but the appropriateness of these diagnoses should be weighed in the context of the significant impairments in general intellectual and executive functioning that are often associated with prenatal alcohol exposure. Mood symptoms, including symptoms of bipolar disorder and depressive disorders, have been described. History of prenatal alcohol exposure is associated with an increased risk for later tobacco, alcohol, and other substance use disorders.

Suicidal Behavior Disorder

Proposed Criteria

A. Within the last 24 months, the individual has made a suicide attempt.

   Note: A suicide attempt is a self-initiated sequence of behaviors by an individual who, at the time of initiation, expected that the set of actions would lead to his or her own death. (The "time of initiation" is the time when a behavior took place that involved applying the method.)

B. The act does not meet criteria for nonsuicidal self-injury—that is, it does not involve self-injury directed to the surface of the body undertaken to induce relief from a negative feeling/cognitive state or to achieve a positive mood state.

C. The diagnosis is not applied to suicidal ideation or to preparatory acts.

D. The act was not initiated during a state of delirium or confusion.

E. The act was not undertaken solely for a political or religious objective.

Specify if:

Current: Not more than 12 months since the last attempt.

In early remission: 12–24 months since the last attempt.

Note: ICD-10-CM codes to indicate whether suicidal behavior is part of the current clinical presentation (**T14.91A** for initial encounter and **T14.91D** for subsequent encounters) and/or whether there has been a prior history of suicidal behavior **(Z91.51)** are available for clinical use to accompany any DSM-5 diagnosis; in addition, the codes can be recorded in the absence of a DSM-5 diagnosis. The definition of these codes is included in Section II, "Other Conditions That May Be a Focus of Clinical Attention" (see "Suicidal Behavior").

Specifiers

Suicidal behavior is often categorized in terms of violence of the method. Generally, overdoses with legal or illegal substances are considered

nonviolent in method, whereas jumping, gunshot wounds, and other methods are considered violent. Another dimension for classification is medical consequences of the behavior, with high-lethality attempts being defined as those requiring medical hospitalization beyond a visit to an emergency

department. An additional dimension considered includes the degree of planning versus impulsiveness of the attempt, a characteristic that might have consequences for the medical outcome of a suicide attempt.

If the suicidal behavior occurred 12–24 months prior to evaluation, the condition is considered to be in early remission.

Diagnostic Features

The essential manifestation of suicidal behavior disorder is a suicide attempt. A *suicide attempt* is a behavior that the individual has undertaken with at least some intent to die. The behavior might or might not lead to injury or serious medical consequences. Several factors can influence the medical consequences of the suicide attempt, including poor planning, lack of knowledge about the lethality of the method chosen, low intentionality or ambivalence, or chance intervention by others after the behavior has been initiated. These should not be considered in assigning the diagnosis.

Determining the degree of intent can be challenging. Individuals might not acknowledge intent, especially in situations where doing so could result in hospitalization or cause distress to loved ones. Markers of risk include degree of planning, including selection of a time and place to minimize rescue or interruption; the individual’s mental state at the time of the behavior, with acute agitation being especially concerning; recent discharge from inpatient care; or recent discontinuation of a mood stabilizer such as lithium or an antipsychotic such as clozapine in the case of schizophrenia. Examples of environmental “triggers” include recently learning of a potentially fatal medical diagnosis such as cancer, experiencing the sudden and unexpected loss of a close relative or partner, loss of employment, or displacement from housing. Conversely, features such as talking to others

about future events or preparedness to sign a contract for safety are less reliable indicators.

In order for the criteria to be met, the individual must have made at least one suicide attempt. Suicide attempts can include behaviors in which, after initiating the suicide attempt, the individual changed his or her mind or someone intervened. For example, an individual might intend to ingest a given amount of medication or poison, but either stop or be stopped by another before ingesting the full amount. If the individual is dissuaded by another or changes his or her mind before initiating the behavior, the diagnosis should not be made. The acts qualifying for a diagnosis of suicidal behavior disorder should not have been initiated exclusively during a state of delirium or confusion. If the individual deliberately became intoxicated before initiating the suicidal behavior in order to reduce anticipatory anxiety and to minimize interference with the intended behavior, the diagnosis can still be made.

Currently there are no clinical instruments that yield positive predictive values sufficient to make them useful tools for predicting suicidal behavior at the patient level. It is not surprising that single clinical or biological factors are poor indicators of suicide risk, because suicidal behavior emerges from a convergence of multiple risk factors. Moreover, given the clinical heterogeneity of suicidal behavior, it is likely that there are multiple pathways to suicidal behavior that can only be captured if this heterogeneity is considered. Similarly, numerous biomarkers have been studied, but no robust predictor has emerged.

Development and Course

Suicidal behavior disorder can occur at any time in the life span but is rarely seen in children under the age of 5. Approximately 25%–30% of persons who attempt suicide will go on to make more attempts. There is significant variability in terms of frequency, method, and lethality of attempts. However, this is not different from what is observed in other illnesses, such as major depressive disorder, in which frequency of episode, subtype of episode, and impairment for a given episode can vary significantly.

Risk and Prognostic Factors

Genetic and physiological. The largest genome-wide association study of suicide attempt to date, from the Psychiatric Genomics Consortium, found that the genetic risk for depression increases the risk for suicide attempt across diagnostic cohorts with major depressive disorder, bipolar disorder, and schizophrenia. In other words, across diagnostic categories, attempters carry more risk alleles for depression than nonattempters, rather than simply for their primary psychiatric diagnosis. These results suggest that the genetic associations with suicide attempt are partly unique and partly shared with the genetic associations with depression.

Culture-Related Diagnostic Issues

Cultural contexts affect the frequency and form of suicidal behavior disorder, including variations in incidence and prevalence, methods used (e.g., poisoning with pesticides in low-income countries; gunshot wounds in the southwestern United States), motivations, circumstances, and meanings. These patterns vary over time, by migrant or ethnic group, and by service setting. Culturally mediated social stressors and predicaments such as family breakdowns, perceived loss of dignity or interpersonal status, conflicting intergenerational roles and expectations due to differential acculturation, changing levels of sociocultural integration, stigma and self-stigma about suicide, and systemic discrimination and structural inequity (institutionalized socioeconomic bias and oppression) may contribute to the risk of suicidal behavior disorder. Attitudes toward suicide and suicidal behaviors are influenced by historical, environmental, economic, political, legal, social, cultural, moral, and spiritual or religious factors. For example, in a longitudinal U.S. sample followed across generations, parental belief (self-identified as mostly Protestant and Catholic) in the importance of religion was associated with lower risk of suicidal behavior in their offspring, independent of an offspring's own belief about religious importance and other known parental factors, such as parental depression, suicidal behavior, and divorce. The reasons for suicide attempts and choice of suicide methods may have cultural significance, which may be associated

with specific individual and social responses (e.g., of stigma, shame, or respect).

Sex- and Gender-Related Diagnostic Issues

Suicidal behavior disorder varies in prevalence and form across sex and gender. On average, suicides are about twice as common in men compared with women, although the prevalence ratio varies by country and cultural context. Estimates also vary because the intent of self-harm behaviors is not always clearly measured; however, suicidal behavior that does not result in death is more common in women than in men. Men generally use more lethal methods such as gunshots and hanging, whereas less lethal means such as self-poisoning are more common in women. The frequency of suicidal behaviors is higher in women (i.e., the average number of suicide attempts for a woman is generally higher than the average number for a man), but this could be explained by the more frequent use of less lethal methods among women. Suicide rates among individuals who identify as transgender are high, and transgender individuals are also at higher risk for suicidal behavior than cisgender individuals.

Diagnostic Markers

Laboratory abnormalities consequent to the suicidal attempt are often evident. Suicidal behavior that leads to blood loss can be accompanied by anemia, hypotension, or shock. Overdoses might lead to coma or obtundation and associated laboratory abnormalities such as electrolyte imbalances.

Comorbidity

Suicidal behavior disorder is seen in the context of a variety of mental disorders, most commonly bipolar disorder, major depressive disorder, schizophrenia, schizoaffective disorder, anxiety disorders (in particular, panic disorders associated with catastrophic content and PTSD flashbacks), substance use disorders (especially alcohol use disorders), borderline

personality disorder, antisocial personality disorder, eating disorders, and adjustment disorders.

Nonsuicidal Self-Injury Disorder

Proposed Criteria

A. In the last year, the individual has, on 5 or more days, engaged in intentional self-inflicted damage to the surface of his or her body of a sort likely to induce bleeding, bruising, or pain (e.g., cutting, burning, stabbing, hitting, excessive rubbing), with the expectation that the injury will lead to only minor or moderate physical harm (i.e., there is no suicidal intent).

   Note: The absence of suicidal intent has either been stated by the individual or can be inferred by the individual's repeated engagement in a behavior that the individual knows, or has learned, is not likely to result in death.

B. The individual engages in the self-injurious behavior with one or more of the following expectations:

   1. To obtain relief from a negative feeling or cognitive state.
   2. To resolve an interpersonal difficulty.
   3. To induce a positive feeling state.

   Note: The desired relief or response is experienced during or shortly after the self-injury, and the individual may display patterns of behavior suggesting a dependence on repeatedly engaging in it.

C. The intentional self-injury is associated with at least one of the following:

   1. Interpersonal difficulties or negative feelings or thoughts, such as depression, anxiety, tension, anger, generalized

distress, or self-criticism, occurring in the period immediately prior to the self-injurious act.

2. Prior to engaging in the act, a period of preoccupation with the intended behavior that is difficult to control.
3. Thinking about self-injury that occurs frequently, even when it is not acted upon.

D. The behavior is not socially sanctioned (e.g., body piercing, tattooing, part of a religious or cultural ritual) and is not restricted to picking a scab or nail biting.

E. The behavior or its consequences cause clinically significant distress or interference in interpersonal, academic, or other important areas of functioning.

F. The behavior does not occur exclusively during psychotic episodes, delirium, substance intoxication, or substance withdrawal. In individuals with a neurodevelopmental disorder, the behavior is not part of a pattern of repetitive stereotypies. The behavior is not better explained by another mental disorder or medical condition (e.g., psychotic disorder, autism spectrum disorder, intellectual developmental disorder [intellectual disability], Lesch-Nyhan syndrome, stereotypic movement disorder with self-injury, trichotillomania [hair-pulling disorder], excoriation [skin-picking] disorder).

Note: ICD-10-CM codes to indicate whether nonsuicidal self-injury is part of the current clinical presentation **(R45.88)** and/or whether there has been a prior history of nonsuicidal

self-injury **(Z91.52)** are available for clinical use to accompany any DSM-5 diagnosis; in addition, the codes can be recorded in the absence of a DSM-5 diagnosis. The definition of these codes is included in Section II, “Other Conditions That May Be a Focus of Clinical Attention” (see “Nonsuicidal Self-Injury”).

Diagnostic Features

The essential feature of nonsuicidal self-injury disorder is that the individual repeatedly inflicts minor-to-moderate, often painful injuries to the surface of his or her body without suicidal intent. Most commonly, the purpose is to reduce negative emotions, such as tension, anxiety, sadness, or self-reproach, or less often to resolve an interpersonal difficulty. In some cases, the injury is conceived of as a deserved self-punishment. The individual will often report an immediate sensation of relief that occurs during the process. When the behavior occurs frequently, it might be associated with a sense of urgency and craving, the resultant behavioral pattern resembling an addiction. The inflicted wounds can become deeper and more numerous.

Cutting is the most common method of injury and is most often inflicted with a knife, needle, razor, or other sharp object. Common areas for injury include the dorsal side of the forearm and frontal area of the thighs. A single session of injury might involve a series of superficial, parallel cuts—separated by 1 or 2 centimeters—on a visible or accessible location. The resulting cuts will often bleed and will often leave a characteristic pattern of scars.

Other relatively common methods used include superficial scratching or burning of the skin, as well as self-hitting or banging, biting, and interfering with wound healing. Many will use different methods over time, and use of multiple methods is associated with more severe psychopathology, including engagement in suicide attempts.

Many, and possibly most, of those who engage in nonsuicidal self-injury do not seek clinical attention. This tendency may reflect a reluctance to disclose self-injury due to concerns over stigma. In addition, many individuals who engage in these behaviors experience them positively because of the effectiveness of nonsuicidal self-injury in regulating negative emotion, thereby reducing or eliminating motivation for treatment. Children and adolescents might experiment with these behaviors but not experience relief. In such cases, youths often report that the procedure is painful or distressing and might then discontinue the practice.

Associated Features

Nonsuicidal self-injury disorder appears predominantly maintained by negative reinforcement, in that the behavior is reported to quickly reduce negative emotion and aversive emotional arousal. Some who engage in the behavior also report that nonsuicidal self-injury can quickly reduce unwanted dissociative experiences and even suicidal ideation, as well as serve as a way to cope with trauma-related symptoms such as self-directed anger and/or disgust. However, other forms of social and emotional reinforcement can also sustain the behavior, such as a desire to elicit reactions from others or generate positive feelings.

Prevalence

In an international meta-analysis, prevalence of nonsuicidal self-injury disorder was found overall to be modestly higher in girls/women than in boys/men. This is in contrast to suicidal behavior, in which the gender ratio of girls/women to boys/men is much higher. The gender difference for nonsuicidal self-injury disorder is more pronounced in clinical samples. Across cultural contexts, the gender ratio of nonsuicidal self-injury may vary, being more prevalent among girls/women in some contexts (e.g., among high school students in rural areas of China) and among boys/men in others (e.g., among youth ages

11–19 in Jordan). Nonsuicidal self-injury disorder is substantially more common among sexual minorities, especially those who identify as bisexual.

Development and Course

Nonsuicidal self-injury disorder most often starts in the early to mid-teen years and can continue for many years, with earlier ages at onset being associated with more severe manifestations. Nonsuicidal self-injury disorder may peak in late adolescence and the early 20s and then decline into adulthood. Additional prospective research is needed to outline the natural history of nonsuicidal self-injury disorder and the factors that promote or inhibit its course. Individuals often learn of the behavior on the

recommendation or observation of another, through media outlets, and through social media. Individuals exposed to others who self-injure, including in inpatient, school, correctional, and community settings, are more likely to initiate self-injury, potentially through social modeling or social learning mechanisms.

Culture-Related Diagnostic Issues

Nonsuicidal self-injury disorder should not be diagnosed if the behavior is motivated by a widely accepted cultural practice. This is true even if the practice is only carried out by a minority of the population (e.g., engaging in self-flagellation as a collective activity during religious festivals). Nonsuicidal self-injury may be a way of expressing group belongingness rather than individual distress or emotion regulation, as suggested by research with “alternative” (i.e., Goth, Emo, and Punk) youth groups in Germany, and nonsuicidal self-injury disorder should also not be diagnosed in such instances.

Association With Suicidal Thoughts or Behavior

Because individuals with nonsuicidal self-injury can and do attempt suicide, it is important to evaluate these individuals for suicide risk and to obtain information from a third party concerning any recent change in stress exposure and mood. Likelihood of a suicide attempt has been associated with a history of nonsuicidal self-injury, with the onset of nonsuicidal self-injury typically preceding suicide attempts by approximately 1–2 years, as shown by research in clinical and community settings in three high-income countries. The use of multiple previous methods of nonsuicidal self-injury, high frequencies of self-injurious acts, younger age at onset, and using nonsuicidal self-injury to obtain relief from internal distress or for self-punishment are strongly predictive of both suicidal ideation and suicide attempts.

Functional Consequences of Nonsuicidal Self-Injury Disorder

The act of cutting might be performed with shared implements, raising the possibility of blood-borne disease transmission. Severe burns, infection from poor care of injuries, and permanent scarring can also result, negatively impacting the individual.

Differential Diagnosis

Borderline personality disorder. Many have regarded nonsuicidal self-injury as pathognomonic of borderline personality disorder. However, although nonsuicidal self-injury disorder is often comorbid with borderline personality disorder, many individuals with nonsuicidal self-injury disorder do not have a personality pattern that meets criteria for borderline personality disorder. Nonsuicidal self-injury disorder not only occurs without borderline personality disorder but frequently co-occurs with many other disorders, including depressive disorders, eating disorders, and substance disorders.

Suicidal behavior. The differentiation between nonsuicidal self-injury disorder and suicidal behavior is based on the stated goal of the behavior, either as a wish to die (suicidal behavior) or to experience relief (as described in the criteria for nonsuicidal self-injury disorder). In contrast to suicidal behavior, nonsuicidal self-injury episodes are, in the short-term, typically benign in individuals with a history of frequent episodes. Further, some individuals report using their nonsuicidal self-injury to avoid attempting suicide.

Trichotillomania (hair-pulling disorder). Trichotillomania is defined by self-injurious behavior confined to pulling out one's own hair, most commonly from the scalp, eyebrows, or eyelashes. The behavior occurs in "sessions" that can last for hours. It is most likely to occur during a period of relaxation or distraction. If the self-injurious behavior is confined to hair-pulling, trichotillomania should be diagnosed instead of nonsuicidal self-injury disorder.

Stereotypic movement disorder. Stereotypic movement disorder involves repetitive, seemingly driven, and apparently purposeless motor behavior

(e.g., hand shaking or waving, body rocking, head banging, self-biting, hitting own body) that can sometimes result in self-injury and is often associated with a known medical or genetic condition, neurodevelopmental disorder, or environmental factor (e.g., Lesch-Nyhan syndrome, intellectual developmental disorder, intrauterine alcohol exposure). If the self-injurious behavior meets criteria for stereotypic movement disorder, it should be diagnosed instead of nonsuicidal self-injury disorder.

Excoriation (skin-picking) disorder. Excoriation disorder is usually directed to picking at an area of the skin that the individual feels is unsightly or a blemish, usually on the face or the scalp. If the self-injurious behavior is confined to skin-picking, excoriation disorder should be diagnosed instead of nonsuicidal self-injury disorder.

APPENDIX

Alphabetical Listing of DSM-5-TR Diagnoses and ICD-10-CM Codes

Numerical Listing of DSM-5-TR Diagnoses and ICD-10-CM Codes

DSM-5 Advisors and Other Contributors

Alphabetical Listing of DSM-5-TR Diagnoses and ICD-10-CM Codes

For periodic DSM-5-TR coding and other updates, see www.dsm5.org.

ICD-10-CM	Disorder, condition, or problem
Z60.3	Acculturation difficulty
F43.0	Acute stress disorder
	Adjustment disorders
F43.22	With anxiety
F43.21	With depressed mood
F43.24	With disturbance of conduct
F43.23	With mixed anxiety and depressed mood
F43.25	With mixed disturbance of emotions and conduct
F43.20	Unspecified
Z72.811	Adult antisocial behavior
F98.5	Adult-onset fluency disorder
	Adult physical abuse by nonspouse or nonpartner, Confirmed
T74.11XA	Initial encounter
T74.11XD	Subsequent encounter
	Adult physical abuse by nonspouse or nonpartner, Suspected
T76.11XA	Initial encounter
T76.11XD	Subsequent encounter
	Adult psychological abuse by nonspouse or nonpartner, Confirmed
T74.31XA	Initial encounter
T74.31XD	Subsequent encounter
	Adult psychological abuse by nonspouse or nonpartner, Suspected
T76.31XA	Initial encounter
T76.31XD	Subsequent encounter

ICD-10-CM	Disorder, condition, or problem
	Adult sexual abuse by nonspouse or nonpartner, Confirmed
T74.21XA	Initial encounter
T74.21XD	Subsequent encounter
	Adult sexual abuse by nonspouse or nonpartner, Suspected
T76.21XA	Initial encounter
T76.21XD	Subsequent encounter
R41.81	Age-related cognitive decline
F40.00	Agoraphobia

	Alcohol-induced anxiety disorder
F10.180	With mild use disorder
F10.280	With moderate or severe use disorder
F10.980	Without use disorder
	Alcohol-induced bipolar and related disorder
F10.14	With mild use disorder
F10.24	With moderate or severe use disorder
F10.94	Without use disorder
	Alcohol-induced depressive disorder
F10.14	With mild use disorder
F10.24	With moderate or severe use disorder
F10.94	Without use disorder
	Alcohol-induced major neurocognitive disorder, Amnestic-confabulatory type
F10.26	With moderate or severe use disorder
F10.96	Without use disorder
	Alcohol-induced major neurocognitive disorder, Nonamnestic-confabulatory type
F10.27	With moderate or severe use disorder
F10.97	Without use disorder
	Alcohol-induced mild neurocognitive disorder
F10.188	With mild use disorder
F10.288	With moderate or severe use disorder
F10.988	Without use disorder
	Alcohol-induced psychotic disorder

ICD-10-CM	Disorder, condition, or problem
F10.159	With mild use disorder
F10.259	With moderate or severe use disorder
F10.959	Without use disorder
	Alcohol-induced sexual dysfunction
F10.181	With mild use disorder
F10.281	With moderate or severe use disorder
F10.981	Without use disorder
	Alcohol-induced sleep disorder
F10.182	With mild use disorder
F10.282	With moderate or severe use disorder
F10.982	Without use disorder
	Alcohol intoxication
F10.120	With mild use disorder
F10.220	With moderate or severe use disorder
F10.920	Without use disorder
	Alcohol intoxication delirium
F10.121	With mild use disorder
F10.221	With moderate or severe use disorder
F10.921	Without use disorder
	Alcohol use disorder

F10.10	Mild
F10.11	In early remission
F10.11	In sustained remission
F10.20	Moderate
F10.21	In early remission
F10.21	In sustained remission
F10.20	Severe
F10.21	In early remission
F10.21	In sustained remission
	Alcohol withdrawal, With perceptual disturbances
F10.132	With mild use disorder

F10.232 With moderate or severe use disorder

F10.932 Without use disorder

Alcohol withdrawal, Without perceptual disturbances

F10.130 With mild use disorder

F10.230 With moderate or severe use disorder

F10.930 Without use disorder

Alcohol withdrawal delirium

F10.131 With mild use disorder

F10.231 With moderate or severe use disorder

F10.931 Without use disorder

F15.921 Amphetamine-type (or other stimulant) medication–induced delirium (amphetamine-type or other stimulant medication taken as prescribed)

Amphetamine-type substance (or other stimulant)–induced anxiety disorder

F15.180 With mild use disorder

F15.280 With moderate or severe use disorder

F15.980 Without use disorder

Amphetamine-type substance (or other stimulant)–induced bipolar and related disorder

F15.14 With mild use disorder

F15.24 With moderate or severe use disorder

F15.94 Without use disorder

Amphetamine-type substance (or other stimulant)–induced depressive disorder

F15.14 With mild use disorder

F15.24 With moderate or severe use disorder

F15.94 Without use disorder

Amphetamine-type substance (or other stimulant)–induced mild neurocognitive disorder

F15.188 With mild use disorder

F15.288 With moderate or severe use disorder

F15.988 Without use disorder

Amphetamine-type substance (or other stimulant)–induced obsessive-compulsive and related disorder

F15.188 With mild use disorder

F15.288 With moderate or severe use disorder

F15.988 Without use disorder

	Amphetamine-type substance (or other stimulant)–induced psychotic disorder
F15.159	With mild use disorder
F15.259	With moderate or severe use disorder
F15.959	Without use disorder
	Amphetamine-type substance (or other stimulant)–induced sexual dysfunction
F15.181	With mild use disorder
F15.281	With moderate or severe use disorder
F15.981	Without use disorder
	Amphetamine-type substance (or other stimulant)–induced sleep disorder
F15.182	With mild use disorder
F15.282	With moderate or severe use disorder
F15.982	Without use disorder
	Amphetamine-type substance intoxication
	Amphetamine-type substance intoxication, With perceptual disturbances
F15.122	With mild use disorder
F15.222	With moderate or severe use disorder
F15.922	Without use disorder
	Amphetamine-type substance intoxication, Without perceptual disturbances
F15.120	With mild use disorder
F15.220	With moderate or severe use disorder
F15.920	Without use disorder
	Amphetamine-type substance (or other stimulant) intoxication delirium
F15.121	With mild use disorder
F15.221	With moderate or severe use disorder
F15.921	Without use disorder
	Amphetamine-type substance use disorder
F15.10	Mild
F15.11	In early remission
F15.11	In sustained remission
F15.20	Moderate
F15.21	In early remission
F15.21	In sustained remission
F15.20	Severe
F15.21	In early remission
F15.21	In sustained remission
	Amphetamine-type substance withdrawal
F15.13	With mild use disorder

F15.23	With moderate or severe use disorder
F15.93	Without use disorder

	Anorexia nervosa
F50.02	Binge-eating/purging type
F50.01	Restricting type
	Antidepressant discontinuation syndrome
T43.205A	Initial encounter
T43.205S	Sequelae
T43.205D	Subsequent encounter
G21.11	Antipsychotic medication– and other dopamine receptor blocking agent–induced parkinsonism
F60.2	Antisocial personality disorder
F06.4	Anxiety disorder due to another medical condition
	Attention-deficit/hyperactivity disorder
F90.2	Combined presentation
F90.1	Predominantly hyperactive/impulsive presentation
F90.0	Predominantly inattentive presentation
F84.0	Autism spectrum disorder
F60.6	Avoidant personality disorder
F50.82	Avoidant/restrictive food intake disorder
F50.81	Binge-eating disorder
	Bipolar I disorder, Current or most recent episode depressed
F31.76	In full remission
F31.75	In partial remission
F31.31	Mild
F31.32	Moderate
F31.4	Severe
F31.5	With psychotic features
F31.9	Unspecified
F31.0	Bipolar I disorder, Current or most recent episode hypomanic
F31.72	In full remission
F31.71	In partial remission
F31.9	Unspecified
	Bipolar I disorder, Current or most recent episode manic

F31.74	In full remission
F31.73	In partial remission
F31.11	Mild
F31.12	Moderate
F31.13	Severe
F31.2	With psychotic features
F31.9	Unspecified
F31.9	Bipolar I disorder, Current or most recent episode unspecified
F31.81	Bipolar II disorder
	Bipolar and related disorder due to another medical condition
F06.33	With manic features
F06.33	With manic- or hypomanic-like episode
F06.34	With mixed features

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F45.22	Body dysmorphic disorder
R41.83	Borderline intellectual functioning
F60.3	Borderline personality disorder
F23	Brief psychotic disorder
F50.2	Bulimia nervosa
F15.980	Caffeine-induced anxiety disorder, Without use disorder
F15.982	Caffeine-induced sleep disorder, Without use disorder
F15.920	Caffeine intoxication
F15.93	Caffeine withdrawal
	Cannabis-induced anxiety disorder
F12.180	With mild use disorder
F12.280	With moderate or severe use disorder
F12.980	Without use disorder
	Cannabis-induced psychotic disorder
F12.159	With mild use disorder
F12.259	With moderate or severe use disorder
F12.959	Without use disorder
	Cannabis-induced sleep disorder
F12.188	With mild use disorder
F12.288	With moderate or severe use disorder
F12.988	Without use disorder

	Cannabis intoxication, With perceptual disturbances
F12.122	With mild use disorder
F12.222	With moderate or severe use disorder
F12.922	Without use disorder
	Cannabis intoxication, Without perceptual disturbances
F12.120	With mild use disorder
F12.220	With moderate or severe use disorder
F12.920	Without use disorder
	Cannabis intoxication delirium
F12.121	With mild use disorder
F12.221	With moderate or severe use disorder
F12.921	Without use disorder
F12.921	Cannabis receptor agonist–induced delirium, pharmaceutical (medication taken as prescribed)
	Cannabis use disorder
F12.10	Mild
F12.11	In early remission
F12.11	In sustained remission
F12.20	Moderate
F12.21	In early remission
F12.21	In sustained remission
F12.20	Severe
F12.21	In early remission
F12.21	In sustained remission

	Cannabis withdrawal
F12.13	With mild use disorder
F12.23	With moderate or severe use disorder
F12.93	Without use disorder
F06.1	Catatonia associated with another mental disorder (catatonia specifier)
F06.1	Catatonic disorder due to another medical condition
	Central sleep apnea
G47.37	Central sleep apnea comorbid with opioid use
R06.3	Cheyne-Stokes breathing
G47.31	Idiopathic central sleep apnea

Z56.1	Change of job
Z72.810	Child or adolescent antisocial behavior
Z62.898	Child affected by parental relationship distress
F80.81	Childhood-onset fluency disorder (stuttering)
	Child neglect, Confirmed
T74.02XA	Initial encounter
T74.02XD	Subsequent encounter
	Child neglect, Suspected
T76.02XA	Initial encounter
T76.02XD	Subsequent encounter
	Child physical abuse, Confirmed
T74.12XA	Initial encounter
T74.12XD	Subsequent encounter
	Child physical abuse, Suspected
T76.12XA	Initial encounter
T76.12XD	Subsequent encounter
	Child psychological abuse, Confirmed
T74.32XA	Initial encounter
T74.32XD	Subsequent encounter
	Child psychological abuse, Suspected
T76.32XA	Initial encounter
T76.32XD	Subsequent encounter
	Child sexual abuse, Confirmed
T74.22XA	Initial encounter
T74.22XD	Subsequent encounter
	Child sexual abuse, Suspected
T76.22XA	Initial encounter
T76.22XD	Subsequent encounter
	Circadian rhythm sleep-wake disorders
G47.22	Advanced sleep phase type
G47.21	Delayed sleep phase type
G47.23	Irregular sleep-wake type
G47.24	Non-24-hour sleep-wake type
G47.26	Shift work type
G47.20	Unspecified type

	Cocaine-induced anxiety disorder
F14.180	With mild use disorder
F14.280	With moderate or severe use disorder
F14.980	Without use disorder
	Cocaine-induced bipolar and related disorder
F14.14	With mild use disorder
F14.24	With moderate or severe use disorder
F14.94	Without use disorder
	Cocaine-induced depressive disorder
F14.14	With mild use disorder
F14.24	With moderate or severe use disorder
F14.94	Without use disorder
	Cocaine-induced mild neurocognitive disorder
F14.188	With mild use disorder
F14.288	With moderate or severe use disorder
F14.988	Without use disorder
	Cocaine-induced obsessive-compulsive and related disorder
F14.188	With mild use disorder
F14.288	With moderate or severe use disorder
F14.988	Without use disorder
	Cocaine-induced psychotic disorder
F14.159	With mild use disorder
F14.259	With moderate or severe use disorder
F14.959	Without use disorder
	Cocaine-induced sexual dysfunction
F14.181	With mild use disorder
F14.281	With moderate or severe use disorder
F14.981	Without use disorder
	Cocaine-induced sleep disorder
F14.182	With mild use disorder
F14.282	With moderate or severe use disorder
F14.982	Without use disorder
	Cocaine intoxication, With perceptual disturbances
F14.122	With mild use disorder
F14.222	With moderate or severe use disorder
F14.922	Without use disorder
	Cocaine intoxication, Without perceptual disturbances

F14.120 With mild use disorder
F14.220 With moderate or severe use disorder
F14.920 Without use disorder
Cocaine intoxication delirium
F14.121 With mild use disorder

F14.221 With moderate or severe use disorder
F14.921 Without use disorder

Cocaine use disorder
F14.10 Mild
F14.11 In early remission
F14.11 In sustained remission
F14.20 Moderate
F14.21 In early remission
F14.21 In sustained remission
F14.20 Severe
F14.21 In early remission
F14.21 In sustained remission
Cocaine withdrawal
F14.13 With mild use disorder
F14.23 With moderate or severe use disorder
F14.93 Without use disorder
Conduct disorder
F91.2 Adolescent-onset type
F91.1 Childhood-onset type
F91.9 Unspecified onset
Conversion disorder (*see* Functional neurological symptom disorder)
Z65.0 Conviction in civil or criminal proceedings without imprisonment
R45.88 Current nonsuicidal self-injury
Current suicidal behavior
T14.91A Initial encounter
T14.91D Subsequent encounter
F34.0 Cyclothymic disorder
F52.32 Delayed ejaculation

	Delirium
F05	Delirium due to another medical condition
F05	Delirium due to multiple etiologies
	Medication-induced delirium *(for ICD-10-CM codes, see specific substances*)
	Substance intoxication delirium *(see specific substances for codes)*
	Substance withdrawal delirium *(see specific substances for codes)*
F22	Delusional disorder
F60.7	Dependent personality disorder
F48.1	Depersonalization/derealization disorder
	Depressive disorder due to another medical condition
F06.31	With depressive features
F06.32	With major depressive–like episode
F06.34	With mixed features
F82	Developmental coordination disorder
Z71.3	Dietary counseling
Z56.4	Discord with boss and workmates
Z59.2	Discord with neighbor, lodger, or landlord
Z64.4	Discord with social service provider, including probation officer, case manager, or social services worker

F94.2	Disinhibited social engagement disorder
Z63.5	Disruption of family by separation or divorce
F34.81	Disruptive mood dysregulation disorder
F44.0	Dissociative amnesia
F44.1	Dissociative amnesia, with dissociative fugue
F44.81	Dissociative identity disorder
Z55.4	Educational maladjustment and discord with teachers and classmates
F98.1	Encopresis
F98.0	Enuresis
F52.21	Erectile disorder
F42.4	Excoriation (skin-picking) disorder
F65.2	Exhibitionistic disorder
Z65.5	Exposure to disaster, war, or other hostilities
Z59.5	Extreme poverty

F68.A	Factitious disorder imposed on another
F68.10	Factitious disorder imposed on self
Z55.2	Failed school examination
F52.31	Female orgasmic disorder
F52.22	Female sexual interest/arousal disorder
F65.0	Fetishistic disorder
F59.41	Food insecurity
F65.81	Frotteuristic disorder
	Functional neurological symptom disorder (conversion disorder)
F44.4	With abnormal movement
F44.6	With anesthesia or sensory loss
F44.5	With attacks or seizures
F44.7	With mixed symptoms
F44.6	With special sensory symptom
F44.4	With speech symptom
F44.4	With swallowing symptoms
F44.4	With weakness/paralysis
F63.0	Gambling disorder
F64.0	Gender dysphoria in adolescents and adults
F64.2	Gender dysphoria in children
F41.1	Generalized anxiety disorder
Z31.5	Genetic counseling
F52.6	Genito-pelvic pain/penetration disorder
F88	Global developmental delay
F16.983	Hallucinogen persisting perception disorder
	For additional hallucinogen-related substance disorders and hallucinogen-induced mental disorders, see entries for Other hallucinogen and Phencyclidine
Z63.8	High expressed emotion level within family
Z91.52	History of nonsuicidal self-injury
Z91.51	History of suicidal behavior
F60.4	Histrionic personality disorder

F42.3	Hoarding disorder
Z59.01	Homelessness, sheltered
Z59.02	Homelessness, unsheltered

F51.11	Hypersomnolence disorder
Z55.0	Illiteracy and low-level literacy
F45.21	Illness anxiety disorder
Z65.1	Imprisonment or other incarceration
Z59.1	Inadequate housing
	Inhalant-induced anxiety disorder
F18.180	With mild use disorder
F18.280	With moderate or severe use disorder
F18.980	Without use disorder
	Inhalant-induced depressive disorder
F18.14	With mild use disorder
F18.24	With moderate or severe use disorder
F18.94	Without use disorder
	Inhalant-induced major neurocognitive disorder
F18.17	With mild use disorder
F18.27	With moderate or severe use disorder
F18.97	Without use disorder
	Inhalant-induced mild neurocognitive disorder
F18.188	With mild use disorder
F18.288	With moderate or severe use disorder
F18.988	Without use disorder
	Inhalant-induced psychotic disorder
F18.159	With mild use disorder
F18.259	With moderate or severe use disorder
F18.959	Without use disorder
	Inhalant intoxication
F18.120	With mild use disorder
F18.220	With moderate or severe use disorder
F18.920	Without use disorder
	Inhalant intoxication delirium
F18.121	With mild use disorder
F18.221	With moderate or severe use disorder
F18.921	Without use disorder
	Inhalant use disorder
F18.10	Mild
F18.11	In early remission
F18.11	In sustained remission

F18.20	Moderate
F18.21	In early remission
F18.21	In sustained remission
F18.20	Severe
F18.21	In early remission
F18.21	In sustained remission

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F51.01	Insomnia disorder
Z59.7	Insufficient social or health insurance or welfare support
	Intellectual developmental disorder (intellectual disability)
F70	Mild
F71	Moderate
F72	Severe
F73	Profound
F63.81	Intermittent explosive disorder
F16.921	Ketamine or other hallucinogen–induced delirium (ketamine or other hallucinogen medication taken as prescribed or for medical reasons)
F63.2	Kleptomania
Z58.6	Lack of safe drinking water
F80.2	Language disorder
Z59.6	Low income
	Major depressive disorder, Recurrent episode
F33.42	In full remission
F33.41	In partial remission
F33.0	Mild
F33.1	Moderate
F33.2	Severe
F33.3	With psychotic features
F33.9	Unspecified
	Major depressive disorder, Single episode
F32.5	In full remission
F32.4	In partial remission
F32.0	Mild
F32.1	Moderate
F32.2	Severe

F32.3 With psychotic features

F32.9 Unspecified

Major neurocognitive disorder due to another medical condition

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

Major neurocognitive disorder due to HIV infection (*code first* B20 HIV infection)

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

Major neurocognitive disorder due to Huntington's disease (*code first* G10 Huntington's disease)

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

Major neurocognitive disorder due to multiple etiologies

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

Major neurocognitive disorder due to possible Alzheimer's disease (*code first* G30.9 Alzheimer's disease)

Major neurocognitive disorder due to probable Alzheimer's disease (*code first* G30.9 Alzheimer's disease)

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

Major neurocognitive disorder due to possible frontotemporal degeneration (*code first* G31.09 frontotemporal degeneration)

Major neurocognitive disorder due to probable frontotemporal degeneration (*code first* G31.09 frontotemporal degeneration)

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

Major neurocognitive disorder with possible Lewy bodies (*code first* G31.83 Lewy body disease)

Major neurocognitive disorder with probable Lewy bodies (*code first* G31.83 Lewy body disease)

F02.81 With behavioral disturbance

F02.80 Without behavioral disturbance

Major neurocognitive disorder possibly due to Parkinson's disease (*code first* G20 Parkinson's disease)

	Major neurocognitive disorder probably due to Parkinson's disease (*code first* G20 Parkinson's disease)
F02.81	With behavioral disturbance
F02.80	Without behavioral disturbance
	Major neurocognitive disorder possibly due to vascular disease
	Major neurocognitive disorder probably due to vascular disease
F01.51	With behavioral disturbance
F01.50	Without behavioral disturbance
	Major neurocognitive disorder due to prion disease (*code first* A81.9 prion disease)
F02.81	With behavioral disturbance
F02.80	Without behavioral disturbance
	Major neurocognitive disorder due to traumatic brain injury (*code first* S06.2X9S diffuse traumatic brain injury with loss of consciousness of unspecified duration, sequela)
F02.81	With behavioral disturbance
F02.80	Without behavioral disturbance

F52.0	Male hypoactive sexual desire disorder
Z76.5	Malingering
G25.71	Medication-induced acute akathisia
G24.02	Medication-induced acute dystonia
	Medication-induced delirium *(for ICD-10-CM codes, see specific substances)*
G25.1	Medication-induced postural tremor
G31.84	Mild frontotemporal neurocognitive disorder
G31.84	Mild neurocognitive disorder due to Alzheimer's disease

G31.84	Mild neurocognitive disorder due to another medical condition
G31.84	Mild neurocognitive disorder due to HIV infection
G31.84	Mild neurocognitive disorder due to Huntington's disease
G31.84	Mild neurocognitive disorder with Lewy bodies
G31.84	Mild neurocognitive disorder due to multiple etiologies
G31.84	Mild neurocognitive disorder due to Parkinson's disease
G31.84	Mild neurocognitive disorder due to prion disease
G31.84	Mild neurocognitive disorder due to traumatic brain injury
G31.84	Mild vascular neurocognitive disorder

F60.81	Narcissistic personality disorder
	Narcolepsy
G47.411	Narcolepsy with cataplexy or hypocretin deficiency (type 1)
G47.421	Narcolepsy with cataplexy or hypocretin deficiency due to a medical condition
G47.419	Narcolepsy without cataplexy and either without hypocretin deficiency or hypocretin unmeasured (type 2)
G47.429	Narcolepsy without cataplexy and without hypocretin deficiency due to a medical condition
G21.0	Neuroleptic malignant syndrome
F51.5	Nightmare disorder
Z03.89	No diagnosis or condition
Z91.19	Nonadherence to medical treatment
	Non–rapid eye movement sleep arousal disorders
F51.4	Sleep terror type
F51.3	Sleepwalking type
R45.88	Nonsuicidal self-injury, current
Z91.52	Nonsuicidal self-injury, history of
F42.2	Obsessive-compulsive disorder
F60.5	Obsessive-compulsive personality disorder
F06.8	Obsessive-compulsive and related disorder due to another medical condition
G47.33	Obstructive sleep apnea hypopnea
	Opioid-induced anxiety disorder
F11.188	With mild use disorder
F11.288	With moderate or severe use disorder
F11.988	Without use disorder
F11.921	Opioid-induced delirium (opioid medication taken as prescribed)
F11.988	Opioid-induced delirium (during withdrawal from opioid medication taken as prescribed)
	Opioid-induced depressive disorder
F11.14	With mild use disorder
F11.24	With moderate or severe use disorder
F11.94	Without use disorder

	Opioid-induced sexual dysfunction
F11.181	With mild use disorder

F11.281 With moderate or severe use disorder
F11.981 Without use disorder
Opioid-induced sleep disorder
F11.182 With mild use disorder
F11.282 With moderate or severe use disorder
F11.982 Without use disorder
Opioid intoxication, With perceptual disturbances
F11.122 With mild use disorder
F11.222 With moderate or severe use disorder
F11.922 Without use disorder
Opioid intoxication, Without perceptual disturbances
F11.120 With mild use disorder
F11.220 With moderate or severe use disorder
F11.920 Without use disorder
Opioid intoxication delirium
F11.121 With mild use disorder
F11.221 With moderate or severe use disorder
F11.921 Without use disorder
Opioid use disorder
F11.10 Mild
F11.11 In early remission
F11.11 In sustained remission
F11.20 Moderate
F11.21 In early remission
F11.21 In sustained remission
F11.20 Severe
F11.21 In early remission
F11.21 In sustained remission
Opioid withdrawal
F11.13 With mild use disorder
F11.23 With moderate or severe use disorder
F11.93 Without use disorder
Opioid withdrawal delirium
F11.188 With mild use disorder
F11.288 With moderate or severe use disorder
F11.988 Without use disorder
F91.3 Oppositional defiant disorder

Other adverse effect of medication

T50.905A Initial encounter

T50.905S Sequelae

T50.905D Subsequent encounter

Other circumstances related to adult abuse by nonspouse or nonpartner

Z69.82 Encounter for mental health services for perpetrator of nonspousal or nonpartner adult abuse

Z69.81 Encounter for mental health services for victim of nonspousal or nonpartner adult abuse

Other circumstances related to child neglect

Z69.021 Encounter for mental health services for perpetrator of nonparental child neglect

Z69.011 Encounter for mental health services for perpetrator of parental child neglect

Z69.010 Encounter for mental health services for victim of child neglect by parent

Z69.020 Encounter for mental health services for victim of nonparental child neglect

Z62.812 Personal history (past history) of neglect in childhood

Other circumstances related to child physical abuse

Z69.021 Encounter for mental health services for perpetrator of nonparental child physical abuse

Z69.011 Encounter for mental health services for perpetrator of parental child physical abuse

Z69.010 Encounter for mental health services for victim of child physical abuse by parent

Z69.020 Encounter for mental health services for victim of nonparental child physical abuse

Z62.810 Personal history (past history) of physical abuse in childhood

Other circumstances related to child psychological abuse

Z69.021 Encounter for mental health services for perpetrator of nonparental child psychological abuse

Z69.011 Encounter for mental health services for perpetrator of parental child psychological abuse

Z69.010 Encounter for mental health services for victim of child psychological abuse by parent

Z69.020 Encounter for mental health services for victim of nonparental child psychological abuse

Z62.811 Personal history (past history) of psychological abuse in childhood

Other circumstances related to child sexual abuse

Z69.021	Encounter for mental health services for perpetrator of nonparental child sexual abuse
Z69.011	Encounter for mental health services for perpetrator of parental child sexual abuse
Z69.010	Encounter for mental health services for victim of child sexual abuse by parent
Z69.020	Encounter for mental health services for victim of nonparental child sexual abuse
Z62.810	Personal history (past history) of sexual abuse in childhood
	Other circumstances related to spouse or partner abuse, Psychological
Z69.12	Encounter for mental health services for perpetrator of spouse or partner psychological abuse
Z69.11	Encounter for mental health services for victim of spouse or partner psychological abuse
Z91.411	Personal history (past history) of spouse or partner psychological abuse

	Other circumstances related to spouse or partner neglect
Z69.12	Encounter for mental health services for perpetrator of spouse or partner neglect
Z69.11	Encounter for mental health services for victim of spouse or partner neglect
Z91.412	Personal history (past history) of spouse or partner neglect
	Other circumstances related to spouse or partner violence, Physical
Z69.12	Encounter for mental health services for perpetrator of spouse or partner violence, Physical
Z69.11	Encounter for mental health services for victim of spouse or partner violence, Physical
Z91.410	Personal history (past history) of spouse or partner violence, Physical
	Other circumstances related to spouse or partner violence, Sexual
Z69.12	Encounter for mental health services for perpetrator of spouse or partner violence, Sexual
Z69.81	Encounter for mental health services for victim of spouse or partner violence, Sexual
Z91.410	Personal history (past history) of spouse or partner violence, Sexual
Z71.9	Other counseling or consultation
Z59.9	Other economic problem
	Other hallucinogen–induced anxiety disorder
F16.180	With mild use disorder
F16.280	With moderate or severe use disorder

F16.980	Without use disorder
	Other hallucinogen–induced bipolar and related disorder
F16.14	With mild use disorder
F16.24	With moderate or severe use disorder
F16.94	Without use disorder
F16.921	Other hallucinogen–induced delirium (other hallucinogen medication taken as prescribed or for medical reasons)
	Other hallucinogen–induced depressive disorder
F16.14	With mild use disorder
F16.24	With moderate or severe use disorder
F16.94	Without use disorder
	Other hallucinogen–induced psychotic disorder
F16.159	With mild use disorder
F16.259	With moderate or severe use disorder
F16.959	Without use disorder
	Other hallucinogen intoxication
F16.120	With mild use disorder
F16.220	With moderate or severe use disorder
F16.920	Without use disorder
	Other hallucinogen intoxication delirium
F16.121	With mild use disorder
F16.221	With moderate or severe use disorder
F16.921	Without use disorder

	Other hallucinogen use disorder
F16.10	Mild
F16.11	In early remission
F16.11	In sustained remission
F16.20	Moderate
F16.21	In early remission
F16.21	In sustained remission
F16.20	Severe
F16.21	In early remission
F16.21	In sustained remission
Z59.9	Other housing problem

G25.79	Other medication-induced movement disorder
G21.19	Other medication-induced parkinsonism
Z91.49	Other personal history of psychological trauma
Z91.89	Other personal risk factors
Z56.6	Other physical and mental strain related to work
Z56.9	Other problem related to employment
Z60.9	Other problem related to social environment
Z55.9	Other problems related to education and literacy
F41.8	Other specified anxiety disorder
F90.8	Other specified attention-deficit/hyperactivity disorder
F31.89	Other specified bipolar and related disorder
R41.0	Other specified delirium
F32.89	Other specified depressive disorder
F91.8	Other specified disruptive, impulse-control, and conduct disorder
F44.89	Other specified dissociative disorder
	Other specified elimination disorder
R15.9	With fecal symptoms
N39.498	With urinary symptoms
F50.89	Other specified feeding or eating disorder
F64.8	Other specified gender dysphoria
G47.19	Other specified hypersomnolence disorder
G47.09	Other specified insomnia disorder
F99	Other specified mental disorder
F06.8	Other specified mental disorder due to another medical condition
F88	Other specified neurodevelopmental disorder
F42.8	Other specified obsessive-compulsive and related disorder
F65.89	Other specified paraphilic disorder
F60.89	Other specified personality disorder
F28	Other specified schizophrenia spectrum and other psychotic disorder
F52.8	Other specified sexual dysfunction
G47.8	Other specified sleep-wake disorder
F45.8	Other specified somatic symptom and related disorder
F95.8	Other specified tic disorder
F43.8	Other specified trauma- and stressor-related disorder

	Other stimulant intoxication, With perceptual disturbances
F15.122	With mild use disorder
F15.222	With moderate or severe use disorder
F15.922	Without use disorder

	Other stimulant intoxication, Without perceptual disturbances
F15.120	With mild use disorder
F15.220	With moderate or severe use disorder
F15.920	Without use disorder
	See also Other or unspecified stimulant use disorder
	Other stimulant withdrawal
F15.13	With mild use disorder
F15.23	With moderate or severe use disorder
F15.93	Without use disorder
F19.921	Other (or unknown) medication–induced delirium (other [or unknown] medication taken as prescribed)
F19.931	Other (or unknown) medication–induced delirium (during withdrawal from other [or unknown] medication taken as prescribed)
	Other (or unknown) substance–induced anxiety disorder
F19.180	With mild use disorder
F19.280	With moderate or severe use disorder
F19.980	Without use disorder
	Other (or unknown) substance–induced bipolar and related disorder
F19.14	With mild use disorder
F19.24	With moderate or severe use disorder
F19.94	Without use disorder
	Other (or unknown) substance–induced depressive disorder
F19.14	With mild use disorder
F19.24	With moderate or severe use disorder
F19.94	Without use disorder
	Other (or unknown) substance–induced major neurocognitive disorder
F19.17	With mild use disorder
F19.27	With moderate or severe use disorder
F19.97	Without use disorder
	Other (or unknown) substance–induced mild neurocognitive disorder
F19.188	With mild use disorder
F19.288	With moderate or severe use disorder
F19.988	Without use disorder

Other (or unknown) substance–induced obsessive-compulsive and related disorder

F19.188 With mild use disorder

F19.288 With moderate or severe use disorder

F19.988 Without use disorder

Other (or unknown) substance–induced psychotic disorder

F19.159 With mild use disorder

F19.259 With moderate or severe use disorder

F19.959 Without use disorder

Other (or unknown) substance–induced sexual dysfunction

F19.181 With mild use disorder

F19.281 With moderate or severe use disorder

F19.981 Without use disorder

Other (or unknown) substance–induced sleep disorder

F19.182 With mild use disorder

F19.282 With moderate or severe use disorder

F19.982 Without use disorder

Other (or unknown) substance intoxication, With perceptual disturbances

F19.122 With mild use disorder

F19.222 With moderate or severe use disorder

F19.922 Without use disorder

Other (or unknown) substance intoxication, Without perceptual disturbances

F19.120 With mild use disorder

F19.220 With moderate or severe use disorder

F19.920 Without use disorder

Other (or unknown) substance intoxication delirium

F19.121 With mild use disorder

F19.221 With moderate or severe use disorder

F19.921 Without use disorder

Other (or unknown) substance use disorder

F19.10 Mild

F19.11 In early remission

F19.11 In sustained remission

F19.20 Moderate

F19.21	In early remission
F19.21	In sustained remission
F19.20	Severe
F19.21	In early remission
F19.21	In sustained remission
	Other (or unknown) substance withdrawal, With perceptual disturbances
F19.132	With mild use disorder
F19.232	With moderate or severe use disorder
F19.932	Without use disorder
	Other (or unknown) substance withdrawal, Without perceptual disturbances
F19.130	With mild use disorder
F19.230	With moderate or severe use disorder
F19.930	Without use disorder
	Other (or unknown) substance withdrawal delirium
F19.131	With mild use disorder
F19.231	With moderate or severe use disorder
F19.931	Without use disorder

	Other or unspecified stimulant use disorder
F15.10	Mild
F15.11	In early remission
F15.11	In sustained remission
F15.20	Moderate
F15.21	In early remission
F15.21	In sustained remission
F15.20	Severe
F15.21	In early remission
F15.21	In sustained remission
E66.9	Overweight or obesity
no code	Panic attack specifier
F41.0	Panic disorder
F60.0	Paranoid personality disorder
	Parent-child relational problem
Z62.821	Parent–adopted child
Z62.820	Parent–biological child

Z62.822	Parent–foster child
Z62.898	Other caregiver–child
F65.4	Pedophilic disorder
F95.1	Persistent (chronic) motor or vocal tic disorder
F34.1	Persistent depressive disorder
Z91.82	Personal history of military deployment
Z91.49	Personal history of psychological trauma
F07.0	Personality change due to another medical condition
F12.921	Pharmaceutical cannabis receptor agonist–induced delirium (pharmaceutical cannabis receptor agonist medication taken as prescribed)
Z60.0	Phase of life problem
	Phencyclidine-induced anxiety disorder
F16.180	With mild use disorder
F16.280	With moderate or severe use disorder
F16.980	Without use disorder
	Phencyclidine-induced bipolar and related disorder
F16.14	With mild use disorder
F16.24	With moderate or severe use disorder
F16.94	Without use disorder
	Phencyclidine-induced depressive disorder
F16.14	With mild use disorder
F16.24	With moderate or severe use disorder
F16.94	Without use disorder
	Phencyclidine-induced psychotic disorder
F16.159	With mild use disorder
F16.259	With moderate or severe use disorder
F16.959	Without use disorder

	Phencyclidine intoxication
F16.120	With mild use disorder
F16.220	With moderate or severe use disorder
F16.920	Without use disorder
	Phencyclidine intoxication delirium
F16.121	With mild use disorder
F16.221	With moderate or severe use disorder

F16.921	Without use disorder
	Phencyclidine use disorder
F16.10	Mild
F16.11	In early remission
F16.11	In sustained remission
F16.20	Moderate
F16.21	In early remission
F16.21	In sustained remission
F16.20	Severe
F16.21	In early remission
F16.21	In sustained remission
	Pica
F50.89	In adults
F98.3	In children
F43.10	Posttraumatic stress disorder
F52.4	Premature (early) ejaculation
F32.81	Premenstrual dysphoric disorder
Z56.82	Problem related to current military deployment status
Z72.9	Problem related to lifestyle
Z60.2	Problem related to living alone
Z59.3	Problem related to living in a residential institution
Z55.8	Problems related to inadequate teaching
Z64.1	Problems related to multiparity
Z65.3	Problems related to other legal circumstances
Z65.2	Problems related to release from prison
Z64.0	Problems related to unwanted pregnancy
F43.8	Prolonged grief disorder
F95.0	Provisional tic disorder
F54	Psychological factors affecting other medical conditions
	Psychotic disorder due to another medical condition
F06.2	With delusions
F06.0	With hallucinations
F63.1	Pyromania
G47.52	Rapid eye movement sleep behavior disorder
F94.1	Reactive attachment disorder
Z63.0	Relationship distress with spouse or intimate partner
Z65.8	Religious or spiritual problem

G25.81	Restless legs syndrome
F98.21	Rumination disorder
	Schizoaffective disorder
F25.0	Bipolar type
F25.1	Depressive type
F60.1	Schizoid personality disorder
F20.9	Schizophrenia
F20.81	Schizophreniform disorder
F21	Schizotypal personality disorder
Z55.1	Schooling unavailable and unattainable
	Sedative-, hypnotic-, or anxiolytic-induced anxiety disorder
F13.180	With mild use disorder
F13.280	With moderate or severe use disorder
F13.980	Without use disorder
	Sedative-, hypnotic-, or anxiolytic-induced bipolar and related disorder
F13.14	With mild use disorder
F13.24	With moderate or severe use disorder
F13.94	Without use disorder
F13.921	Sedative-, hypnotic-, or anxiolytic-induced delirium (sedative, hypnotic, or anxiolytic medication taken as prescribed)
F13.931	Sedative, hypnotic, or anxiolytic–induced delirium (during withdrawal from sedative, hypnotic, or anxiolytic medication taken as prescribed)
	Sedative-, hypnotic-, or anxiolytic-induced depressive disorder
F13.14	With mild use disorder
F13.24	With moderate or severe use disorder
F13.94	Without use disorder
	Sedative-, hypnotic-, or anxiolytic-induced major neurocognitive disorder
F13.27	With moderate or severe use disorder
F13.97	Without use disorder
	Sedative-, hypnotic-, or anxiolytic-induced mild neurocognitive disorder
F13.188	With mild use disorder
F13.288	With moderate or severe use disorder
F13.988	Without use disorder
	Sedative-, hypnotic-, or anxiolytic-induced psychotic disorder
F13.159	With mild use disorder
F13.259	With moderate or severe use disorder

F13.959	Without use disorder
	Sedative-, hypnotic-, or anxiolytic-induced sexual dysfunction
F13.181	With mild use disorder
F13.281	With moderate or severe use disorder
F13.981	Without use disorder
	Sedative-, hypnotic-, or anxiolytic-induced sleep disorder
F13.182	With mild use disorder
F13.282	With moderate or severe use disorder
F13.982	Without use disorder

	Sedative, hypnotic, or anxiolytic intoxication
F13.120	With mild use disorder
F13.220	With moderate or severe use disorder
F13.920	Without use disorder
	Sedative, hypnotic, or anxiolytic intoxication delirium
F13.121	With mild use disorder
F13.221	With moderate or severe use disorder
F13.921	Without use disorder
	Sedative, hypnotic, or anxiolytic use disorder
F13.10	Mild
F13.11	In early remission
F13.11	In sustained remission
F13.20	Moderate
F13.21	In early remission
F13.21	In sustained remission
F13.20	Severe
F13.21	In early remission
F13.21	In sustained remission
	Sedative, hypnotic, or anxiolytic withdrawal, With perceptual disturbances
F13.132	With mild use disorder
F13.232	With moderate or severe use disorder
F13.932	Without use disorder
	Sedative, hypnotic, or anxiolytic withdrawal, Without perceptual disturbances
F13.130	With mild use disorder
F13.230	With moderate or severe use disorder

F13.930	Without use disorder
	Sedative, hypnotic, or anxiolytic withdrawal delirium
F13.131	With mild use disorder
F13.231	With moderate or severe use disorder
F13.931	Without use disorder
F94.0	Selective mutism
F93.0	Separation anxiety disorder
Z70.9	Sex counseling
Z56.81	Sexual harassment on the job
F65.51	Sexual masochism disorder
F65.52	Sexual sadism disorder
Z62.891	Sibling relational problem
	Sleep-related hypoventilation
G47.36	Comorbid sleep-related hypoventilation
G47.35	Congenital central alveolar hypoventilation
G47.34	Idiopathic hypoventilation
F40.10	Social anxiety disorder
Z60.4	Social exclusion or rejection
F80.82	Social (pragmatic) communication disorder

F45.1	Somatic symptom disorder
	Specific learning disorder
F81.2	With impairment in mathematics
F81.0	With impairment in reading
F81.81	With impairment in written expression
	Specific phobia
F40.218	Animal
	Blood-injection-injury
F40.230	Fear of blood
F40.231	Fear of injections and transfusions
F40.233	Fear of injury
F40.232	Fear of other medical care
F40.228	Natural environment
F40.298	Other
F40.248	Situational

F80.0	Speech sound disorder
	Spouse or partner abuse, Psychological, Confirmed
T74.31XA	Initial encounter
T74.31XD	Subsequent encounter
	Spouse or partner abuse, Psychological, Suspected
T76.31XA	Initial encounter
T76.31XD	Subsequent encounter
	Spouse or partner neglect, Confirmed
T74.01XA	Initial encounter
T74.01XD	Subsequent encounter
	Spouse or partner neglect, Suspected
T76.01XA	Initial encounter
T76.01XD	Subsequent encounter
	Spouse or partner violence, Physical, Confirmed
T74.11XA	Initial encounter
T74.11XD	Subsequent encounter
	Spouse or partner violence, Physical, Suspected
T76.11XA	Initial encounter
T76.11XD	Subsequent encounter
	Spouse or partner violence, Sexual, Confirmed
T74.21XA	Initial encounter
T74.21XD	Subsequent encounter
	Spouse or partner violence, Sexual, Suspected
T76.21XA	Initial encounter
T76.21XD	Subsequent encounter
F98.4	Stereotypic movement disorder
	Stimulant intoxication *(see amphetamine-type substance, cocaine, or other or unspecified stimulant intoxication for specific codes)*
	Stimulant use disorder *(see amphetamine-type substance, cocaine, or other or unspecified stimulant use disorder for specific codes)*

	Stimulant withdrawal *(see amphetamine-type substance, cocaine, or other or unspecified stimulant withdrawal for specific codes)*
Z56.3	Stressful work schedule
F80.81	Stuttering (childhood-onset fluency disorder)

	Substance intoxication delirium *(see specific substances for codes)*
	Substance withdrawal delirium *(see specific substances for codes)*
	Substance/medication-induced anxiety disorder *(see specific substances for codes)*
	Substance/medication-induced bipolar and related disorder *(see specific substances for codes)*
	Substance/medication-induced depressive disorder *(see specific substances for codes)*
	Substance/medication-induced major or mild neurocognitive disorder *(see specific substances for codes)*
	Substance/medication-induced obsessive-compulsive and related disorder *(see specific substances for codes)*
	Substance/medication-induced psychotic disorder *(see specific substances for codes)*
	Substance/medication-induced sexual dysfunction *(see specific substances for codes)*
	Substance/medication-induced sleep disorder *(see specific substances for codes)*
	Suicidal behavior, current
T14.91A	Initial encounter
T14.91D	Subsequent encounter
Z91.51	Suicidal behavior, history of
G25.71	Tardive akathisia
G24.01	Tardive dyskinesia
G24.09	Tardive dystonia
Z60.5	Target of (perceived) adverse discrimination or persecution
Z56.2	Threat of job loss
	Tic disorders
F95.8	Other specified tic disorder
F95.1	Persistent (chronic) motor or vocal tic disorder
F95.0	Provisional tic disorder
F95.2	Tourette's disorder
F95.9	Unspecified tic disorder
F17.208	Tobacco-induced sleep disorder, With moderate or severe use disorder
	Tobacco use disorder
Z72.0	Mild
F17.200	Moderate
F17.201	In early remission
F17.201	In sustained remission

F17.200	Severe
F17.201	In early remission
F17.201	In sustained remission

F17.203	Tobacco withdrawal
F95.2	Tourette's disorder
F65.1	Transvestic disorder
F63.3	Trichotillomania (hair-pulling disorder)
Z75.3	Unavailability or inaccessibility of health care facilities
Z75.4	Unavailability or inaccessibility of other helping agencies
Z63.4	Uncomplicated bereavement
Z56.5	Uncongenial work environment
Z55.3	Underachievement in school
Z56.0	Unemployment
F10.99	Unspecified alcohol-related disorder
F41.9	Unspecified anxiety disorder
F90.9	Unspecified attention-deficit/hyperactivity disorder
F31.9	Unspecified bipolar and related disorder
F15.99	Unspecified caffeine-related disorder
F12.99	Unspecified cannabis-related disorder
F06.1	Unspecified catatonia (*code first* R29.818 other symptoms involving nervous and musculoskeletal systems)
F80.9	Unspecified communication disorder
R41.0	Unspecified delirium
F32.A	Unspecified depressive disorder
F91.9	Unspecified disruptive, impulse-control, and conduct disorder
F44.9	Unspecified dissociative disorder
	Unspecified elimination disorder
R15.9	With fecal symptoms
R32	With urinary symptoms
F50.9	Unspecified feeding or eating disorder
F64.9	Unspecified gender dysphoria
F16.99	Unspecified hallucinogen-related disorder
G47.10	Unspecified hypersomnolence disorder
F18.99	Unspecified inhalant-related disorder

G47.00	Unspecified insomnia disorder
F79	Unspecified intellectual developmental disorder (intellectual disability)
F99	Unspecified mental disorder
F09	Unspecified mental disorder due to another medical condition
F39	Unspecified mood disorder
R41.9	Unspecified neurocognitive disorder
F89	Unspecified neurodevelopmental disorder
F42.9	Unspecified obsessive-compulsive and related disorder
F11.99	Unspecified opioid-related disorder
F19.99	Unspecified other (or unknown) substance–related disorder
F65.9	Unspecified paraphilic disorder
F60.9	Unspecified personality disorder
F16.99	Unspecified phencyclidine-related disorder

F29	Unspecified schizophrenia spectrum and other psychotic disorder
F13.99	Unspecified sedative-, hypnotic-, or anxiolytic-related disorder
F52.9	Unspecified sexual dysfunction
G47.9	Unspecified sleep-wake disorder
F45.9	Unspecified somatic symptom and related disorder
	Unspecified stimulant-related disorder
F15.99	Unspecified amphetamine-type substance–related disorder
F14.99	Unspecified cocaine-related disorder
F15.99	Unspecified other stimulant–related disorder
F95.9	Unspecified tic disorder
F17.209	Unspecified tobacco-related disorder
F43.9	Unspecified trauma- and stressor-related disorder
Z62.29	Upbringing away from parents
Z65.4	Victim of crime
Z65.4	Victim of terrorism or torture
F65.3	Voyeuristic disorder
Z91.83	Wandering associated with a mental disorder

Numerical Listing of DSM-5-TR Diagnoses and ICD-10-CM Codes

For periodic DSM-5-TR coding and other updates, see www.dsm5.org.

ICD-10-CM	Disorder, condition, or problem
E66.9	Overweight or obesity
F01.50	Major neurocognitive disorder possibly due to vascular disease, Without behavioral disturbance
F01.50	Major neurocognitive disorder probably due to vascular disease, Without behavioral disturbance
F01.51	Major neurocognitive disorder possibly due to vascular disease, With behavioral disturbance
F01.51	Major neurocognitive disorder probably due to vascular disease, With behavioral disturbance
F02.80	Major neurocognitive disorder due to another medical condition, Without behavioral disturbance
F02.80	Major neurocognitive disorder due to HIV infection, Without behavioral disturbance (*code first* B20 HIV infection)
F02.80	Major neurocognitive disorder due to Huntington's disease, Without behavioral disturbance (*code first* G10 Huntington's disease)
F02.80	Major neurocognitive disorder due to multiple etiologies, Without behavioral disturbance
F02.80	Major neurocognitive disorder due to possible Alzheimer's disease, Without behavioral disturbance (*code first* G30.9 Alzheimer's disease)
F02.80	Major neurocognitive disorder due to possible frontotemporal degeneration, Without behavioral disturbance (*code first* G31.09 frontotemporal degeneration)
F02.80	Major neurocognitive disorder with possible Lewy bodies, Without behavioral disturbance (*code first* G31.83 Lewy body disease)
F02.80	Major neurocognitive disorder possibly due to Parkinson's disease, Without behavioral disturbance (*code first* G20 Parkinson's disease)

ICD-10-CM	Disorder, condition, or problem
F02.80	Major neurocognitive disorder due to prion disease, Without behavioral disturbance (*code first* A81.9 prion disease)
F02.80	Major neurocognitive disorder due to probable Alzheimer's disease, Without behavioral disturbance (*code first* G30.9 Alzheimer's disease)
F02.80	Major neurocognitive disorder due to probable frontotemporal degeneration, Without behavioral disturbance (*code first* G31.09 frontotemporal degeneration)

F02.80	Major neurocognitive disorder with probable Lewy bodies, Without behavioral disturbance (*code first* G31.83 Lewy body disease)
F02.80	Major neurocognitive disorder probably due to Parkinson's disease, Without behavioral disturbance (*code first* G20 Parkinson's disease)
F02.80	Major neurocognitive disorder due to traumatic brain injury, Without behavioral disturbance (*code first* S06.2X9S diffuse traumatic brain injury with loss of consciousness of unspecified duration, sequela)
F02.81	Major neurocognitive disorder due to another medical condition, With behavioral disturbance
F02.81	Major neurocognitive disorder due to HIV infection, With behavioral disturbance (*code first* B20 HIV infection)
F02.81	Major neurocognitive disorder due to Huntington's disease, With behavioral disturbance (*code first* G10 Huntington's disease)
F02.81	Major neurocognitive disorder due to multiple etiologies, With behavioral disturbance
F02.81	Major neurocognitive disorder due to possible Alzheimer's disease, With behavioral disturbance (*code first* G30.9 Alzheimer's disease)
F02.81	Major neurocognitive disorder due to possible frontotemporal degeneration, With behavioral disturbance (*code first* G31.09 frontotemporal degeneration)
F02.81	Major neurocognitive disorder with possible Lewy bodies, With behavioral disturbance (*code first* G31.83 Lewy body disease)
F02.81	Major neurocognitive disorder possibly due to Parkinson's disease, With behavioral disturbance (*code first* G20 Parkinson's disease)
F02.81	Major neurocognitive disorder due to prion disease, With behavioral disturbance (*code first* A81.9 prion disease)
F02.81	Major neurocognitive disorder due to probable Alzheimer's disease, With behavioral disturbance (*code first* G30.9 Alzheimer's disease)
F02.81	Major neurocognitive disorder due to probable frontotemporal degeneration, With behavioral disturbance (*code first* G31.09 frontotemporal degeneration)

ICD-10-CM	Disorder, condition, or problem
F02.81	Major neurocognitive disorder with probable Lewy bodies, With behavioral disturbance (*code first* G31.83 Lewy body disease)
F02.81	Major neurocognitive disorder probably due to Parkinson's disease, With behavioral disturbance (*code first* G20 Parkinson's disease)
F02.81	Major neurocognitive disorder due to traumatic brain injury, With behavioral disturbance (*code first* S06.2X9S diffuse traumatic brain injury with loss of consciousness of unspecified duration, sequela)
F05	Delirium due to another medical condition
F05	Delirium due to multiple etiologies
F06.0	Psychotic disorder due to another medical condition, With hallucinations
F06.1	Catatonia associated with another mental disorder (catatonia specifier)
F06.1	Catatonic disorder due to another medical condition
F06.1	Unspecified catatonia (*code first* R29.818 other symptoms involving nervous and musculoskeletal systems)
F06.2	Psychotic disorder due to another medical condition, With delusions
F06.31	Depressive disorder due to another medical condition, With depressive features
F06.32	Depressive disorder due to another medical condition, With major depressive–like episode

F06.33	Bipolar and related disorder due to another medical condition, With manic features
F06.33	Bipolar and related disorder due to another medical condition, With manic- or hypomanic-like episode
F06.34	Bipolar and related disorder due to another medical condition, With mixed features
F06.34	Depressive disorder due to another medical condition, With mixed features
F06.4	Anxiety disorder due to another medical condition
F06.8	Obsessive-compulsive and related disorder due to another medical condition
F06.8	Other specified mental disorder due to another medical condition
F07.0	Personality change due to another medical condition
F09	Unspecified mental disorder due to another medical condition
F10.10	Alcohol use disorder, Mild
F10.11	Alcohol use disorder, Mild, In early remission
F10.11	Alcohol use disorder, Mild, In sustained remission
F10.120	Alcohol intoxication, With mild use disorder

ICD-10-CM	Disorder, condition, or problem
F10.121	Alcohol intoxication delirium, With mild use disorder
F10.130	Alcohol withdrawal, Without perceptual disturbances, With mild use disorder
F10.131	Alcohol withdrawal delirium, With mild use disorder
F10.132	Alcohol withdrawal, With perceptual disturbances, With mild use disorder
F10.14	Alcohol-induced bipolar and related disorder, With mild use disorder
F10.14	Alcohol-induced depressive disorder, With mild use disorder
F10.159	Alcohol-induced psychotic disorder, With mild use disorder
F10.180	Alcohol-induced anxiety disorder, With mild use disorder
F10.181	Alcohol-induced sexual dysfunction, With mild use disorder
F10.182	Alcohol-induced sleep disorder, With mild use disorder
F10.188	Alcohol-induced mild neurocognitive disorder, With mild use disorder
F10.20	Alcohol use disorder, Moderate
F10.20	Alcohol use disorder, Severe
F10.21	Alcohol use disorder, Moderate, In early remission
F10.21	Alcohol use disorder, Moderate, In sustained remission
F10.21	Alcohol use disorder, Severe, In early remission
F10.21	Alcohol use disorder, Severe, In sustained remission
F10.220	Alcohol intoxication, With moderate or severe use disorder
F10.221	Alcohol intoxication delirium, With moderate or severe use disorder
F10.230	Alcohol withdrawal, Without perceptual disturbances, With moderate or severe use disorder
F10.231	Alcohol withdrawal delirium, With moderate or severe use disorder
F10.232	Alcohol withdrawal, With perceptual disturbances, With moderate or severe use disorder
F10.24	Alcohol-induced bipolar and related disorder, With moderate or severe use disorder
F10.24	Alcohol-induced depressive disorder, With moderate or severe use disorder
F10.259	Alcohol-induced psychotic disorder, With moderate or severe use disorder
F10.26	Alcohol-induced major neurocognitive disorder, Amnestic-confabulatory type, With moderate or severe use disorder

F10.27	Alcohol-induced major neurocognitive disorder, Nonamnestic-confabulatory type, With moderate or severe use disorder
F10.280	Alcohol-induced anxiety disorder, With moderate or severe use disorder
F10.281	Alcohol-induced sexual dysfunction, With moderate or severe use disorder

ICD-10-CM	Disorder, condition, or problem
F10.282	Alcohol-induced sleep disorder, With moderate or severe use disorder
F10.288	Alcohol-induced mild neurocognitive disorder, With moderate or severe use disorder
F10.920	Alcohol intoxication, Without use disorder
F10.921	Alcohol intoxication delirium, Without use disorder
F10.930	Alcohol withdrawal, Without perceptual disturbances, Without use disorder
F10.931	Alcohol withdrawal delirium, without use disorder
F10.932	Alcohol withdrawal, With perceptual disturbances, Without use disorder
F10.94	Alcohol-induced bipolar and related disorder, Without use disorder
F10.94	Alcohol-induced depressive disorder, Without use disorder
F10.959	Alcohol-induced psychotic disorder, Without use disorder
F10.96	Alcohol-induced major neurocognitive disorder, Amnestic-confabulatory type, Without use disorder
F10.97	Alcohol-induced major neurocognitive disorder, Nonamnestic-confabulatory type, Without use disorder
F10.980	Alcohol-induced anxiety disorder, Without use disorder
F10.981	Alcohol-induced sexual dysfunction, Without use disorder
F10.982	Alcohol-induced sleep disorder, Without use disorder
F10.988	Alcohol-induced mild neurocognitive disorder, Without use disorder
F10.99	Unspecified alcohol-related disorder
F11.10	Opioid use disorder, Mild
F11.11	Opioid use disorder, Mild, In early remission
F11.11	Opioid use disorder, Mild, In sustained remission
F11.120	Opioid intoxication, Without perceptual disturbances, With mild use disorder
F11.121	Opioid intoxication delirium, With mild use disorder

F11.122	Opioid intoxication, With perceptual disturbances, With mild use disorder
F11.13	Opioid withdrawal, With mild use disorder
F11.14	Opioid-induced depressive disorder, With mild use disorder
F11.181	Opioid-induced sexual dysfunction, With mild use disorder
F11.182	Opioid-induced sleep disorder, With mild use disorder
F11.188	Opioid-induced anxiety disorder, With mild use disorder
F11.188	Opioid withdrawal delirium, With mild use disorder
F11.20	Opioid use disorder, Moderate

ICD-10-CM	Disorder, condition, or problem
F11.20	Opioid use disorder, Severe
F11.21	Opioid use disorder, Moderate, In early remission
F11.21	Opioid use disorder, Moderate, In sustained remission
F11.21	Opioid use disorder, Severe, In early remission
F11.21	Opioid use disorder, Severe, In sustained remission
F11.220	Opioid intoxication, Without perceptual disturbances, With moderate or severe use disorder
F11.221	Opioid intoxication delirium, With moderate or severe use disorder
F11.222	Opioid intoxication, With perceptual disturbances, With moderate or severe use disorder
F11.23	Opioid withdrawal, With moderate or severe use disorder
F11.24	Opioid-induced depressive disorder, With moderate or severe use disorder
F11.281	Opioid-induced sexual dysfunction, With moderate or severe use disorder
F11.282	Opioid-induced sleep disorder, With moderate or severe use disorder
F11.288	Opioid-induced anxiety disorder, With moderate or severe use disorder
F11.288	Opioid withdrawal delirium, With moderate or severe use disorder
F11.920	Opioid intoxication, Without perceptual disturbances, Without use disorder
F11.921	Opioid-induced delirium (opioid medication taken as prescribed)
F11.921	Opioid intoxication delirium, Without use disorder
F11.922	Opioid intoxication, With perceptual disturbances, Without use disorder
F11.93	Opioid withdrawal, Without use disorder
F11.94	Opioid-induced depressive disorder, Without use disorder
F11.981	Opioid-induced sexual dysfunction, Without use disorder
F11.982	Opioid-induced sleep disorder, Without use disorder
F11.988	Opioid-induced anxiety disorder, Without use disorder
F11.988	Opioid-induced delirium (during withdrawal from opioid medication taken as prescribed)
F11.988	Opioid withdrawal delirium, Without use disorder
F11.99	Unspecified opioid-related disorder
F12.10	Cannabis use disorder, Mild
F12.11	Cannabis use disorder, Mild, In early remission
F12.11	Cannabis use disorder, Mild, In sustained remission
F12.120	Cannabis intoxication, Without perceptual disturbances, With mild use disorder
F12.121	Cannabis intoxication delirium, With mild use disorder
F12.122	Cannabis intoxication, With perceptual disturbances, With mild use disorder
F12.13	Cannabis withdrawal, With mild use disorder

ICD-10-CM	Disorder, condition, or problem
F12.159	Cannabis-induced psychotic disorder, With mild use disorder
F12.180	Cannabis-induced anxiety disorder, With mild use disorder
F12.188	Cannabis-induced sleep disorder, With mild use disorder
F12.20	Cannabis use disorder, Moderate
F12.20	Cannabis use disorder, Severe
F12.21	Cannabis use disorder, Moderate, In early remission
F12.21	Cannabis use disorder, Moderate, In sustained remission
F12.21	Cannabis use disorder, Severe, In early remission
F12.21	Cannabis use disorder, Severe, In sustained remission
F12.220	Cannabis intoxication, Without perceptual disturbances, With moderate or severe use disorder
F12.221	Cannabis intoxication delirium, With moderate or severe use disorder
F12.222	Cannabis intoxication, With perceptual disturbances, With moderate or severe use disorder
F12.23	Cannabis withdrawal, With moderate or severe use disorder
F12.259	Cannabis-induced psychotic disorder, With moderate or severe use disorder

F12.280	Cannabis-induced anxiety disorder, With moderate or severe use disorder
F12.288	Cannabis-induced sleep disorder, With moderate or severe use disorder
F12.920	Cannabis intoxication, Without perceptual disturbances, Without use disorder
F12.921	Cannabis intoxication delirium, Without use disorder
F12.921	Pharmaceutical cannabis receptor agonist–induced delirium (pharmaceutical cannabis receptor agonist medication taken as prescribed)
F12.922	Cannabis intoxication, With perceptual disturbances, Without use disorder
F12.93	Cannabis withdrawal, Without use disorder
F12.959	Cannabis-induced psychotic disorder, Without use disorder
F12.980	Cannabis-induced anxiety disorder, Without use disorder
F12.988	Cannabis-induced sleep disorder, Without use disorder
F12.99	Unspecified cannabis-related disorder
F13.10	Sedative, hypnotic, or anxiolytic use disorder, Mild
F13.11	Sedative, hypnotic, or anxiolytic use disorder, Mild, In early remission
F13.11	Sedative, hypnotic, or anxiolytic use disorder, Mild, In sustained remission
F13.120	Sedative, hypnotic, or anxiolytic intoxication, With mild use disorder

F13.121	Sedative, hypnotic, or anxiolytic intoxication delirium, With mild use disorder
F13.130	Sedative, hypnotic, or anxiolytic withdrawal, Without perceptual disturbances, With mild use disorder
F13.131	Sedative, hypnotic, or anxiolytic withdrawal delirium, With mild use disorder
F13.132	Sedative, hypnotic, or anxiolytic withdrawal, With perceptual disturbances, With mild use disorder
F13.14	Sedative-, hypnotic-, or anxiolytic-induced bipolar and related disorder, With mild use disorder
F13.14	Sedative-, hypnotic-, or anxiolytic-induced depressive disorder, With mild use disorder
F13.159	Sedative-, hypnotic-, or anxiolytic-induced psychotic disorder, With mild use disorder
F13.180	Sedative-, hypnotic-, or anxiolytic-induced anxiety disorder, With mild use disorder
F13.181	Sedative-, hypnotic-, or anxiolytic-induced sexual dysfunction, With mild use disorder
F13.182	Sedative-, hypnotic-, or anxiolytic-induced sleep disorder, With mild use disorder
F13.188	Sedative-, hypnotic-, or anxiolytic-induced mild neurocognitive disorder, With mild use disorder
F13.20	Sedative, hypnotic, or anxiolytic use disorder, Moderate
F13.20	Sedative, hypnotic, or anxiolytic use disorder, Severe
F13.21	Sedative, hypnotic, or anxiolytic use disorder, Moderate, In early remission
F13.21	Sedative, hypnotic, or anxiolytic use disorder, Moderate, In sustained remission
F13.21	Sedative, hypnotic, or anxiolytic use disorder, Severe, In early remission
F13.21	Sedative, hypnotic, or anxiolytic use disorder, Severe, In sustained remission
F13.220	Sedative, hypnotic, or anxiolytic intoxication, With moderate or severe use disorder

F13.221	Sedative, hypnotic, or anxiolytic intoxication delirium, With moderate or severe use disorder
F13.230	Sedative, hypnotic, or anxiolytic withdrawal, Without perceptual disturbances, With moderate or severe use disorder
F13.231	Sedative, hypnotic, or anxiolytic withdrawal delirium, With moderate or severe use disorder
F13.232	Sedative, hypnotic, or anxiolytic withdrawal, With perceptual disturbances, With moderate or severe use disorder
F13.24	Sedative-, hypnotic-, or anxiolytic-induced bipolar and related disorder, With moderate or severe use disorder
F13.24	Sedative-, hypnotic-, or anxiolytic-induced depressive disorder, With moderate

	or severe use disorder
F13.259	Sedative-, hypnotic-, or anxiolytic-induced psychotic disorder, With moderate or severe use disorder
F13.27	Sedative-, hypnotic-, or anxiolytic-induced major neurocognitive disorder, With moderate or severe use disorder
F13.280	Sedative-, hypnotic-, or anxiolytic-induced anxiety disorder, With moderate or severe use disorder
F13.281	Sedative-, hypnotic-, or anxiolytic-induced sexual dysfunction, With moderate or severe use disorder
F13.282	Sedative-, hypnotic-, or anxiolytic-induced sleep disorder, With moderate or severe use disorder
F13.288	Sedative-, hypnotic-, or anxiolytic-induced mild neurocognitive disorder, With moderate or severe use disorder
F13.920	Sedative, hypnotic, or anxiolytic intoxication, Without use disorder
F13.921	Sedative-, hypnotic-, or anxiolytic-induced delirium (sedative, hypnotic, or anxiolytic medication taken as prescribed)
F13.921	Sedative, hypnotic, or anxiolytic intoxication delirium, Without use disorder
F13.930	Sedative, hypnotic, or anxiolytic withdrawal, Without perceptual disturbances, Without use disorder
F13.931	Sedative, hypnotic, or anxiolytic–induced delirium (during withdrawal from sedative, hypnotic, or anxiolytic medication taken as prescribed)
F13.931	Sedative, hypnotic, or anxiolytic withdrawal delirium, Without use disorder
F13.932	Sedative, hypnotic, or anxiolytic withdrawal, With perceptual disturbances, Without use disorder
F13.94	Sedative-, hypnotic-, or anxiolytic-induced bipolar and related disorder, Without use disorder
F13.94	Sedative-, hypnotic-, or anxiolytic-induced depressive disorder, Without use disorder
F13.959	Sedative-, hypnotic-, or anxiolytic-induced psychotic disorder, Without use disorder
F13.97	Sedative-, hypnotic-, or anxiolytic-induced major neurocognitive disorder, Without use disorder
F13.980	Sedative-, hypnotic-, or anxiolytic-induced anxiety disorder, Without use disorder
F13.981	Sedative-, hypnotic-, or anxiolytic-induced sexual dysfunction, Without use disorder

F13.982	Sedative-, hypnotic-, or anxiolytic-induced sleep disorder, Without use disorder
F13.988	Sedative-, hypnotic-, or anxiolytic-induced mild neurocognitive disorder, Without use disorder

F13.99	Unspecified sedative-, hypnotic-, or anxiolytic-related disorder
F14.10	Cocaine use disorder, Mild
F14.11	Cocaine use disorder, Mild, In early remission
F14.11	Cocaine use disorder, Mild, In sustained remission
F14.120	Cocaine intoxication, Without perceptual disturbances, With mild use disorder
F14.121	Cocaine intoxication delirium, With mild use disorder
F14.122	Cocaine intoxication, With perceptual disturbances, With mild use disorder
F14.13	Cocaine withdrawal, With mild use disorder
F14.14	Cocaine-induced bipolar and related disorder, With mild use disorder
F14.14	Cocaine-induced depressive disorder, With mild use disorder
F14.159	Cocaine-induced psychotic disorder, With mild use disorder
F14.180	Cocaine-induced anxiety disorder, With mild use disorder
F14.181	Cocaine-induced sexual dysfunction, With mild use disorder
F14.182	Cocaine-induced sleep disorder, With mild use disorder
F14.188	Cocaine-induced mild neurocognitive disorder, With mild use disorder
F14.188	Cocaine-induced obsessive-compulsive and related disorder, With mild use disorder
F14.20	Cocaine use disorder, Moderate
F14.20	Cocaine use disorder, Severe
F14.21	Cocaine use disorder, Moderate, In early remission
F14.21	Cocaine use disorder, Moderate, In sustained remission
F14.21	Cocaine use disorder, Severe, In early remission
F14.21	Cocaine use disorder, Severe, In sustained remission
F14.220	Cocaine intoxication, Without perceptual disturbances, With moderate or severe use disorder
F14.221	Cocaine intoxication delirium, With moderate or severe use disorder
F14.222	Cocaine intoxication, With perceptual disturbances, With moderate or severe use disorder
F14.23	Cocaine withdrawal, With moderate or severe use disorder
F14.24	Cocaine-induced bipolar and related disorder, With moderate or severe use disorder
F14.24	Cocaine-induced depressive disorder, With moderate or severe use disorder
F14.259	Cocaine-induced psychotic disorder, With moderate or severe use disorder
F14.280	Cocaine-induced anxiety disorder, With moderate or severe use disorder
F14.281	Cocaine-induced sexual dysfunction, With moderate or severe use disorder
F14.282	Cocaine-induced sleep disorder, With moderate or severe use disorder
F14.288	Cocaine-induced mild neurocognitive disorder, With moderate or severe use disorder
F14.288	Cocaine-induced obsessive-compulsive and related disorder, With moderate or

	severe use disorder
F14.920	Cocaine intoxication, Without perceptual disturbances, Without use disorder

F14.921	Cocaine intoxication delirium, Without use disorder
F14.922	Cocaine intoxication, With perceptual disturbances, Without use disorder
F14.93	Cocaine withdrawal, Without use disorder
F14.94	Cocaine-induced bipolar and related disorder, Without use disorder
F14.94	Cocaine-induced depressive disorder, Without use disorder
F14.959	Cocaine-induced psychotic disorder, Without use disorder
F14.980	Cocaine-induced anxiety disorder, Without use disorder
F14.981	Cocaine-induced sexual dysfunction, Without use disorder
F14.982	Cocaine-induced sleep disorder, Without use disorder
F14.988	Cocaine-induced mild neurocognitive disorder, Without use disorder
F14.988	Cocaine-induced obsessive-compulsive and related disorder, Without use disorder
F14.99	Unspecified cocaine-related disorder
F15.10	Amphetamine-type substance use disorder, Mild
F15.10	Other or unspecified stimulant use disorder, Mild
F15.11	Amphetamine-type substance use disorder, Mild, In early remission
F15.11	Amphetamine-type substance use disorder, Mild, In sustained remission
F15.11	Other or unspecified stimulant use disorder, Mild, In early remission
F15.11	Other or unspecified stimulant use disorder, Mild, In sustained remission
F15.120	Amphetamine-type substance intoxication, Without perceptual disturbances, With mild use disorder
F15.120	Other stimulant intoxication, Without perceptual disturbances, With mild use disorder
F15.121	Amphetamine-type substance (or other stimulant) intoxication delirium, With mild use disorder
F15.122	Amphetamine-type substance intoxication, With perceptual disturbances, With mild use disorder
F15.122	Other stimulant intoxication, With perceptual disturbances, With mild use disorder
F15.13	Amphetamine-type substance withdrawal, With mild use disorder
F15.13	Other stimulant withdrawal, With mild use disorder
F15.14	Amphetamine-type substance (or other stimulant)–induced bipolar and related disorder, With mild use disorder
F15.14	Amphetamine-type substance (or other stimulant)–induced depressive disorder, With mild use disorder

F15.159	Amphetamine-type substance (or other stimulant)–induced psychotic disorder, With mild use disorder
F15.180	Amphetamine-type substance (or other stimulant)–induced anxiety disorder, With mild use disorder
F15.181	Amphetamine-type substance (or other stimulant)–induced sexual dysfunction, With mild use disorder
F15.182	Amphetamine-type substance (or other stimulant)–induced sleep disorder, With mild use disorder
F15.188	Amphetamine-type substance (or other stimulant)–induced mild neurocognitive disorder, With mild use disorder
F15.188	Amphetamine-type substance (or other stimulant)–induced obsessive-compulsive and related disorder, With mild use disorder

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F15.20	Amphetamine-type substance use disorder, Moderate
F15.20	Amphetamine-type substance use disorder, Severe
F15.20	Other or unspecified stimulant use disorder, Moderate
F15.20	Other or unspecified stimulant use disorder, Severe
F15.21	Amphetamine-type substance use disorder, Moderate, In early remission
F15.21	Amphetamine-type substance use disorder, Moderate, In sustained remission
F15.21	Amphetamine-type substance use disorder, Severe, In early remission
F15.21	Amphetamine-type substance use disorder, Severe, In sustained remission
F15.21	Other or unspecified stimulant use disorder, Moderate, In early remission
F15.21	Other or unspecified stimulant use disorder, Moderate, In sustained remission
F15.21	Other or unspecified stimulant use disorder, Severe, In early remission
F15.21	Other or unspecified stimulant use disorder, Severe, In sustained remission
F15.220	Amphetamine-type substance intoxication, Without perceptual disturbances, With moderate or severe use disorder
F15.220	Other stimulant intoxication, Without perceptual disturbances, With moderate or severe use disorder
F15.221	Amphetamine-type substance (or other stimulant) intoxication delirium, With moderate or severe use disorder
F15.222	Amphetamine-type substance intoxication, With perceptual disturbances, With moderate or severe use disorder
F15.222	Other stimulant intoxication, With perceptual disturbances, With moderate or severe use disorder
F15.23	Amphetamine-type substance withdrawal, With moderate or severe use disorder
F15.23	Other stimulant withdrawal, With moderate or severe use disorder
F15.24	Amphetamine-type substance (or other stimulant)–induced bipolar and related disorder, With moderate or severe use disorder

F15.24	Amphetamine-type substance (or other stimulant)–induced depressive disorder, With moderate or severe use disorder
F15.259	Amphetamine-type substance (or other stimulant)–induced psychotic disorder, With moderate or severe use disorder
F15.280	Amphetamine-type substance (or other stimulant)–induced anxiety disorder, With moderate or severe use disorder
F15.281	Amphetamine-type substance (or other stimulant)–induced sexual dysfunction, With moderate or severe use disorder
F15.282	Amphetamine-type substance (or other stimulant)–induced sleep disorder, With moderate or severe use disorder
F15.288	Amphetamine-type substance (or other stimulant)–induced mild neurocognitive disorder, With moderate or severe use disorder
F15.288	Amphetamine-type substance (or other stimulant)–induced obsessive-compulsive and related disorder, With moderate or severe use disorder
F15.920	Amphetamine-type substance intoxication, Without perceptual disturbances, Without use disorder
F15.920	Caffeine intoxication
F15.920	Other stimulant intoxication, Without perceptual disturbances, Without use disorder

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F15.921	Amphetamine-type (or other stimulant) medication–induced delirium (amphetamine-type or other stimulant medication taken as prescribed)
F15.921	Amphetamine-type substance (or other stimulant) intoxication delirium, Without use disorder
F15.922	Amphetamine-type substance intoxication, With perceptual disturbances, Without use disorder
F15.922	Other stimulant intoxication, With perceptual disturbances, Without use disorder
F15.93	Amphetamine-type substance withdrawal, Without use disorder
F15.93	Caffeine withdrawal
F15.93	Other stimulant withdrawal, Without use disorder
F15.94	Amphetamine-type substance (or other stimulant)–induced bipolar and related disorder, Without use disorder
F15.94	Amphetamine-type substance (or other stimulant)–induced depressive disorder, Without use disorder
F15.959	Amphetamine-type substance (or other stimulant)–induced psychotic disorder, Without use disorder
F15.980	Amphetamine-type substance (or other stimulant)–induced anxiety disorder, Without use disorder
F15.980	Caffeine-induced anxiety disorder, Without use disorder

F15.981	Amphetamine-type substance (or other stimulant)–induced sexual dysfunction, Without use disorder
F15.982	Amphetamine-type substance (or other stimulant)–induced sleep disorder, Without use disorder
F15.982	Caffeine-induced sleep disorder, Without use disorder
F15.988	Amphetamine-type substance (or other stimulant)–induced mild neurocognitive disorder, Without use disorder
F15.988	Amphetamine-type substance (or other stimulant)–induced obsessive-compulsive and related disorder, Without use disorder
F15.99	Unspecified amphetamine-type substance-related disorder
F15.99	Unspecified caffeine-related disorder
F15.99	Unspecified other stimulant–related disorder
F16.10	Other hallucinogen use disorder, Mild
F16.10	Phencyclidine use disorder, Mild
F16.11	Other hallucinogen use disorder, Mild, In early remission
F16.11	Other hallucinogen use disorder, Mild, In sustained remission
F16.11	Phencyclidine use disorder, Mild, In early remission
F16.11	Phencyclidine use disorder, Mild, In sustained remission
F16.120	Other hallucinogen intoxication, With mild use disorder
F16.120	Phencyclidine intoxication, With mild use disorder
F16.121	Other hallucinogen intoxication delirium, With mild use disorder
F16.121	Phencyclidine intoxication delirium, With mild use disorder
F16.14	Other hallucinogen–induced bipolar and related disorder, With mild use disorder
F16.14	Other hallucinogen–induced depressive disorder, With mild use disorder
F16.14	Phencyclidine-induced bipolar and related disorder, With mild use disorder

F16.14	Phencyclidine-induced depressive disorder, With mild use disorder
F16.159	Other hallucinogen–induced psychotic disorder, With mild use disorder
F16.159	Phencyclidine-induced psychotic disorder, With mild use disorder
F16.180	Other hallucinogen–induced anxiety disorder, With mild use disorder
F16.180	Phencyclidine-induced anxiety disorder, With mild use disorder
F16.20	Other hallucinogen use disorder, Moderate
F16.20	Other hallucinogen use disorder, Severe
F16.20	Phencyclidine use disorder, Moderate
F16.20	Phencyclidine use disorder, Severe
F16.21	Other hallucinogen use disorder, Moderate, In early remission

F16.21	Other hallucinogen use disorder, Moderate, In sustained remission
F16.21	Other hallucinogen use disorder, Severe, In early remission
F16.21	Other hallucinogen use disorder, Severe, In sustained remission
F16.21	Phencyclidine use disorder, Moderate, In early remission
F16.21	Phencyclidine use disorder, Moderate, In sustained remission
F16.21	Phencyclidine use disorder, Severe, In early remission
F16.21	Phencyclidine use disorder, Severe, In sustained remission
F16.220	Other hallucinogen intoxication, With moderate or severe use disorder
F16.220	Phencyclidine intoxication, With moderate or severe use disorder
F16.221	Other hallucinogen intoxication delirium, With moderate or severe use disorder
F16.221	Phencyclidine intoxication delirium, With moderate or severe use disorder
F16.24	Other hallucinogen–induced bipolar and related disorder, With moderate or severe use disorder
F16.24	Other hallucinogen–induced depressive disorder, With moderate or severe use disorder
F16.24	Phencyclidine-induced bipolar and related disorder, With moderate or severe use disorder
F16.24	Phencyclidine-induced depressive disorder, With moderate or severe use disorder
F16.259	Other hallucinogen–induced psychotic disorder, With moderate or severe use disorder
F16.259	Phencyclidine-induced psychotic disorder, With moderate or severe use disorder
F16.280	Other hallucinogen–induced anxiety disorder, With moderate or severe use disorder
F16.280	Phencyclidine-induced anxiety disorder, With moderate or severe use disorder
F16.920	Other hallucinogen intoxication, Without use disorder
F16.920	Phencyclidine intoxication, Without use disorder
F16.921	Ketamine or other hallucinogen–induced delirium (ketamine or other hallucinogen medication taken as prescribed or for medical reasons)
F16.921	Other hallucinogen intoxication delirium, Without use disorder
F16.921	Phencyclidine intoxication delirium, Without use disorder
F16.94	Other hallucinogen–induced bipolar and related disorder, Without use disorder

F16.94	Other hallucinogen–induced depressive disorder, Without use disorder
F16.94	Phencyclidine-induced bipolar and related disorder, Without use disorder
F16.94	Phencyclidine-induced depressive disorder, Without use disorder

F16.959	Other hallucinogen–induced psychotic disorder, Without use disorder
F16.959	Phencyclidine-induced psychotic disorder, Without use disorder
F16.980	Other hallucinogen–induced anxiety disorder, Without use disorder
F16.980	Phencyclidine-induced anxiety disorder, Without use disorder
F16.983	Hallucinogen persisting perception disorder
F16.99	Unspecified hallucinogen-related disorder
F16.99	Unspecified phencyclidine-related disorder
F17.200	Tobacco use disorder, Moderate
F17.200	Tobacco use disorder, Severe
F17.201	Tobacco use disorder, Moderate, In early remission
F17.201	Tobacco use disorder, Moderate, In sustained remission
F17.201	Tobacco use disorder, Severe, In early remission
F17.201	Tobacco use disorder, Severe, In sustained remission
F17.203	Tobacco withdrawal
F17.208	Tobacco-induced sleep disorder, With moderate or severe use disorder
F17.209	Unspecified tobacco-related disorder
F18.10	Inhalant use disorder, Mild
F18.11	Inhalant use disorder, Mild, In early remission
F18.11	Inhalant use disorder, Mild, In sustained remission
F18.120	Inhalant intoxication, With mild use disorder
F18.121	Inhalant intoxication delirium, With mild use disorder
F18.14	Inhalant-induced depressive disorder, With mild use disorder
F18.159	Inhalant-induced psychotic disorder, With mild use disorder
F18.17	Inhalant-induced major neurocognitive disorder, With mild use disorder
F18.180	Inhalant-induced anxiety disorder, With mild use disorder
F18.188	Inhalant-induced mild neurocognitive disorder, With mild use disorder
F18.20	Inhalant use disorder, Moderate
F18.20	Inhalant use disorder, Severe
F18.21	Inhalant use disorder, Moderate, In early remission
F18.21	Inhalant use disorder, Moderate, In sustained remission
F18.21	Inhalant use disorder, Severe, In early remission
F18.21	Inhalant use disorder, Severe, In sustained remission
F18.220	Inhalant intoxication, With moderate or severe use disorder
F18.221	Inhalant intoxication delirium, With moderate or severe use disorder
F18.24	Inhalant-induced depressive disorder, With moderate or severe use disorder
F18.259	Inhalant-induced psychotic disorder, With moderate or severe use disorder
F18.27	Inhalant-induced major neurocognitive disorder, With moderate or severe use disorder

F18.280	Inhalant-induced anxiety disorder, With moderate or severe use disorder
F18.288	Inhalant-induced mild neurocognitive disorder, With moderate or severe use disorder
F18.920	Inhalant intoxication, Without use disorder

F18.921	Inhalant intoxication delirium, Without use disorder
F18.94	Inhalant-induced depressive disorder, Without use disorder
F18.959	Inhalant-induced psychotic disorder, Without use disorder
F18.97	Inhalant-induced major neurocognitive disorder, Without use disorder
F18.980	Inhalant-induced anxiety disorder, Without use disorder
F18.988	Inhalant-induced mild neurocognitive disorder, Without use disorder
F18.99	Unspecified inhalant-related disorder
F19.10	Other (or unknown) substance use disorder, Mild
F19.11	Other (or unknown) substance use disorder, Mild, In early remission
F19.11	Other (or unknown) substance use disorder, Mild, In sustained remission
F19.120	Other (or unknown) substance intoxication, Without perceptual disturbances, With mild use disorder
F19.121	Other (or unknown) substance intoxication delirium, With mild use disorder
F19.122	Other (or unknown) substance intoxication, With perceptual disturbances, With mild use disorder
F19.130	Other (or unknown) substance withdrawal, Without perceptual disturbances, With mild use disorder
F19.131	Other (or unknown) substance withdrawal delirium, With mild use disorder
F19.132	Other (or unknown) substance withdrawal, With perceptual disturbances, With mild use disorder
F19.14	Other (or unknown) substance–induced bipolar and related disorder, With mild use disorder
F19.14	Other (or unknown) substance–induced depressive disorder, With mild use disorder
F19.159	Other (or unknown) substance–induced psychotic disorder, With mild use disorder
F19.17	Other (or unknown) substance–induced major neurocognitive disorder, With mild use disorder
F19.180	Other (or unknown) substance–induced anxiety disorder, With mild use disorder
F19.181	Other (or unknown) substance–induced sexual dysfunction, With mild use disorder
F19.182	Other (or unknown) substance–induced sleep disorder, With mild use disorder
F19.188	Other (or unknown) substance–induced mild neurocognitive disorder, With mild

use disorder

F19.188 Other (or unknown) substance–induced obsessive-compulsive and related disorder, With mild use disorder

F19.20 Other (or unknown) substance use disorder, Moderate

F19.20 Other (or unknown) substance use disorder, Severe

F19.21 Other (or unknown) substance use disorder, Moderate, In early remission

F19.21 Other (or unknown) substance use disorder, Moderate, In sustained remission

F19.21 Other (or unknown) substance use disorder, Severe, In early remission

F19.21 Other (or unknown) substance use disorder, Severe, In sustained remission

F19.220 Other (or unknown) substance intoxication, Without perceptual disturbances, With moderate or severe use disorder

F19.221 Other (or unknown) substance intoxication delirium, With moderate or severe use disorder

F19.222 Other (or unknown) substance intoxication, With perceptual disturbances, With moderate or severe use disorder

F19.230 Other (or unknown) substance withdrawal, Without perceptual disturbances, With moderate or severe use disorder

F19.231 Other (or unknown) substance withdrawal delirium, With moderate or severe use disorder

F19.232 Other (or unknown) substance withdrawal, With perceptual disturbances, With moderate or severe use disorder

F19.24 Other (or unknown) substance–induced bipolar and related disorder, With moderate or severe use disorder

F19.24 Other (or unknown) substance–induced depressive disorder, With moderate or severe use disorder

F19.259 Other (or unknown) substance–induced psychotic disorder, With moderate or severe use disorder

F19.27 Other (or unknown) substance–induced major neurocognitive disorder, With moderate or severe use disorder

F19.280 Other (or unknown) substance–induced anxiety disorder, With moderate or severe use disorder

F19.281 Other (or unknown) substance–induced sexual dysfunction, With moderate or severe use disorder

F19.282 Other (or unknown) substance–induced sleep disorder, With moderate or severe use disorder

F19.288 Other (or unknown) substance–induced mild neurocognitive disorder, With moderate or severe use disorder

F19.288 Other (or unknown) substance–induced obsessive-compulsive and related disorder, With moderate or severe use disorder

F19.920	Other (or unknown) substance intoxication, Without perceptual disturbances, Without use disorder
F19.921	Other (or unknown) medication–induced delirium (other [or unknown] medication taken as prescribed)
F19.921	Other (or unknown) substance intoxication delirium, Without use disorder
F19.922	Other (or unknown) substance intoxication, With perceptual disturbances, Without use disorder
F19.930	Other (or unknown) substance withdrawal, Without perceptual disturbances, Without use disorder
F19.931	Other (or unknown) medication–induced delirium (during withdrawal from other [or unknown] medication taken as prescribed)
F19.931	Other (or unknown) substance withdrawal delirium, Without use disorder
F19.932	Other (or unknown) substance withdrawal, With perceptual disturbances, Without use disorder
F19.94	Other (or unknown) substance–induced bipolar and related disorder, Without use disorder
F19.94	Other (or unknown) substance–induced depressive disorder, Without use disorder
F19.959	Other (or unknown) substance–induced psychotic disorder, Without use disorder

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F19.97	Other (or unknown) substance–induced major neurocognitive disorder, Without use disorder
F19.980	Other (or unknown) substance–induced anxiety disorder, Without use disorder
F19.981	Other (or unknown) substance–induced sexual dysfunction, Without use disorder
F19.982	Other (or unknown) substance–induced sleep disorder, Without use disorder
F19.988	Other (or unknown) substance–induced mild neurocognitive disorder, Without use disorder
F19.988	Other (or unknown) substance–induced obsessive-compulsive and related disorder, Without use disorder
F19.99	Unspecified other (or unknown) substance–related disorder
F20.81	Schizophreniform disorder
F20.9	Schizophrenia
F21	Schizotypal personality disorder
F22	Delusional disorder
F23	Brief psychotic disorder
F25.0	Schizoaffective disorder, Bipolar type
F25.1	Schizoaffective disorder, Depressive type
F28	Other specified schizophrenia spectrum and other psychotic disorder

F29	Unspecified schizophrenia spectrum and other psychotic disorder
F31.0	Bipolar I disorder, Current or most recent episode hypomanic
F31.11	Bipolar I disorder, Current or most recent episode manic, Mild
F31.12	Bipolar I disorder, Current or most recent episode manic, Moderate
F31.13	Bipolar I disorder, Current or most recent episode manic, Severe
F31.2	Bipolar I disorder, Current or most recent episode manic, With psychotic features
F31.31	Bipolar I disorder, Current or most recent episode depressed, Mild
F31.32	Bipolar I disorder, Current or most recent episode depressed, Moderate
F31.4	Bipolar I disorder, Current or most recent episode depressed, Severe
F31.5	Bipolar I disorder, Current or most recent episode depressed, With psychotic features
F31.71	Bipolar I disorder, Current or most recent episode hypomanic, In partial remission
F31.72	Bipolar I disorder, Current or most recent episode hypomanic, In full remission
F31.73	Bipolar I disorder, Current or most recent episode manic, In partial remission
F31.74	Bipolar I disorder, Current or most recent episode manic, In full remission
F31.75	Bipolar I disorder, Current or most recent episode depressed, In partial remission
F31.76	Bipolar I disorder, Current or most recent episode depressed, In full remission
F31.81	Bipolar II disorder
F31.89	Other specified bipolar and related disorder
F31.9	Bipolar I disorder, Current or most recent episode depressed, Unspecified
F31.9	Bipolar I disorder, Current or most recent episode hypomanic, Unspecified
F31.9	Bipolar I disorder, Current or most recent episode manic, Unspecified

F31.9	Bipolar I disorder, Current or most recent episode unspecified
F31.9	Unspecified bipolar and related disorder
F32.0	Major depressive disorder, Single episode, Mild
F32.1	Major depressive disorder, Single episode, Moderate
F32.2	Major depressive disorder, Single episode, Severe
F32.3	Major depressive disorder, Single episode, With psychotic features
F32.4	Major depressive disorder, Single episode, In partial remission
F32.5	Major depressive disorder, Single episode, In full remission
F32.81	Premenstrual dysphoric disorder
F32.89	Other specified depressive disorder

F32.9	Major depressive disorder, Single episode, Unspecified
F32.A	Unspecified depressive disorder
F33.0	Major depressive disorder, Recurrent episode, Mild
F33.1	Major depressive disorder, Recurrent episode, Moderate
F33.2	Major depressive disorder, Recurrent episode, Severe
F33.3	Major depressive disorder, Recurrent episode, With psychotic features
F33.41	Major depressive disorder, Recurrent episode, In partial remission
F33.42	Major depressive disorder, Recurrent episode, In full remission
F33.9	Major depressive disorder, Recurrent episode, Unspecified
F34.0	Cyclothymic disorder
F34.1	Persistent depressive disorder
F34.81	Disruptive mood dysregulation disorder
F39	Unspecified mood disorder
F40.00	Agoraphobia
F40.10	Social anxiety disorder
F40.218	Specific phobia, Animal
F40.228	Specific phobia, Natural environment
F40.230	Specific phobia, Fear of blood
F40.231	Specific phobia, Fear of injections and transfusions
F40.232	Specific phobia, Fear of other medical care
F40.233	Specific phobia, Fear of injury
F40.248	Specific phobia, Situational
F40.298	Specific phobia, Other
F41.0	Panic disorder
F41.1	Generalized anxiety disorder
F41.8	Other specified anxiety disorder
F41.9	Unspecified anxiety disorder
F42.2	Obsessive-compulsive disorder
F42.3	Hoarding disorder
F42.4	Excoriation (skin-picking) disorder
F42.8	Other specified obsessive-compulsive and related disorder
F42.9	Unspecified obsessive-compulsive and related disorder
F43.0	Acute stress disorder
F43.10	Posttraumatic stress disorder

F43.20	Adjustment disorders, Unspecified
F43.21	Adjustment disorders, With depressed mood
F43.22	Adjustment disorders, With anxiety
F43.23	Adjustment disorders, With mixed anxiety and depressed mood
F43.24	Adjustment disorders, With disturbance of conduct
F43.25	Adjustment disorders, With mixed disturbance of emotions and conduct
F43.8	Other specified trauma- and stressor-related disorder
F43.8	Prolonged grief disorder
F43.9	Unspecified trauma- and stressor-related disorder
F44.0	Dissociative amnesia
F44.1	Dissociative amnesia, With dissociative fugue
F44.4	Functional neurological symptom disorder (conversion disorder), With abnormal movement
F44.4	Functional neurological symptom disorder (conversion disorder), With speech symptom
F44.4	Functional neurological symptom disorder (conversion disorder), With swallowing symptoms
F44.4	Functional neurological symptom disorder (conversion disorder), With weakness/paralysis
F44.5	Functional neurological symptom disorder (conversion disorder), With attacks or seizures
F44.6	Functional neurological symptom disorder (conversion disorder), With anesthesia or sensory loss
F44.6	Functional neurological symptom disorder (conversion disorder), With special sensory symptom
F44.7	Functional neurological symptom disorder (conversion disorder), With mixed symptoms
F44.81	Dissociative identity disorder
F44.89	Other specified dissociative disorder
F44.9	Unspecified dissociative disorder
F45.1	Somatic symptom disorder
F45.21	Illness anxiety disorder
F45.22	Body dysmorphic disorder
F45.8	Other specified somatic symptom and related disorder
F45.9	Unspecified somatic symptom and related disorder
F48.1	Depersonalization/derealization disorder
F50.01	Anorexia nervosa, Restricting type
F50.02	Anorexia nervosa, Binge-eating/purging type
F50.2	Bulimia nervosa
F50.81	Binge-eating disorder

F50.82	Avoidant/restrictive food intake disorder
F50.89	Other specified feeding or eating disorder
F50.89	Pica, in adults
F50.9	Unspecified feeding or eating disorder
F51.01	Insomnia disorder
F51.11	Hypersomnolence disorder

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F51.3	Non–rapid eye movement sleep arousal disorders, Sleepwalking type
F51.4	Non–rapid eye movement sleep arousal disorders, Sleep terror type
F51.5	Nightmare disorder
F52.0	Male hypoactive sexual desire disorder
F52.21	Erectile disorder
F52.22	Female sexual interest/arousal disorder
F52.31	Female orgasmic disorder
F52.32	Delayed ejaculation
F52.4	Premature (early) ejaculation

F52.6	Genito-pelvic pain/penetration disorder
F52.8	Other specified sexual dysfunction
F52.9	Unspecified sexual dysfunction
F54	Psychological factors affecting other medical conditions
F60.0	Paranoid personality disorder
F60.1	Schizoid personality disorder
F60.2	Antisocial personality disorder
F60.3	Borderline personality disorder
F60.4	Histrionic personality disorder
F60.5	Obsessive-compulsive personality disorder
F60.6	Avoidant personality disorder
F60.7	Dependent personality disorder
F60.81	Narcissistic personality disorder
F60.89	Other specified personality disorder
F60.9	Unspecified personality disorder
F63.0	Gambling disorder
F63.1	Pyromania
F63.2	Kleptomania

F63.3	Trichotillomania (hair-pulling disorder)
F63.81	Intermittent explosive disorder
F64.0	Gender dysphoria in adolescents and adults
F64.2	Gender dysphoria in children
F64.8	Other specified gender dysphoria
F64.9	Unspecified gender dysphoria
F65.0	Fetishistic disorder
F65.1	Transvestic disorder
F65.2	Exhibitionistic disorder
F65.3	Voyeuristic disorder
F65.4	Pedophilic disorder
F65.51	Sexual masochism disorder
F65.52	Sexual sadism disorder
F65.81	Frotteuristic disorder
F65.89	Other specified paraphilic disorder
F65.9	Unspecified paraphilic disorder
F68.10	Factitious disorder imposed on self

F68.A	Factitious disorder imposed on another
F70	Intellectual developmental disorder (intellectual disability), Mild
F71	Intellectual developmental disorder (intellectual disability), Moderate
F72	Intellectual developmental disorder (intellectual disability), Severe
F73	Intellectual developmental disorder (intellectual disability), Profound
F79	Unspecified intellectual developmental disorder (intellectual disability)
F80.0	Speech sound disorder
F80.2	Language disorder
F80.81	Childhood-onset fluency disorder (stuttering)
F80.82	Social (pragmatic) communication disorder
F80.9	Unspecified communication disorder
F81.0	Specific learning disorder, With impairment in reading
F81.2	Specific learning disorder, With impairment in mathematics
F81.81	Specific learning disorder, With impairment in written expression
F82	Developmental coordination disorder
F84.0	Autism spectrum disorder
F88	Global developmental delay

F88	Other specified neurodevelopmental disorder
F89	Unspecified neurodevelopmental disorder
F90.0	Attention-deficit/hyperactivity disorder, Predominantly inattentive presentation
F90.1	Attention-deficit/hyperactivity disorder, Predominantly hyperactive/impulsive presentation
F90.2	Attention-deficit/hyperactivity disorder, Combined presentation
F90.8	Other specified attention-deficit/hyperactivity disorder
F90.9	Unspecified attention-deficit/hyperactivity disorder
F91.1	Conduct disorder, Childhood-onset type
F91.2	Conduct disorder, Adolescent-onset type
F91.3	Oppositional defiant disorder
F91.8	Other specified disruptive, impulse-control, and conduct disorder
F91.9	Conduct disorder, Unspecified onset
F91.9	Unspecified disruptive, impulse-control, and conduct disorder
F93.0	Separation anxiety disorder
F94.0	Selective mutism
F94.1	Reactive attachment disorder
F94.2	Disinhibited social engagement disorder
F95.0	Provisional tic disorder
F95.1	Persistent (chronic) motor or vocal tic disorder
F95.2	Tourette’s disorder
F95.8	Other specified tic disorder
F95.9	Unspecified tic disorder
F98.0	Enuresis
F98.1	Encopresis
F98.21	Rumination disorder
F98.3	Pica, in children

F98.4	Stereotypic movement disorder
F98.5	Adult-onset fluency disorder
F99	Other specified mental disorder
F99	Unspecified mental disorder
G21.0	Neuroleptic malignant syndrome
G21.11	Antipsychotic medication– and other dopamine receptor blocking agent–induced parkinsonism
G21.19	Other medication-induced parkinsonism

G24.01	Tardive dyskinesia
G24.02	Medication-induced acute dystonia
G24.09	Tardive dystonia
G25.1	Medication-induced postural tremor
G25.71	Medication-induced acute akathisia
G25.71	Tardive akathisia
G25.79	Other medication-induced movement disorder
G25.81	Restless legs syndrome
G31.84	Mild frontotemporal neurocognitive disorder
G31.84	Mild neurocognitive disorder due to Alzheimer’s disease
G31.84	Mild neurocognitive disorder due to another medical condition
G31.84	Mild neurocognitive disorder due to HIV infection
G31.84	Mild neurocognitive disorder due to Huntington’s disease
G31.84	Mild neurocognitive disorder with Lewy bodies
G31.84	Mild neurocognitive disorder due to multiple etiologies
G31.84	Mild neurocognitive disorder due to Parkinson’s disease
G31.84	Mild neurocognitive disorder due to prion disease
G31.84	Mild neurocognitive disorder due to traumatic brain injury
G31.84	Mild vascular neurocognitive disorder
G47.00	Unspecified insomnia disorder
G47.09	Other specified insomnia disorder
G47.10	Unspecified hypersomnolence disorder
G47.19	Other specified hypersomnolence disorder
G47.20	Circadian rhythm sleep-wake disorders, Unspecified type
G47.21	Circadian rhythm sleep-wake disorders, Delayed sleep phase type
G47.22	Circadian rhythm sleep-wake disorders, Advanced sleep phase type
G47.23	Circadian rhythm sleep-wake disorders, Irregular sleep-wake type
G47.24	Circadian rhythm sleep-wake disorders, Non-24-hour sleep-wake type
G47.26	Circadian rhythm sleep-wake disorders, Shift work type
G47.31	Central sleep apnea, Idiopathic central sleep apnea
G47.33	Obstructive sleep apnea hypopnea
G47.34	Sleep-related hypoventilation, Idiopathic hypoventilation
G47.35	Sleep-related hypoventilation, Congenital central alveolar hypoventilation
G47.36	Sleep-related hypoventilation, Comorbid sleep-related hypoventilation
G47.37	Central sleep apnea comorbid with opioid use
G47.411	Narcolepsy with cataplexy or hypocretin deficiency (type 1)

G47.419	Narcolepsy without cataplexy and either without hypocretin deficiency or hypocretin unmeasured (type 2)
G47.421	Narcolepsy with cataplexy or hypocretin deficiency due to a medical condition
G47.429	Narcolepsy without cataplexy and without hypocretin deficiency due to a medical condition
G47.52	Rapid eye movement sleep behavior disorder
G47.8	Other specified sleep-wake disorder
G47.9	Unspecified sleep-wake disorder
N39.498	Other specified elimination disorder, With urinary symptoms
R06.3	Central sleep apnea, Cheyne-Stokes breathing
R15.9	Other specified elimination disorder, With fecal symptoms
R15.9	Unspecified elimination disorder, With fecal symptoms
R32	Unspecified elimination disorder, With urinary symptoms
R41.0	Other specified delirium
R41.0	Unspecified delirium
R41.81	Age-related cognitive decline
R41.83	Borderline intellectual functioning
R41.9	Unspecified neurocognitive disorder
R45.88	Current nonsuicidal self-injury
	Current suicidal behavior
T14.91A	Initial encounter
T14.91D	Subsequent encounter
T43.205A	Antidepressant discontinuation syndrome, Initial encounter
T43.205D	Antidepressant discontinuation syndrome, Subsequent encounter
T43.205S	Antidepressant discontinuation syndrome, Sequelae
T50.905A	Other adverse effect of medication, Initial encounter
T50.905D	Other adverse effect of medication, Subsequent encounter
T50.905S	Other adverse effect of medication, Sequelae
T74.01XA	Spouse or partner neglect, Confirmed, Initial encounter
T74.01XD	Spouse or partner neglect, Confirmed, Subsequent encounter
T74.02XA	Child neglect, Confirmed, Initial encounter
T74.02XD	Child neglect, Confirmed, Subsequent encounter
T74.11XA	Adult physical abuse by nonspouse or nonpartner, Confirmed, Initial encounter
T74.11XA	Spouse or partner violence, Physical, Confirmed, Initial encounter
T74.11XD	Adult physical abuse by nonspouse or nonpartner, Confirmed, Subsequent encounter

T74.11XD	Spouse or partner violence, Physical, Confirmed, Subsequent encounter
T74.12XA	Child physical abuse, Confirmed, Initial encounter
T74.12XD	Child physical abuse, Confirmed, Subsequent encounter
T74.21XA	Adult sexual abuse by nonspouse or nonpartner, Confirmed, Initial encounter
T74.21XA	Spouse or partner violence, Sexual, Confirmed, Initial encounter
T74.21XD	Adult sexual abuse by nonspouse or nonpartner, Confirmed, Subsequent encounter

T74.21XD	Spouse or partner violence, Sexual, Confirmed, Subsequent encounter
T74.22XA	Child sexual abuse, Confirmed, Initial encounter
T74.22XD	Child sexual abuse, Confirmed, Subsequent encounter
T74.31XA	Adult psychological abuse by nonspouse or nonpartner, Confirmed, Initial encounter
T74.31XA	Spouse or partner abuse, Psychological, Confirmed, Initial encounter
T74.31XD	Adult psychological abuse by nonspouse or nonpartner, Confirmed, Subsequent encounter
T74.31XD	Spouse or partner abuse, Psychological, Confirmed, Subsequent encounter
T74.32XA	Child psychological abuse, Confirmed, Initial encounter
T74.32XD	Child psychological abuse, Confirmed, Subsequent encounter
T76.01XA	Spouse or partner neglect, Suspected, Initial encounter
T76.01XD	Spouse or partner neglect, Suspected, Subsequent encounter
T76.02XA	Child neglect, Suspected, Initial encounter
T76.02XD	Child neglect, Suspected, Subsequent encounter
T76.11XA	Adult physical abuse by nonspouse or nonpartner, Suspected, Initial encounter
T76.11XA	Spouse or partner violence, Physical, Suspected, Initial encounter
T76.11XD	Adult physical abuse by nonspouse or nonpartner, Suspected, Subsequent encounter
T76.11XD	Spouse or partner violence, Physical, Suspected, Subsequent encounter

T76.12XA	Child physical abuse, Suspected, Initial encounter
T76.12XD	Child physical abuse, Suspected, Subsequent encounter
T76.21XA	Adult sexual abuse by nonspouse or nonpartner, Suspected, Initial encounter
T76.21XA	Spouse or partner violence, Sexual, Suspected, Initial encounter
T76.21XD	Adult sexual abuse by nonspouse or nonpartner, Suspected, Subsequent encounter
T76.21XD	Spouse or partner violence, Sexual, Suspected, Subsequent encounter

T76.22XA	Child sexual abuse, Suspected, Initial encounter
T76.22XD	Child sexual abuse, Suspected, Subsequent encounter
T76.31XA	Adult psychological abuse by nonspouse or nonpartner, Suspected, Initial encounter
T76.31XA	Spouse or partner abuse, Psychological, Suspected, Initial encounter
T76.31XD	Adult psychological abuse by nonspouse or nonpartner, Suspected, Subsequent encounter
T76.31XD	Spouse or partner abuse, Psychological, Suspected, Subsequent encounter
T76.32XA	Child psychological abuse, Suspected, Initial encounter
T76.32XD	Child psychological abuse, Suspected, Subsequent encounter
Z03.89	No diagnosis or condition
Z31.5	Genetic counseling
Z55.0	Illiteracy and low-level literacy
Z55.1	Schooling unavailable and unattainable
Z55.2	Failed school examinations
Z55.3	Underachievement in school
Z55.4	Educational maladjustment and discord with teachers and classmates
Z55.8	Problems related to inadequate teaching

Z55.9	Other problems related to education and literacy
Z56.0	Unemployment
Z56.1	Change of job
Z56.2	Threat of job loss
Z56.3	Stressful work schedule
Z56.4	Discord with boss and workmates
Z56.5	Uncongenial work environment
Z56.6	Other physical and mental strain related to work
Z56.81	Sexual harassment on the job
Z56.82	Problem related to current military deployment status
Z56.9	Other problem related to employment
Z58.6	Lack of safe drinking water
Z59.01	Sheltered homelessness
Z59.02	Unsheltered homelessness
Z59.1	Inadequate housing
Z59.2	Discord with neighbor, lodger, or landlord
Z59.3	Problem related to living in a residential institution

Z59.41	Food insecurity
Z59.5	Extreme poverty
Z59.6	Low income
Z59.7	Insufficient social or health insurance or welfare support
Z59.9	Other economic problem
Z59.9	Other housing problem
Z60.0	Phase of life problem
Z60.2	Problem related to living alone
Z60.3	Acculturation difficulty
Z60.4	Social exclusion or rejection
Z60.5	Target of (perceived) adverse discrimination or persecution
Z60.9	Other problem related to social environment
Z62.29	Upbringing away from parents
Z62.810	Personal history (past history) of physical abuse in childhood
Z62.810	Personal history (past history) of sexual abuse in childhood
Z62.811	Personal history (past history) of psychological abuse in childhood
Z62.812	Personal history (past history) of neglect in childhood
Z62.820	Parent-child relational problem, Parent–biological child
Z62.821	Parent-child relational problem, Parent–adopted child
Z62.822	Parent-child relational problem, Parent–foster child
Z62.891	Sibling relational problem
Z62.898	Child affected by parental relationship distress
Z62.898	Parent-child relational problem, Other caregiver–child
Z63.0	Relationship distress with spouse or intimate partner
Z63.4	Uncomplicated bereavement
Z63.5	Disruption of family by separation or divorce
Z63.8	High expressed emotion level within family

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Z64.0	Problems related to unwanted pregnancy
Z64.1	Problems related to multiparity
Z64.4	Discord with social service provider, including probation officer, case manager, or social services worker
Z65.0	Conviction in civil or criminal proceedings without imprisonment
Z65.1	Imprisonment or other incarceration
Z65.2	Problems related to release from prison

Z65.3	Problems related to other legal circumstances
Z65.4	Victim of crime
Z65.4	Victim of terrorism or torture
Z65.5	Exposure to disaster, war, or other hostilities
Z65.8	Religious or spiritual problem
Z69.010	Encounter for mental health services for victim of child neglect by parent
Z69.010	Encounter for mental health services for victim of child physical abuse by parent
Z69.010	Encounter for mental health services for victim of child psychological abuse by parent
Z69.010	Encounter for mental health services for victim of child sexual abuse by parent
Z69.011	Encounter for mental health services for perpetrator of parental child neglect
Z69.011	Encounter for mental health services for perpetrator of parental child physical abuse
Z69.011	Encounter for mental health services for perpetrator of parental child psychological abuse
Z69.011	Encounter for mental health services for perpetrator of parental child sexual abuse
Z69.020	Encounter for mental health services for victim of nonparental child neglect
Z69.020	Encounter for mental health services for victim of nonparental child physical abuse
Z69.020	Encounter for mental health services for victim of nonparental child psychological abuse
Z69.020	Encounter for mental health services for victim of nonparental child sexual abuse
Z69.021	Encounter for mental health services for perpetrator of nonparental child neglect
Z69.021	Encounter for mental health services for perpetrator of nonparental child physical abuse
Z69.021	Encounter for mental health services for perpetrator of nonparental child psychological abuse
Z69.021	Encounter for mental health services for perpetrator of nonparental child sexual abuse
Z69.11	Encounter for mental health services for victim of spouse or partner neglect
Z69.11	Encounter for mental health services for victim of spouse or partner psychological abuse
Z69.11	Encounter for mental health services for victim of spouse or partner violence, Physical

Z69.12	Encounter for mental health services for perpetrator of spouse or partner neglect

Z69.12	Encounter for mental health services for perpetrator of spouse or partner psychological abuse
Z69.12	Encounter for mental health services for perpetrator of spouse or partner violence, Physical
Z69.12	Encounter for mental health services for perpetrator of spouse or partner violence, Sexual
Z69.81	Encounter for mental health services for victim of nonspousal or nonpartner adult abuse
Z69.81	Encounter for mental health services for victim of spouse or partner violence, Sexual
Z69.82	Encounter for mental health services for perpetrator of nonspousal or nonpartner adult abuse
Z70.9	Sex counseling
Z71.3	Dietary counseling
Z71.9	Other counseling or consultation
Z72.0	Tobacco use disorder, mild
Z72.810	Child or adolescent antisocial behavior
Z72.811	Adult antisocial behavior
Z72.9	Problem related to lifestyle
Z75.3	Unavailability or inaccessibility of health care facilities
Z75.4	Unavailability or inaccessibility of other helping agencies
Z76.5	Malingering
Z91.19	Nonadherence to medical treatment
Z91.410	Personal history (past history) of spouse or partner violence, Physical
Z91.410	Personal history (past history) of spouse or partner violence, Sexual
Z91.411	Personal history (past history) of spouse or partner psychological abuse
Z91.412	Personal history (past history) of spouse or partner neglect
Z91.49	Personal history of psychological trauma
Z91.51	History of suicidal behavior
Z91.52	History of nonsuicidal self-injury
Z91.82	Personal history of military deployment
Z91.83	Wandering associated with a mental disorder

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DSM-5 Advisors and Other Contributors

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Kyla Pope, M.D.
Victor I. Reus, M.D.
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Joel Yager, M.D.

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Gregory A. Miller, M.D.
Roger Peele, M.D.
Charles S. Price, M.D.
Deepika Sastry, M.D.
John P.D. Shemo, M.D.
Eliot Sorel, M.D.

DSM-5 Summit Group

Dilip V. Jeste, M.D. (Chair)
R. Scott Benson, M.D.
Kenneth S. Kendler, M.D.
Helena C. Kraemer, Ph.D.

David J. Kupfer, M.D.
Jeffrey A. Lieberman, M.D.
Glenn A. Martin, M.D.
John S. McIntyre, M.D.
John M. Oldham, M.D.
Roger Peele, M.D.
Darrel A. Regier, M.D., M.P.H.
James H. Scully Jr., M.D.
Joel Yager, M.D.
Paul S. Appelbaum, M.D. (Consultant)
Michael B. First, M.D. (Consultant)

DSM-5 Field Trials Review

Robert D. Gibbons, Ph.D.
Craig Nelson, M.D.

DSM-5 Forensic Review

Paul S. Appelbaum, M.D.
Lama Bazzi, M.D.
Alec W. Buchanan, M.D., Ph.D.
Carissa Cabán Alemán, M.D.
Michael Champion, M.D.
Jeffrey C. Eisen, M.D.
Elizabeth Ford, M.D.
Daniel T. Hackman, M.D.
Mark Hauser, M.D.
Steven K. Hoge, M.D., M.B.A.
Debra A. Pinals, M.D.
Guillermo Portillo, M.D.
Patricia Recupero, M.D., J.D.
Robert Weinstock, M.D.
Cheryl Wills, M.D.

Howard V. Zonana, M.D.

Past DSM-5 APA Staff

Erin J. Dalder-Alpher
Kristin Edwards
Leah I. Engel
Lenna Jawdat
Elizabeth C. Martin
Rocio J. Salvador

Work Group Advisors

ADHD and Disruptive Behavior Disorders

Emil F. Coccaro, M.D.
Deborah Dabrick, Ph.D.
Prudence W. Fisher, Ph.D.
Benjamin B. Lahey, Ph.D.
Salvatore Mannuzza, Ph.D.
Mary Solanto, Ph.D.
J. Blake Turner, Ph.D.
Eric Youngstrom, Ph.D.

Anxiety, Obsessive-Compulsive Spectrum, Posttraumatic, and Dissociative Disorders

Lynn E. Alden, Ph.D.
David B. Arciniegas, M.D.
David H. Barlow, Ph.D.
Katja Beesdo-Baum, Ph.D.
Chris R. Brewin, Ph.D.
Richard J. Brown, Ph.D.
Timothy A. Brown, Ph.D.

Richard A. Bryant, Ph.D.
Joan M. Cook, Ph.D.
Joop de Jong, M.D., Ph.D.
Paul F. Dell, Ph.D.
Damiaan Denys, M.D.
Bruce P. Dohrenwend, Ph.D.
Brian A. Fallon, M.D., M.P.H.
Edna B. Foa, Ph.D.
Martin E. Franklin, Ph.D.
Wayne K. Goodman, M.D.
Jon E. Grant, J.D., M.D.
Bonnie L. Green, Ph.D.
Richard G. Heimberg, Ph.D.
Judith L. Herman, M.D.
Devon E. Hinton, M.D., Ph.D.
Stefan G. Hofmann, Ph.D.
Charles W. Hoge, M.D.
Terence M. Keane, Ph.D.
Nancy J. Keuthen, Ph.D.
Dean G. Kilpatrick, Ph.D.
Katharina Kircanski, Ph.D.
Laurence J. Kirmayer, M.D.
Donald F. Klein, M.D., D.Sc.
Amaro J. Laria, Ph.D.
Richard T. LeBeau, M.A.
Richard J. Loewenstein, M.D.
David Mataix-Cols, Ph.D.
Thomas W. McAllister, M.D.
Harrison G. Pope, M.D., M.P.H.
Ronald M. Rapee, Ph.D.
Steven A. Rasmussen, M.D.

Patricia A. Resick, Ph.D.
Vedat Şar, M.D.
Sanjaya Saxena, M.D.
Paula P. Schnurr, Ph.D.
M. Katherine Shear, M.D.
Daphne Simeon, M.D.
Harvey S. Singer, M.D.
Melinda A. Stanley, Ph.D.
James J. Strain, M.D.
Kate Wolitzky Taylor, Ph.D.
Onno van der Hart, Ph.D.
Eric Vermetten, M.D., Ph.D.
John T. Walkup, M.D.
Sabine Wilhelm, Ph.D.
Douglas W. Woods, Ph.D.
Richard E. Zinbarg, Ph.D.
Joseph Zohar, M.D.

Childhood and Adolescent Disorders

Adrian Angold, Ph.D.
Deborah Beidel, Ph.D.
David Brent, M.D.
John Campo, M.D.
Gabrielle Carlson, M.D.
Prudence W. Fisher, Ph.D.
David Klonsky, Ph.D.
Matthew Nock, Ph.D.
J. Blake Turner, Ph.D.

Eating Disorders

Michael J. Devlin, M.D.
Denise E. Wilfley, Ph.D.

Susan Z. Yanovski, M.D.

Mood Disorders

Boris Birmaher, M.D.
Yeates Conwell, M.D.
Ellen B. Dennehy, Ph.D.
S. Ann Hartlage, Ph.D.
Jack M. Hettema, M.D., Ph.D.
Michael C. Neale, Ph.D.
Gordon B. Parker, M.D., Ph.D., D.Sc.
Roy H. Perlis, M.D., M.Sc.
Holly G. Prigerson, Ph.D.
Norman E. Rosenthal, M.D.
Peter J. Schmidt, M.D.

Mort M. Silverman, M.D.
Meir Steiner, M.D., Ph.D.
Mauricio Tohen, M.D., Dr.P.H., M.B.A.
Sidney Zisook, M.D.

Neurocognitive Disorders

Jiska Cohen-Mansfield, Ph.D.
Vladimir Hachinski, M.D., C.M., D.Sc.
Sharon Inouye, M.D., M.P.H.
Grant Iverson, Ph.D.
Laura Marsh, M.D.
Bruce Miller, M.D.
Jacobo Mintzer, M.D., M.B.A.
Bruce G. Pollock, M.D., Ph.D.
George Prigatano, Ph.D.
Ron Ruff, Ph.D.

Ingmar Skoog, M.D., Ph.D.
Robert Sweet, M.D.
Paula Trzepacz, M.D.

Neurodevelopmental Disorders

Ari Ne'eman
Nickola Nelson, Ph.D.
Diane Paul, Ph.D.
Eva Petrova, Ph.D.
Andrew Pickles, Ph.D.
Jan Piek, Ph.D.
Helene Polatajko, Ph.D.
Alya Reeve, M.D.
Mabel Rice, Ph.D.
Joseph Sergeant, Ph.D.
Bennett Shaywitz, M.D.
Sally Shaywitz, M.D.
Audrey Thurm, Ph.D.
Keith Widaman, Ph.D.
Warren Zigman, Ph.D.

Personality and Personality Disorders

Eran Chemerinski, M.D.
Thomas N. Crawford, Ph.D.
Harold W. Koenigsberg, M.D.
Kristian E. Markon, Ph.D.
Rebecca L. Shiner, Ph.D.
Kenneth R. Silk, M.D.
Jennifer L. Tackett, Ph.D.
David Watson, Ph.D.

Psychotic Disorders

Kamaldeep Bhui, M.D.

Manuel J. Cuesta, M.D., Ph.D.
Richard Douyon, M.D.
Paolo Fusar-Poli, Ph.D.
John H. Krystal, M.D.
Thomas H. McGlashan, M.D.
Victor Peralta, M.D., Ph.D.
Anita Riecher-Rössler, M.D.
Mary V. Seeman, M.D.

Sexual and Gender Identity Disorders

Stan E. Althof, Ph.D.
Richard Balon, M.D.
John H.J. Bancroft, M.D., M.A., D.P.M.
Howard E. Barbaree, Ph.D., M.A.
Rosemary J. Basson, M.D.
Sophie Bergeron, Ph.D.
Anita H. Clayton, M.D.
David L. Delmonico, Ph.D.
Domenico Di Ceglie, M.D.
Esther Gomez-Gil, M.D.
Jamison Green, Ph.D.
Richard Green, M.D., J.D.
R. Karl Hanson, Ph.D.
Lawrence Hartmann, M.D.
Stephen J. Hucker, M.B.
Eric S. Janus, J.D.
Patrick M. Jern, Ph.D.
Megan S. Kaplan, Ph.D.
Raymond A. Knight, Ph.D.
Ellen T.M. Laan, Ph.D.
Stephen B. Levine, M.D.

Christopher G. McMahon, M.B.
Marta Meana, Ph.D.
Michael H. Miner, Ph.D., M.A.
William T. O'Donohue, Ph.D.
Michael A. Perelman, Ph.D.
Caroline F. Pukall, Ph.D.
Robert E. Pyke, M.D., Ph.D.
Vernon L. Quinsey, Ph.D., M.Sc.
David L. Rowland, Ph.D., M.A.
Michael Sand, Ph.D., M.P.H.
Leslie R. Schover, Ph.D., M.A.
Paul Stern, B.S., J.D.
David Thornton, Ph.D.
Leonore Tiefer, Ph.D.
Douglas E. Tucker, M.D.
Jacques van Lankveld, Ph.D.
Marcel D. Waldinger, M.D., Ph.D.

Sleep-Wake Disorders

Donald L. Bliwise, Ph.D.
Daniel J. Buysse, M.D.
Vishesh K. Kapur, M.D., M.P.H.
Sanjeeve V. Kothare, M.D.
Kenneth L. Lichstein, Ph.D.
Mark W. Mahowald, M.D.
Rachel Manber, Ph.D.
Emmanuel Mignot, M.D., Ph.D.
Timothy H. Monk, Ph.D., D.Sc.
Thomas C. Neylan, M.D.
Maurice M. Ohayon, M.D., D.Sc., Ph.D.
Judith Owens, M.D., M.P.H.

Daniel L. Picchietti, M.D.
Stuart F. Quan, M.D.
Thomas Roth, Ph.D.
Daniel Weintraub, M.D.

Theresa B. Young, Ph.D.
Phyllis C. Zee, M.D., Ph.D.

Somatic Symptom Disorders

Brenda Bursch, Ph.D.
Kurt Kroenke, M.D.
W. Curt LaFrance Jr., M.D., M.P.H.
Jon Stone, M.B., Ch.B., Ph.D.
Lynn M. Wegner, M.D.

Substance-Related Disorders

Raymond F. Anton Jr., M.D.
Deborah A. Dawson, Ph.D.
Roland R. Griffiths, Ph.D.
Dorothy K. Hatsukami, Ph.D.
John E. Helzer, M.D.
Marilyn A. Huestis, Ph.D.
John R. Hughes, M.D.
Laura M. Juliano, Ph.D.
Thomas R. Kosten, M.D.
Nora D. Volkow, M.D.

DSM-5 Study Group and Other DSM-5 Group Advisors

Lifespan Developmental Approaches

Christina Bryant, Ph.D.
Amber Gum, Ph.D.

Thomas Meeks, M.D.
Jan Mohlman, Ph.D.
Steven Thorp, Ph.D.
Julie Wetherell, Ph.D.

Gender and Cross-Cultural Issues

Neil K. Aggarwal, M.D., M.B.A., M.A.
Sofie Bäärnhielm, M.D., Ph.D.
José J. Bauermeister, Ph.D.
James Boehnlein, M.D., M.Sc.
Jaswant Guzder, M.D.
Alejandro Interian, Ph.D.
Sushrut S. Jadhav, M.B.B.S., M.D., Ph.D.
Laurence J. Kirmayer, M.D.
Alex J. Kopelowicz, M.D.
Amaro J. Laria, Ph.D.
Steven R. Lopez, Ph.D.
Kwame J. McKenzie, M.D.
John R. Peteet, M.D.
Hans (J.G.B.M.) Rohlof, M.D.
Cecile Rousseau, M.D.
Mitchell G. Weiss, M.D., Ph.D.

Psychiatric/General Medical Interface

Daniel L. Coury, M.D.
Bernard P. Dreyer, M.D.
Danielle Laraque, M.D.
Lynn M. Wegner, M.D.

Impairment and Disability

Prudence W. Fisher, Ph.D.
Martin Prince, M.D., M.Sc.
Michael R. Von Korff, Sc.D.

Diagnostic Assessment Instruments

Prudence W. Fisher, Ph.D.
Robert D. Gibbons, Ph.D.
Ruben Gur, Ph.D.
John E. Helzer, M.D.
John Houston, M.D., Ph.D.
Kurt Kroenke, M.D.

Other Contributors/Consultants

ADHD and Disruptive Behavior Disorders

Patrick E. Shrout, Ph.D.
Erik Willcutt, Ph.D.

Anxiety, Obsessive-Compulsive Spectrum, Posttraumatic, and Dissociative Disorders

Etzel Cardeña, Ph.D.
Richard J. Castillo, Ph.D.
Eric Hollander, M.D.
Charlie Marmar, M.D.
Alfonso Martínez-Taboas, Ph.D.
Mark W. Miller, Ph.D.
Mark H. Pollack, M.D.
Heidi S. Resnick, Ph.D.

Childhood and Adolescent Disorders

Grace T. Baranek, Ph.D.
Colleen Jacobson, Ph.D.
Maria Oquendo, M.D.
Sir Michael Rutter, M.D.

Eating Disorders

Nancy L. Zucker, Ph.D.

Mood Disorders

Keith Hawton, M.D., Ph.D.
David A. Jobes, Ph.D.
Maria A. Oquendo, M.D.
Alan C. Swann, M.D.

987

Neurocognitive Disorders

J. Eric Ahlskog, M.D., Ph.D.
Allen J. Aksamit, M.D.
Marilyn Albert, Ph.D.
Guy Mckhann, M.D.
Bradley Boeve, M.D.
Helena Chui, M.D.
Sureyya Dikmen, Ph.D.
Douglas Galasko, M.D.
Harvey Levin, Ph.D.
Mark Lovell, Ph.D.
Jeffery Max, M.B.B.Ch.
Ian McKeith, M.D.
Cynthia Munro, Ph.D.
Marlene Oscar-Berman, Ph.D.
Alexander Troster, Ph.D.

Neurodevelopmental Disorders

Anna Barnett, Ph.D.
Martha Denckla, M.D.
Jack M. Fletcher, Ph.D.
Dido Green, Ph.D.
Stephen Greenspan, Ph.D.
Bruce Pennington, Ph.D.
Ruth Shalev, M.D.

Larry B. Silver, M.D.
Lauren Swineford, Ph.D.
Michael Von Aster, M.D.

Personality and Personality Disorders

Patricia R. Cohen, Ph.D.
Jaime L. Derringer, Ph.D.
Lauren Helm, M.D.
Christopher J. Patrick, Ph.D.
Anthony Pinto, Ph.D.

Psychotic Disorders

Scott W. Woods, M.D.

Sexual and Gender Identity Disorders

Alan J. Riley, M.Sc.
Ray C. Rosen, Ph.D.

Sleep-Wake Disorders

Jack D. Edinger, Ph.D.
David Gozal, M.D.
Hochang B. Lee, M.D.
Tore A. Nielsen, Ph.D.
Michael J. Sateia, M.D.
Jamie M. Zeitzer, Ph.D.

Somatic Symptom Disorders

Chuck V. Ford, M.D.
Patricia I. Rosebush, M.Sc.N., M.D.

Substance-Related Disorders

Sally M. Anderson, Ph.D.
Julie A. Kable, Ph.D.
Christopher Martin, Ph.D.
Sarah N. Mattson, Ph.D.

Edward V. Nunes Jr., M.D.
Mary J. O’Connor, Ph.D.
Heather Carmichael Olson, Ph.D.
Blair Paley, Ph.D.
Edward P. Riley, Ph.D.
Tulshi D. Saha, Ph.D.
Wim van den Brink, M.D., Ph.D.
George E. Woody, M.D.

Diagnostic Spectra and DSM/ICD Harmonization

Bruce Cuthbert, Ph.D.

Lifespan Developmental Approaches

Aartjan Beekman, Ph.D.
Alistair Flint, M.B.
David Sultzer, M.D.
Ellen Whyte, M.D.

Gender and Cross-Cultural Issues

Sergio Aguilar-Gaxiola, M.D., Ph.D.
Kavoos G. Bassiri, M.S.
Venkataramana Bhat, M.D.
Marit Boiler, M.P.H.
Paul Brodwin, Ph.D.
Denise Canso, M.Sc.
Richard J. Castillo, Ph.D.
Smita N. Deshpande, M.D., D.P.M.
Ravi DeSilva, M.D.
Esperanza Diaz, M.D.
Byron J. Good, Ph.D.
Simon Groen, M.A.
Peter J. Guarnaccia, Ph.D.
Devon E. Hinton, M.D., Ph.D.

Ladson Hinton, M.D.
Lincoln I. Khasakhala, Ph.D.
Francis G. Lu, M.D.
Athena Madan, M.A.
Anne W. Mbwayo, Ph.D.
Oanh Meyer, Ph.D.
Victoria N. Mutiso, Ph.D., D.Sc.
David M. Ndetei, M.D.
Andel V. Nicasio, M.S.Ed.
Vasudeo Paralikar, M.D., Ph.D.
Kanak Patil, M.A.
Filipa I. Santos, H.B.Sc.
Sanjeev B. Sarmukaddam, Ph.D., M.Sc.
Monica Z. Scalco, M.D., Ph.D.
Katie Thompson, M.A.
Hendry Ton, M.D., M.Sc.
Rob C.J. van Dijk, M.Sc.

William A. Vega, Ph.D.
Johann M. Vega-Dienstmaier, M.D.
Sergio J. Villaseñor-Bayardo, M.D., Ph.D.
Joseph Westermeyer, M.D., Ph.D.

Psychiatric/General Medical Interface

Daniel J. Balog, M.D.
Charles C. Engel, M.D., M.P.H.
Charles D. Motsinger, M.D.

Impairment and Disability

Cille Kennedy, Ph.D.

Diagnostic Assessment Instruments

Paul J. Pikonis, Ph.D.

Other Conditions That May Be a Focus of Clinical Attention

William E. Narrow, M.D., M.P.H., *Chair*
Roger Peele, M.D.
Lawson R. Wulsin, M.D.
Charles H. Zeanah, M.D.
Prudence W. Fisher, Ph.D., *Advisor*
Stanley N. Caroff, M.D., *Contributor/Consultant*
James B. Lohr, M.D., *Contributor/Consultant*
Marianne Wambolt, Ph.D., *Contributor/Consultant*

DSM-5 Research Group

Allan Donner, Ph.D.

CPHC Peer Reviewers

Kenneth Altshuler, M.D.
Pedro G. Alvarenga, M.D.
Diana J. Antonacci, M.D.
Richard Balon, M.D.
David H. Barlow, Ph.D.
L. Jarrett Barnhill, M.D.
Katja Beesdo-Baum, Ph.D.
Marty Boman, Ed.D.
James Bourgeois, M.D.
David Braff, M.D.
Harry Brandt, M.D.
Kirk Brower, M.D.
Rachel Bryant-Waugh, Ph.D.
Jack D. Burke Jr., M.D., M.P.H.
Brenda Bursch, Ph.D.
Joseph Camilleri, M.D.

Patricia Casey, M.D.
F. Xavier Castellanos, M.D.
Eran Chemerinski, M.D.
Wai Chen, M.D.
Elie Cheniaux, M.D., D.Sc.
Cheryl Chessick, M.D.
J. Richard Ciccone, M.D.
Anita H. Clayton, M.D.
Tihalia J. Coleman, Ph.D.
John Csernansky, M.D.
Manuel J. Cuesta, M.D., Ph.D.
Joanne L. Davis, M.D.
David L. Delmonico, Ph.D.
Ray J. DePaulo, M.D.
Dimitris Dikeos, M.D.
Ina E. Djonlagic, M.D.
C. Neill Epperson, M.D.
Javier I. Escobar, M.D., M.Sc.
Spencer Eth, M.D.
David Fassler, M.D.
Giovanni A. Fava, M.D.
Robert Feinstein, M.D.
Molly Finnerty, M.D.
Mark H. Fleisher, M.D.
Alessio Florentini, M.D.
Laura Fochtmann, M.D.
Marshal Forstein, M.D.
William French, M.D.
Maximillian Gahr, M.D.
Cynthia Geppert, M.D.
Ann Germaine, Ph.D.

Marcia Goin, M.D.
David A. Gorelick, M.D., Ph.D.
David Graeber, M.D.
Cynthia A. Graham, Ph.D.
Andreas Hartmann, M.D.
Victoria Hendrick, M.D.
Merrill Herman, M.D.
David Herzog, M.D.
Mardi Horowitz, M.D.
Ya-fen Huang, M.D.
Anthony Kales, M.D
Niranjan S. Karnik, M.D., Ph.D.
Jeffrey Katzman, M.D.
Bryan King, M.D.
Cecilia Kjellgren, M.D.
Harold W. Koenigsberg, M.D.
Richard B. Krueger, M.D.
Steven Lamberti, M.D.
Ruth A. Lanius, M.D.
John Lauriello, M.D.
Anthony Lehman, M.D.
Michael Linden, M.D.
Mark W. Mahowald, M.D.
Marsha D. Marcus, Ph.D.
Stephen Marder, M.D.
Wendy Marsh, M.D.
Michael S. McCloskey, Ph.D.

Jeffrey Metzner, M.D.

Robert Michels, M.D.
Laura Miller, M.D.
Michael C. Miller, M.D.
Frederick Moeller, M.D.
Peter T. Morgan, M.D., Ph.D.
Madhav Muppa, M.D.
Philip Muskin, M.D.
Joachim Nitschke, M.D.
Abraham Nussbaum, M.D.
Ann Olincy, M.D.
Mark Onslow, Ph.D.
Sally Ozonoff, Ph.D.
John R. Peteet, M.D.
Ismene L. Petrakis, M.D.
Christophe M. Pfeiffer, M.D.
Karen Pierce, M.D.
Belinda Plattner, M.D.
Franklin Putnam, M.D.
Stuart F. Quan, M.D.
John Racy, M.D.
Phillip Resnick, M.D.
Michele Riba, M.D.
Jerold Rosenbaum, M.D.
Stephen Ross, M.D.
Lawrence Scahill, M.S.N., Ph.D.
Daniel Schechter, M.D.
Mary V. Seeman, M.D.
Alessandro Serretti, M.D.
Jianhua Shen, M.D.
Ravi Kumar R. Singareddy, M.D.
Ingmar Skoog, M.D., Ph.D.

Gary Small, M.D.
Paul Soloff, M.D.
Christina Stadler, M.D., Ph.D.
Nada Stotland, M.D.
Neil Swerdlow, M.D.
Kim Tillery, Ph.D.
David Tolin, Ph.D.
Jayne Trachman, M.D.
Luke Tsai, M.D.
Ming T. Tsuang, M.D., Ph.D.
Richard Tuch, M.D.
Johan Verhulst, M.D.
B. Timothy Walsh, M.D.
Michael Weissberg, M.D.
Godehard Weniger, M.D.
Keith Widaman, Ph.D.
Thomas Wise, M.D.
George E. Woods, M.D.
Kimberly A. Yonkers, M.D.
Alexander Young, M.D.

DSM-5 Field Trials in Academic Clinical Centers—Adult Samples

David Geffen School of Medicine, University of California, Los Angeles

Investigator

Helen Lavretsky, M.D., Principal Investigator

Referring and Interviewing Clinicians

Jessica Brommelhoff, Ph.D.
Xavier Cagigas, Ph.D.

Paul Cernin, Ph.D.
Linda Ercoli, Ph.D.
Randall Espinoza, M.D.
Helen Lavretsky, M.D.
Jeanne Kim, Ph.D.
David Merrill, M.D.
Karen Miller, Ph.D.
Christopher Nunez, Ph.D.

Research Coordinators

Natalie St. Cyr, M.A., Lead Research Coordinator
Nora Nazarian, B.A.
Colin Shinn, M.A.

Centre for Addiction and Mental Health, Toronto, Ontario, Canada

Investigators

Bruce G. Pollock, M.D., Ph.D., Lead Principal Investigator
R. Michael Bagby, Ph.D., Principal Investigator
Kwame J. McKenzie, M.D., Principal Investigator
Tony P. George, M.D., Co-investigator
Lena C. Quilty, Ph.D., Co-investigator
Peter Voore, M.D., Co-investigator

Referring and Interviewing Clinicians

Donna E. Akman, Ph.D.
R. Michael Bagby, Ph.D.
Wayne C. V. Baici, M.D.
Crystal Baluyut, M.D.
Eva W. C. Chow, M.D., J.D., M.P.H.
Z. J. Daskalakis, M.D., Ph.D.
Pablo Diaz-Hermosillo, M.D.

Paul A. Frewen, Ph.D.
Ariel Graff-Guerrero, M.D., M.Sc., Ph.D.
Margaret K. Hahn, M.D.
Lorena Hsu, Ph.D.
Justine Joseph, Ph.D.
Sean Kidd, Ph.D.
Kwame J. McKenzie, M.D.
Mahesh Menon, Ph.D.
Romina Mizrahi, M.D., Ph.D.
Daniel J. Mueller, M.D., Ph.D.
Lena C. Quilty, Ph.D.
Anthony C. Ruocco, Ph.D.
Jorge Soni, M.D.
Aristotle N. Voineskos, M.D., Ph.D.
George Voineskos, M.D.
Peter Voore, Ph.D.
Chris Watson, Ph.D.

Referring Clinicians

Ofer Agid, M.D.
Ash Bender, M.D.
Patricia Cavanagh, M.D.
Sarah Colman, M.D.
Vincenzo Deluca, M.D.
Justin Geagea, M.D.
David S. Goldbloom, M.D.
Daniel Greben, M.D.
Malati Gupta, M.D.

Ken Harrison, M.D.
Imraan Jeeva, M.D.
Joel Jeffries, M.B.
Judith Laposa, Ph.D.
Jan Malat, M.D.
Shelley McMain, Ph.D.
Bruce G. Pollock, M.D., Ph.D.
Andriy V. Samokhvalov, M.D., Ph.D.
Martin Strassnig, M.D.
Albert H. C. Wong, M.D., Ph.D.

Research Coordinators

Gloria I. Leo, M.A., Lead Research Coordinator
Anissa D. Bachan, B.A.
Bahar Haji-Khamneh, M.A.
Olga Likhodi, M.Sc.
Eleanor J. Liu, Ph.D.
Sarah A. McGee Ng, B.B.A.

Other Research Staff

Susan E. Dickens, M.A., Clinical Research Manager
Sandy Richards, B.Sc.N., Schizophrenia Research Manager

Dallas VA Medical Center, Dallas, Texas

Investigators

Carol S. North, M.D., M.P.E., Principal Investigator
Alina Suris, Ph.D., A.B.P.P., Principal Investigator

Referring and Interviewing Clinicians

Barry Ardolf, Psy.D.
Abila Awan, M.D.
Joel Baskin, M.D.
John Black, Ph.D.

Jeffrey Dodds, Ph.D.
Gloria Emmett, Ph.D.
Karma Hudson, M.D.
Jamylah Jackson, Ph.D., A.B.P.P.
Lynda Kirkland-Culp, Ph.D., A.B.P.P.
Heidi Koehler, Ph.D., A.B.P.P.
Elizabeth Lewis, Psy.D.
Aashish Parikh, M.D.
Reed Robinson, Ph.D.
Jheel Shah, M.D.
Geetha Shivakumar, M.D.
Sarah Spain, Ph.D., A.B.P.P.
Lisa Thoman, Ph.D.
Lia Thomas, M.D.
Jamie Zabukovec, Psy.D.
Mustafa Zaidi, M.D.
Andrea Zartman, Ph.D.

General Referral Sources

Robert Blake, L.M.S.W.
Evelyn Gibbs, L.M.S.W.
Michelle King-Thompson, L.M.S.W.

Research Coordinators

Jeannie B. Whitman, Ph.D., Lead Research Coordinator
Sunday Adewuyi, M.D.
Elizabeth Anderson, B.A.
Solaleh Azimipour, B.S.
Carissa Barney, B.S.
Kristie Cavazos, B.A.
Robert Devereaux, B.S.
Dana Downs, M.S., M.S.W.

Sharjeel Farooqui, M.D.
Julia Smith, Psy.D.
Kun-Ying H. Sung, B.S.

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School of Medicine, The University of Texas San Antonio, San Antonio, Texas

Investigator

Mauricio Tohen, M.D., Dr.P.H., M.B.A., Principal Investigator

Referring and Interviewing Clinicians

Suman Baddam, Psy.D.
Charles L. Bowden, M.D.
Nancy Diazgranados, M.D., M.S.
Craig A. Dike, Psy.D.
Dianne E. Dunn, Psy.D., M.P.H.
Elena Gherman, M.D.
Jodi M. Gonzalez, Ph.D.
Pablo Gonzalez, M.D.
Phillip Lai, Psy.D.
Natalie Maples-Aguilar, M.A., L.P.A.
Marlon P. Quinones, M.D.
Jeslina J. Raj, Psy.D.
David L. Roberts, Ph.D.
Nancy Sandusky, R.N., F.P.M.H.N.P.-B.C., D.N.P.-C.
Donna S. Stutes, M.S., L.P.C.
Mauricio Tohen, M.D., Dr.P.H., M.B.A.
Dawn I. Velligan, Ph.D.
Weiran Wu, M.D., Ph.D.

Referring Clinicians

Albana Dassori, M.D.

Megan Frederick, M.A.
Robert Gonzalez, M.D.
Uma Kasinath, M.D.
Camis Milam, M.D.
Vivek Singh, M.D.
Peter Thompson, M.D.

Research Coordinators

Melissa Hernandez, B.A., Lead Research Coordinator
Fermin Alejandro Carrizales, B.A.
Martha Dahl, R.N., B.S.N.
Patrick M. Smith, B.A.
Nicole B. Watson, M.A.

Michael E. DeBakey VA Medical Center and the Menninger Clinic, Houston, Texas (Joint Study Site)

Michael E. DeBakey VA Medical Center

Investigator

Laura Marsh, M.D., Principal Investigator

Referring and Interviewing Clinicians

Shalini Aggarwal, M.D.
Su Bailey, Ph.D.
Minnete (Helen) Beckner, Ph.D.
Crystal Clark, M.D.
Charles DeJohn, M.D.
Robert Garza, M.D.
Aruna Gottumakkla, M.D.
Janet Hickey, M.D.
James Ireland, M.D.
Mary Lois Lacey, A.P.R.N.
Wendy Leopoulos, M.D.

Laura Marsh, M.D.
Deleene Menefee, Ph.D.
Brian I. Miller, Ph.D.
Candy Smith, Ph.D.
Avila Steele, Ph.D.
Jill Wanner, Ph.D.
Rachel Wells, Ph.D.
Kaki York-Ward, Ph.D.

Referring Clinicians

Sara Allison, M.D.
Leonard Denney, L.C.S.W.
Catherine Flores, L.C.S.W.
Nathalie Marie, M.D.
Christopher Martin, M.D.
Sanjay Mathew, M.D.
Erica Montgomery, M.D.
Gregory Scholl, P.A.
Jocelyn Ulanday, M.D., M.P.H.

Research Coordinators

Sarah Neely Torres, B.S., Lead Research Coordinator
Kathleen Grout, M.A.
Lea Kiefer, M.P.H.
Jana Tran, M.A.

Volunteer Research Assistants

Catherine Clark
Linh Hoang

Menninger Clinic

Investigator

Efrain Bleiberg, M.D., Principal Investigator

Referring and Interviewing Clinicians

Jennifer Baumgardner, Ph.D.
Elizabeth Dodd Conaway, L.C.S.W., B.C.D.
Warren Christianson, D.O.
Wesley Clayton, L.M.S.W.
J. Christopher Fowler, Ph.D.
Michael Groat, Ph.D.
Edythe Harvey, M.D.
Denise Kagan, Ph.D.
Hans Meyer, L.C.S.W.
Segundo Robert-Ibarra, M.D.
Sandhya Trivedi, M.D.
Rebecca Wagner, Ph.D.
Harrell Woodson, Ph.D.
Amanda Yoder, L.C.S.W.

Referring Clinicians

James Flack, M.D.
David Ness, M.D.

Research Coordinators

Steve Herrera, B.S., M.T., Lead Research Coordinator
Allison Kalpakci, B.A.

Mayo Clinic, Rochester, Minnesota

Investigators

Mark A. Frye, M.D., Principal Investigator
Glenn E. Smith, Ph.D., Principal Investigator
Jeffrey P. Staab, M.D., M.S., Principal Investigator

Referring and Interviewing Clinicians

Osama Abulseoud, M.D.

Jane Cerhan, Ph.D.
Julie Fields, Ph.D.
Mark A. Frye, M.D.
Manuel Fuentes, M.D.
Yonas Geda, M.D.
Maria Harmandayan, M.D.
Reba King, M.D.
Simon Kung, M.D.
Mary Machuda, Ph.D.
Donald McAlpine, M.D.
Alastair McKean, M.D.
Juliana Moraes, M.D.
Teresa Rummans, M.D.
James R. Rundell, M.D.
Richard Seime, Ph.D.
Glenn E. Smith, Ph.D.
Christopher Sola, D.O.
Jeffrey P. Staab, M.D., M.S.
Marin Veldic, M.D.
Mark D. Williams, M.D.
Maya Yustis, Ph.D.

Research Coordinators

Lisa Seymour, B.S., Lead Research Coordinator
Scott Feeder, M.S.
Lee Gunderson, B.S.
Sherrie Hanna, M.A., L.P.
Kelly Harper, B.A.
Katie Mingo, B.A.
Cynthia Stoppel, A.S.

Other Study Staff

Anna Frye
Andrea Hogan

Perelman School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania

Investigators

Mahendra T. Bhati, M.D., Principal Investigator
Marna S. Barrett, Ph.D., Co-investigator
Michael E. Thase, M.D., Co-investigator

Referring and Interviewing Clinicians

Peter B. Bloom, M.D.
Nicole K. Chalmers, L.C.S.W.
Torrey A. Creed, Ph.D.
Mario Cristancho, M.D.
Amy Cunningham, Psy.D.
John P. Dennis, Ph.D.
Josephine Elia, M.D.
Peter Gariti, Ph.D., L.C.S.W.
Philip Gehrman, Ph.D.
Laurie Gray, M.D.
Emily A.P. Haigh, Ph.D.
Nora J. Johnson, M.B.A., M.S., Psy.D.
Paulo Knapp, M.D.
Yong-Tong Li, M.D.
Bill Mace, Ph.D.
Kevin S. McCarthy, Ph.D.
Dimitri Perivoliotis, Ph.D.
Luke Schultz, Ph.D.

Tracy Steen, Ph.D.
Chris Tjoa, M.D.
Nancy A. Wintering, L.C.S.W.

Referring Clinicians

Eleanor Ainslie, M.D.
Kelly C. Allison, Ph.D.
Rebecca Aspden, M.D.
Claudia F. Baldassano, M.D.
Vijayta Bansal, M.D.
Rachel A. Bennett, M.D.
Richard Bollinger, Ph.D.
Andrea Bowen, M.D.
Karla Campanella, M.D.
Anthony Carlino, M.D.
Noah Carroll, M.S.S.
Alysia Cirona, M.D.
Samuel Collier, M.D.
Andreea Crauciuc, L.C.S.W.
Pilar Cristancho, M.D.
Traci D'Almeida, M.D.
Kathleen Diller, M.D.
Benoit Dubé, M.D.
Jon Dukes, M.S.W.
Lauren Elliott, M.D.
Mira Elwell, B.A.
Mia Everett, M.D.
Lucy F. Faulconbridge, Ph.D.
Patricia Furlan, Ph.D.
Joanna Goldstein, L.C.S.W.
Paul Grant, Ph.D.

Jillian Graves, L.C.S.W.
Tamar Gur, M.D., Ph.D.
Alisa Gutman, M.D., Ph.D.
Nora Hymowitz, M.D.
Sofia Jensen, M.D.
Tiffany King, M.S.W.
Katherine Levine, M.D.
Alice Li, M.D.
Janet Light, L.C.S.W.
John Listerud, M.D., Ph.D.
Emily Malcoun, Ph.D.
Donovan Maust, M.D.
Adam Meadows, M.D.
Michelle Moyer, M.D.
Rebecca Naugle, L.C.S.W.
Cory Newman, Ph.D.
John Northrop, M.D., Ph.D.
Elizabeth A. Ellis Ohr, Psy.D.
John O'Reardon, M.D.
Abraham Pachikara, M.D.
Andrea Perelman, M.S.W.
Diana Perez, M.S.W.
Bianca Previdi, M.D.
J. Russell Ramsay, Ph.D.
Jorge Rivera-Colon, M.D.
Jan Smedley, L.C.S.W.
Katie Struble, M.S.W.
Aita Susi, M.D.
Yekaterina Tatarchuk, M.D.
Ellen Tarves, M.A.
Allison Tweedie, M.D.

Holly Valerio, M.D.
Thomas A. Wadden, Ph.D.
Joseph Wright, Ph.D.
Yan Xuan, M.D.
David Yusko, Psy.D.

Research Coordinators

Jordan A. Coello, B.A., Lead Research Coordinator
Eric Wang, B.S.E.

Volunteer Research Assistants/Interns

Jeannine Barker, M.A., A.T.R.
Jacqueline Baron
Kelsey Bogue
Alexandra Ciomek
Martekuor Dodoo, B.A.
Julian Domanico
Laura Heller, B.A.
Leah Hull-Rawson, B.A.
Jacquelyn Klehm, B.A.
Christina Lam
Dante Proetto, B.S.
Molly Roy
Casey Shannon

Stanford University School of Medicine, Stanford, California

Investigators

Carl Feinstein, M.D., Principal Investigator
Debra Safer, M.D., Principal Investigator

Referring and Interviewing Clinicians

Kari Berquist, Ph.D.
Eric Clausell, Ph.D.

Danielle Colborn, Ph.D.
Whitney Daniels, M.D.
Alison Darcy, Ph.D.
Krista Fielding, M.D.
Mina Fisher, M.D.
Kara Fitzpatrick, Ph.D.
Wendy Froehlich, M.D.
Grace Gengoux, Ph.D.
Anna Cassandra Golding, Ph.D.
Lisa Groesz, Ph.D.
Kyle Hinman, M.D.
Rob Holaway, Ph.D.
Matthew Holve, M.D.
Rex Huang, M.D.
Nina Kirz, M.D.

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Megan Klabunde, Ph.D.
John Leckie, Ph.D.
Naomi Leslie, M.D.
Adrianne Lona, M.D.
Ranvinder Rai, M.D.
Rebecca Rialon, Ph.D.
Beverly Rodriguez, M.D., Ph.D.
Debra Safer, M.D.
Mary Sanders, Ph.D.
Jamie Scaletta, Ph.D.
Norah Simpson, Ph.D.
Manpreet Singh, M.D.
Maria-Christina Stewart, Ph.D.

Melissa Vallas, M.D.
Patrick Whalen, Ph.D.
Sanno Zack, Ph.D.

Referring Clinicians

Robin Apple, Ph.D.
Victor Carrion, M.D.
Carl Feinstein, M.D.
Christine Gray, Ph.D.
Antonio Hardan, M.D.
Megan Jones, Psy.D.
Linda Lotspeich, M.D.
Lauren Mikula, Psy.D.
Brandyn Street, Ph.D.
Violeta Tan, M.D.
Heather Taylor, Ph.D.
Jacob Towery, M.D.
Sharon Williams, Ph.D.

Research Coordinators

Kate Arnow, B.A., Lead Research Coordinator
Nandini Datta, B.S.
Stephanie Manasse, B.A.

Volunteer Research Assistants/Interns

Arianna Martin, M.S.
Adriana Nevado, B.A.

Children's Hospital Colorado, Aurora, Colorado

Investigator

Marianne Wamboldt, M.D., Principal Investigator

Referring and Interviewing Clinicians

Galia Abadi, M.D.

Steven Behling, Ph.D.
Jamie Blume, Ph.D.
Adam Burstein, M.D.
Debbie Carter, M.D.
Kelly Caywood, Ph.D.
Meredith Chapman, M.D.
Paulette Christian, A.P.P.M.H.N.
Mary Cook, M.D.
Anthony Cordaro, M.D.
Audrey Dumas, M.D.
Guido Frank, M.D.
Karen Frankel, Ph.D.
Darryl Graham, Ph.D.
Yael Granader, Ph.D.
Isabelle Guillemet, M.D.
Patrece Hairston, Ph.D.
Charles Harrison, Ph.D.
Tammy Herckner, L.C.S.W.
Cassie Karlsson, M.D.
Kimberly Kelsay, M.D.
David Kieval, Ph.D.
Megan Klabunde, Ph.D.
Jaimelyn Kost, L.C.S.W.
Harrison Levine, M.D.
Raven Lipmanson, M.D.
Susan Lurie, M.D.
Asa Marokus, M.D.
Idalia Massa, Ph.D.
Christine McDunn, Ph.D.
Scot McKay, M.D.
Marissa Murgolo, L.C.S.W.

Alyssa Oland, Ph.D.
Lina Patel, Ph.D.
Rheena Pineda, Ph.D.
Gautam Rajendran, M.D.
Diane Reichmuth, Ph.D.
Michael Rollin, M.D.
Marlena Romero, L.C.S.W.
Michelle Roy, Ph.D.
Celeste St. John-Larkin, M.D.
Elise Sannar, Ph.D.
Daniel Savin, M.D.
Claire Dean Sinclair, Ph.D.
Ashley Smith, L.C.S.W.
Mindy Solomon, Ph.D.
Sally Tarbell, Ph.D.
Helen Thilly, L.C.S.W.
Sara Tlustos-Carter, Ph.D.
Holly Vause, A.P.P.M.H.N
Marianne Wamboldt, M.D.
Angela Ward, L.C.S.W.
Jason Williams, Ph.D.
Jason Willoughby, Ph.D.
Brennan Young, Ph.D.

Referring Clinicians

Kelly Bhatnagar, Ph.D.
Jeffery Dolgan, Ph.D.
Jennifer Eichberg, L.C.S.W.
Jennifer Hagman, M.D.
James Masterson, L.C.S.W.

Hy Gia Park, M.D.
Tami Roblek, Ph.D.
Wendy Smith, Ph.D.
David Williams, M.D.

Research Coordinators

Laurie Burnside, M.S.M., C.C.R.C., Lead Research Coordinator
Darci Anderson, B.A., C.C.R.C.
Heather Kennedy, M.P.H.
Amanda Millar, B.A.
Vanessa Waruinge, B.S.
Elizabeth Wallace, B.A.

Volunteer Research Assistants/Interns

Wisdom Amouzou
Ashley Anderson
Michael Richards
Mateya Whyte

Baystate Medical Center, Springfield, Massachusetts

Investigators

Bruce Waslick, M.D., Principal Investigator
Cheryl Bonica, Ph.D., Co-investigator
John Fanton, M.D., Co-investigator
Barry Sarvet, M.D., Co-investigator

Referring and Interviewing Clinicians

Julie Bermant, R.N., M.S.N., N.P.
Cheryl Bonica, Ph.D.
Jodi Devine, L.I.C.S.W.
William Fahey, Ph.D.
John Fanton, M.D.
Stephane Jacobus, Ph.D.

Barry Sarvet, M.D.
Peter Thunfors, Ph.D.
Bruce Waslick, M.D.
Vicki Weld, L.I.C.S.W.
Sara Wiener, L.I.C.S.W.
Shadi Zaghloul, M.D.

Referring Clinicians

Sarah Detenber, L.I.C.S.W.
Gordon Garrison, L.I.C.S.W.
Jacqueline Humpreys, L.I.C.S.W.
Noreen McGirr, L.I.C.S.W.
Sarah Marcotte, L.C.S.W.
Patricia Rogowski, R.N., C.N.S.

Research Coordinators

Julie Kingsbury, C.C.R.P., Lead Research Coordinator
Brenda Martin, B.A.

Volunteer Research Assistant/Intern

Liza Detenber

New York State Psychiatric Institute, New York, N.Y., Weill Cornell Medical College, Payne Whitney and Westchester Divisions, New York and White Plains, N.Y., and North Shore Child and Family Guidance Center, Roslyn Heights, N.Y. (Joint Study Site)

Investigator

Prudence W. Fisher, Ph.D., Principal Investigator

Research Coordinators

Julia K. Carmody, B.A., Lead Research Coordinator
Zvi R. Shapiro, B.A., Lead Research Coordinator

Volunteers

Preeya Desai
Samantha Keller
Jeremy Litfin, M.A.
Sarah L. Pearlstein, B.A.
Cedilla Sacher

New York State Psychiatric Institute

Referring and Interviewing Clinicians

Michele Cohen, L.C.S.W.
Eduvigis Cruz-Arrieta, Ph.D.
Miriam Ehrensaft, Ph.D.
Laurence Greenhill, M.D.
Schuyler Henderson, M.D., M.P.H.
Sharlene Jackson, Ph.D.
Lindsay Moskowitz, M.D.
Sweene C. Oscar, Ph.D.

Xenia Protopopescu, M.D.
James Rodriguez, Ph.D.
Gregory Tau, M.D.
Melissa Tebbs, L.C.S.W.
Carolina Velez-Grau, L.C.S.W.
Khadijah Booth Watkins, M.D.

Referring Clinicians

George Alvarado, M.D.
Alison Baker, M.D.
Elena Baron, Psy.D.
Lincoln Bickford, M.D., Ph.D.
Zachary Blumkin, Psy.D.

Colleen Cullen, L.C.S.W.
Chyristianne DeAlmeida, Ph.D.
Matthew Ehrlich, M.D.
Eve Friedl, M.D.
Clare Gaskins, Ph.D.
Alice Greenfield, L.C.S.W.
Liora Hoffman, M.D.
Kathleen Jung, M.D.
Karimi Mailutha, M.D., M.P.H.
Valentina Nikulina, Ph.D.
Tal Reis, Ph.D.
Moira A. Rynn, M.D.
Jasmine Sawhney, M.D.
Sarajbit Singh, M.D.
Katherine Stratigos, M.D.
Oliver Stroeh, M.D.
Russell Tobe, M.D.
Meghan Tomb, Ph.D.
Michelle Tricamo, M.D.

Research Coordinators

Angel A. Caraballo, M.D.
Erica M. Chin, Ph.D.
Daniel T. Chrzanowski, M.D.
Tess Dougherty, B.A.
Stephanie Hundt, M.A.
Moira A. Rynn, M.D.
Deborah Stedge, R.N.

Weill Cornell Medical College, Payne Whitney and Westchester Divisions

Referring and Interviewing Clinicians

Archana Basu, Ph.D.
Shannon M. Bennett, M.D.
Maria De Pena-Nowak, M.D.
Jill Feldman, L.M.S.W.
Dennis Gee, M.D.
Jo R. Hariton, Ph.D.
Lakshmi P. Reddy, M.D.
Margaret Yoon, M.D.

Referring Clinicians

Margo Benjamin, M.D.
Vanessa Bobb, M.D.
Elizabeth Bochtler, M.D.
Katie Cave, L.C.S.W.
Maalobeeka Gangopadhyay, M.D.
Jodi Gold, M.D.
Tejal Kaur, M.D.
Aaron Krasner, M.D.
Amy Miranda, L.C.S.W.
Cynthia Pfeffer, M.D.
James Rebeta, Ph.D.
Sharon Skariah, M.D.
Jeremy Stone, Ph.D.
Dirk Winter, M.D.

Research Coordinators

Alex Eve Keller, B.S., Lead Research Coordinator
Nomi Bodner (volunteer)
Barbara L. Flye, Ph.D.
Jamie S. Neiman (volunteer)
Rebecca L. Rendleman, M.D.

Referring and Interviewing Clinicians

Casye Brachfeld-Launer, L.C.S.W.
Susan Klein Cohen, Ph.D.
Amy Gelb, L.C.S.W.-R.
Jodi Glasser, L.C.S.W.
Elizabeth Goulding-Tag, L.C.S.W.
Deborah B. Kassimir, L.C.S.W.
Margo Posillico Messina, L.C.S.W.
Andréa Moullin-Heddle, L.M.S.W.
Lisa Pineda, L.C.S.W.
Elissa Smilowitz, L.C.S.W.

Referring Clinicians

Regina Barros-Rivera, L.C.S.W.-R. Assistant Executive Director
Maria Christiansen, B.S.
Amy Davies-Hollander, L.M.S.W.
Eartha Hackett, M.S.Ed., M.Sc., B.Sc.
Bruce Kaufstein, L.C.S.W.-R., Director of Clinical Services
Kathy Knaust, L.C.S.W.
John Levinson, L.C.S.W.-R., B.C.D.
Andrew Maleckoff, L.C.S.W., Executive Director/CEO
Sarah Rosen, L.C.S.W.-R., A.C.S.W.
Abigail Rothenberg, L.M.S.W.

Christine Scotten, A.C.S.W.
Michelle Spatano, L.C.S.W.-R.
Diane Straneri, M.S., R.N., C.S.
Rosara Torrisi, L.M.S.W.
Rob Vichnis, L.C.S.W.

Research Coordinators

Toni Kolb-Papetti, L.C.S.W.

Sheena M. Dauro (volunteer)

DSM-5 Field Trials Pilot Study, Johns Hopkins Medical Institution, Baltimore, Maryland

Adult Sample

Community Psychiatry Outpatient Program, Department of Psychiatry and Behavioral Sciences Main Campus

Investigators

Bernadette Cullen, M.B., B.Ch., B.A.O., Principal Investigator

Holly C. Wilcox, Ph.D., Principal Investigator

Referring and Interviewing Clinicians

Bernadette Cullen, M.B., B.Ch., B.A.O.

Shane Grant, L.C.S.W.-C.

Charee Green, L.C.P.C.

Emily Lorensen, L.C.S.W.-C.

Kathleen Malloy, L.C.P.C.

Gary Pilarchik, L.C.S.W.-C.

Holly Slater, L.C.P.C.

Stanislav Spivak, M.D.

Tarcia Spencer Turner, L.C.P.C.

Nicholas Seldes Windt, L.C.S.W.-C.

Research Coordinators

Mellisha McKitty, B.A.

Alison Newcomer, M.H.S.

Pediatric Sample

Child and Adolescent Outpatient Program, Department of Psychiatry and Behavioral Sciences Bayview Medical Center

Investigators

Joan P. Gerring, M.D., Principal Investigator
Leslie Miller, M.D., Principal Investigator
Holly C. Wilcox, Ph.D., Co-investigator

Referring and Interviewing Clinicians

Shannon Barnett, M.D.
Gwen Condon, L.C.P.C.
Brijan Fellows, L.C.S.W.-C.
Heather Garner, L.C.S.W.-C.
Joan P. Gerring, M.D.
Anna Gonzaga, M.D.
Debra Jenkins, L.C.S.W.-C.
Paige N. Johnston, L.C.P.C.
Brenda Memel, D.N.P., R.N.
Leslie Miller, M.D.
Ryan Moore, L.C.S.W.-C.
Shauna Reinblatt, M.D.
Monique Vardi, L.C.P.C.

Research Coordinators

Mellisha McKitty, B.A.
Alison Newcomer, M.H.S.

DSM-5 Field Trials in Routine Clinical Practice Settings: Collaborating Investigators

Archil Abashidze, M.D.
Francis R. Abueg, Ph.D.
Jennifer Louise Accuardi, M.S.
Balkozar S. Adam, M.D.

Miriam E. Adams, Sc.D., M.S.W., L.I.C.S.W.
Suzanna C. Adams, M.A.
Lawrence Adler, M.D.
Rownak Afroz, M.D.
Khalid I. Afzal, M.D.
Joseph Alimasuya, M.D.
Emily Allen, M.S.
Katherine A. Allen, L.M.F.T., M.A.
William D. Allen, M.S.
Jafar AlMashat, M.D.

998

Anthony T. Alonzo, D.M.F.T.
Guillermo Alvarez, B.A., M.A.
Angela Amoia-Lutz, L.M.F.T.
Krista A. Anderson, M.A., L.M.F.T.
Lisa R. Anderson, M.Ed., L.C.P.C.
Pamela M. Anderson, L.M.F.T.
Shannon N. Anderson, M.A., L.P.C., N.C.C.
Eric S. Andrews, M.A.
Vicki Arbuckle, M.S., Nursing(N.P.)
Namita K. Arora, M.D.
Darryl Arrington, M.A.
Bearlyn Y. Ash, M.S.
Wylie J. Bagley, Ph.D.
Kumar D. Bahl, M.D.
Deborah C. Bailey, M.A., M.S., Ph.D.
Carolyn Baird, D.N.P., M.B.A., R.N.-B.C., C.A.R.N.-A.P., I.C.C.D.P.D.
Joelle Bangsund, M.S.W.
Maria Baratta, M.S.W., Ph.D.

Stan Barnard, M.S.W.
Deborah Barnes, M.S.
Margaret L. Barnes, Ph.D.
David Barnum, Ph.D.
Raymond M. Baum, M.D.
Edward Wescott Beal, M.D.
Michelle Beaudoin, M.A.
Ernest E. Beckham, Ph.D.
Lori L. Beckwith, M.Ed.
Emmet Bellville, M.A.
Randall E. Bennett, M.A.
Lynn Benson, Ph.D.
Robert Scott Benson, M.D.
Linda Benton, M.S.W.
Ditza D. Berger, Ph.D.
Louise I. Bertman, Ph.D.
Robin Bieber, M.S., L.M.F.T.
Diana M. Bigham, M.A.
David R. Blackburn, Ph.D.
Kelley Blackwell, L.M.F.T.
Lancia Blatchley, B.A., L.M.F.T.
Stacey L. Block, L.M.S.W., A.C.S.W.
Karen J. Bloodworth, M.S., N.C.C., L.P.C.
Lester Bloomenstiel, M.S.
Christine M. Blue, D.O.
Marina Bluvshtein, Ph.D.
Callie Gray Bobbitt, M.S.W., L.C.S.W.
Moses L. Boone Jr., L.M.S.W., B.C.D.
Steffanie Boudreau-Thomas, M.A.-L.P.C.
Jay L. Boulter, M.A.
Aaron Daniel Bourne, M.A.

Helen F. Bowden, Ph.D.
Aryn Bowley-Safranek, B.S., M.S.
Elizabeth Boyajian, Ph.D.
Beth K. Boyarsky, M.D.
Gail M. Boyd, Ph.D.
Jeffrey M. Brandler, Ed.S., C.A.S., S.A.P.
Sandra L. Branton, Ed.D.
Karen J. Brocco-Kish, M.D.
Kristin Brooks, P.M.H.N.P.
Ann Marie Brown, M.S.W.
Philip Brown, M.S.W.
Kellie Buckner, Ed.S.
Richard Bunt, M.D.
Neil F. Buono, D.Min.
Janice Bureau, M.S.W., L.C.S.W.
Kimlee Butterfield, M.S.W.
Claudia Byrne, Ph.D.
Quinn Callicott, M.S.W., L.C.S.W.
Alvaro Camacho, M.D., M.P.H.
Sandra Cambra, Ph.D.
Heather Campbell, M.A.
Nancy Campbell, Ph.D., M.S.W.
Karen Ranee Canada, L.M.F.T.
Joseph P. Cannavo, M.D.
Catherine F. Caporale, Ph.D.
Frederick Capps, Ph.D., M.S.
Rebecca J. Carney, M.B.A., M.A., L.M.H.C.
Kelly J. Carroll, M.S.W.
Richard W. Carroll, Ph.D., L.P.C., A.C.S.
Sherry Casper, Ph.D.
Joseph A. Catania, L.I.S.W.S., L.C.D.C. III

Manisha P. Cavendish, Ph.D.
Kenneth M. Certa, M.D.
Shambhavi Chandraiah, M.D.
Calvin Chatlos, M.D.
Daniel C. Chen, M.D.
Darlene Cheryl, M.S.W.
Matthew R. Chirman, M.S.
Carole A. Chisholm, M.S.W.
Shobha A. Chottera, M.D.
Joseph Logue Christenson, M.D.
Pamela Christy, Psy.D.
Sharon M. Freeman Clevenger, Ph.D., P.M.H.C.N.S.-B.C.
Mary Ann Cohen, M.D.
Mitchell J. Cohen, M.D.
Diego L. Coira, M.D.
Melinda A. Lawless Coker, Psy.D.
Carol Cole, M.S.W., L.C.S.W.
Caron Collins, M.A., L.M.F.T.
Wanda Collins, M.S.N.
Linda Cook Cason, M.A.
Ayanna Cooke-Chen, M.D., Ph.D.
Heidi B. Cooperstein, D.O.
Ileana Corbelle, M.S.W.
Kimberly Corbett, Psy.D.
Angelina Cordova, M.A.Ed.
Jennifer Carol Cox, L.P.C.
Sheree Cox, M.A., R.N., N.C.C., D.C.C., L.M.H.C.
William Frederick Cox, M.D.
Sally M. Cox, M.S.Ed.
Debbie Herman Crane, M.S.W.
Arthur Ray Crawford III, Ph.D.

Roula Creighton, M.D.
John R. Crossfield, L.M.H.C.

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Sue Cutbirth, R.N., M.S.N, C.S., P.M.H.N.P.
Marco Antonio Cuyar, M.S.
Rebecca Susan Daily, M.D.
Lori S. Danenberg, Ph.D.
Chan Dang-Vu, M.D.
Mary Hynes Danielak, Psy.D.
Cynthia A. Darby, M.Ed., Ed.S.
Douglas Darnall, Ph.D.
Christopher Davidson, M.D.
Doreen Davis, Ph.D., L.C.S.W.
Sandra Davis, Ph.D., L.M.H.C., N.C.C.
Walter Pitts Davis, M.Th.
Christian J. Dean, Ph.D.
Kent Dean, Ph.D.
Elizabeth Dear, M.A.
Shelby DeBause, M.A.
Rebecca B. DeLaney, M.S.S.W., L.C.S.W., B.C.D.
John R. Delatorre, M.A.
Frank DeLaurentis, M.D.
Eric Denner, M.A., M.B.A.
Mary Dennihan, L.M.F.T.
Kenny Dennis, M.A.
Pamela L. Detrick, Ph.D., M.S., F.N.P.-B.C., P.M.H.N.P.-B.C., R.N.-B.C., C.A.P., G.C.A.C.
Robert Detrinis, M.D.
Daniel A. Deutschman, M.D.
Tania Diaz, Psy.D.

Sharon Dobbs, M.S.W., L.C.S.W.
David Doreau, M.Ed.
Gayle L. Dosher, M.A.
D'Ann Downey, Ph.D., M.S.W.
Beth Doyle, M.A.
Amy J. Driskill, M.S., L.C.M.F.T.
James Drury, M.D.
Brenda-Lee Duarte, M.Ed.
Shane E. Dulemba, M.S.N.
Nancy R. G. Dunbar, M.D.
Cathy Duncan, M.A.
Rebecca S. Dunn, M.S.N., A.R.N.P.
Debbie Earnshaw, M.A.
Shawna Eddy-Kissell, M.A.
Momen El Nesr, M.D.
Jeffrey Bruce Elliott, Psy.D.
Leslie Ellis, Ph.D.
Donna M. Emfield, L.C.P.C.
Gretchen S. Enright, M.D.
John C. Espy, Ph.D.
Renuka Evani, M.B.B.S., M.D.
Heather Evans, M.S.Ed., L.P.C.N.C.C.
Cesar A. Fabiani, M.D.
Fahim Fahim, M.D.
Samuel Fam, M.D.
Edward H. Fankhanel, Ph.D., Ed.D.
Tamara Farmer, M.S.N., A.R.N.P.
Farida Farzana, M.D.
Philip Fast, M.S.
Patricia Feltrup-Exum, M.A.M.F.T.
Hector J. Fernandez-Barillas, Ph.D.

Julie Ferry, M.S.W., L.I.C.S.W.
Jane Fink, Ph.D., M.S.S.A.
Kathy Finkle, L.P.C.M.H.
Steven Finlay, Ph.D.
Rik Fire, M.S.W., L.C.S.W.
Ann Flood, Ph.D.
Jeanine Lee Foreman, M.S.
Thyra Fossum, Ph.D.
Karen S. Franklin, L.I.C.S.W.
Sherre K. Franklin, M.A.
Helen R. Frey, M.A., E.D.
Michael L. Freytag, B.S., M.A.
Beth Gagnon, M.S.W.
Patrice L.R. Gallagher, Ph.D.
Angela J. Gallien, M.A.
Robert Gallo, M.S.W.
Mario Galvarino, M.D.
Vladimir I. Gasca, M.D.
Joshua Gates, Ph.D.
Anthony Gaudioso, Ph.D.
Michelle S. Gauthier, A.P.R.N., M.S.N, P.M.H.N.P.-B.C.
Rachel E. Gearhart, L.C.S.W.
Stephen D. Gelfond, M.D.
Nancy S. Gerow, M.S.
Michael J. Gerson, Ph.D.
Susan M. A. Geyer, L.M.S.W.
Lorrie Gfeller-Strouts, Ph.D.
Shubu Ghosh, M.D.
Richard Dorsey Gillespie, M.Div.
Stuart A. Gitlin, M.S.S.A.
Jeannette E. Given, Ph.D.

Frances Gizzi, L.C.S.W.
Stephen I. Glicksman, Ph.D.
Martha Glisky, Ph.D.
Sonia Godbole, M.D.
Howard M. Goldfischer, Psy.D.
Mary Jane Gonzalez-Huss, Ph.D.
Michael I. Good, M.D.
Dawn Goodman-Martin, M.A.-L.M.H.C.
Robert Gorkin, Ph.D., M.D.
Jeff Gorski, M.S.W.
Linda O. Graf, M.Ed., L.C.P.C.
Ona Graham, Psy.D.
Aubrie M. Graves, L.M.S.W., C.A.S.A.C.
Howard S. Green, M.D.
Karen Torry Green, M.S.W.
Gary Greenberg, Ph.D.
Marjorie Greenhut, M.A.
James L. Greenstone, Ed.D., J.D.
Raymond A. Griffin, Ph.D.
Joseph Grillo, Ph.D.
Janeane M. Grisez, A.A., B.A.
Lawrence S. Gross, M.D.
Robert J. Gross, M.D.
Sally J. Grosscup, Ph.D.
Philip A. Grossi, M.D.

Gabrielle Guedet, Ph.D.
Nicholas Guenzel, B.A., B.S., M.S.N.
Mary G. Hales, M.A.

Tara C. Haley, M.S., L.M.F.T.
John D. Hall, M.D.
Amy Hammer, M.S.W.
Michael S. Hanau, M.D.
Linda K.W. Hansen, M.A., L.P.
Genevieve R. Hansler, M.S.W.
Mary T. Harrington, L.C.S.W.
Lois Hartman, L.C.P.C.
Steven Lee Hartsock, Ph.D., M.S.W.
Victoria Ann Harwood, M.S.W., L.C.S.W.
Rossi A. Hassad, Ph.D., M.P.H.
Erin V. Hatcher, M.S.N.
Richard L. Hauger, M.D.
Kimberly M. Haverly, M.A.
Gale Eisner Heater, M.S., M.F.T.
Katlin Hecox, M.A.
Brenda Heideman, M.S.W.
Melinda Heinen, M.Sc.
Marie-Therese Heitkamp, M.S.
Melissa B. Held, M.A.
Jessica Hellings, M.D.
Bonnie Helmick-O'Brien, M.A., L.M.F.T.
MaLinda T. Henderson, M.S.N., F.P.M.H.N.P.
Gwenn Herman, M.S.W.
Martha W. Hernandez, M.S.N., A.P.R.N., P.M.H.C.N.S.
Robin L. Hewitt, M.S.
Kenneth Hoffman, Ph.D.
Patricia E. Hogan, D.O.
Peggy Holcomb, Ph.D.
Garland H. Holloman Jr., M.D.
Kimberly Huegel, M.S.W., L.C.S.W.

Jason Hughes, L.P.C.-S., N.C.C.
Jennifer C. Hughes, Ph.D., M.S.W., L.I.S.W.-S.
Michelle K. Humke, M.A.
Judith G. Hunt, L.M.F.T.
Tasneem Hussainee, M.D.
Sharlene J. Hutchinson, M.S.N.
Muhammad Ikram, M.D.
Sunday Ilechukwu, M.D., D.Psy. Cli.
Douglas H. Ingram, M.D.
Marilynn Irvine, Ph.D.
Marjorie Isaacs, Psy.D.
Raymond Isackila, Ed.S., P.C.C.-S., L.I.C.D.C.
Mohammed A. Issa, M.D.
John L. Jankord, M.A.
Barbara P. Jannah, L.C.S.W.
C. Stuart Johnson, M.S.
Dawn M. Johnson, M.A.
Deanna V. Johnson, M.S., A.P.R.N., B.C.
Eric C. Johnson, M.F.T.
Joy Johnson, Ph.D., L.C.S.W.
Willard Johnson, Ph.D.
Xenia Johnson-Bhembe, M.D.
Vann S. Joines, Ph.D.
Margaret Jones, Psy.D.
Patricia Jorgenson, M.S.W.
Steven M. Joseph, M.D.
Taylere Joseph, M.A.
Jeanette M. Joyner-Craddock, M.S.S.W.
Melissa Kachapis, M.A.
Charles T. Kaelber, M.D.
Aimee C. Kaempf, M.D.

Peter Andrew Kahn, M.D.
Robert P. Kahn-Rose, M.D.
Maher Karam-Hage, M.D.
Todd H. Kasdan, M.D.
Karen Kaufman, M.S., L.M.F.T.
Rhesa Kaulia, M.A., M.F.T.
Debbie Lynn Kelly, M.S.N, P.M.H.N.P.-B.C.
W. Stephen Kelly, Ph.D.
Selena Kennedy, M.A.
Judith A. Kenney, M.S., L.P.C.
Mark Patrick Kerekes, M.D.
Alyse Kerr, M.S., N.C.C., N.A.D.D.-C.C., L.P.C.
Karen L. Kerschmann, L.C.S.W.
Marcia Kesner, M.S.
Ashan Khan, Ph.D.
Shaukat Khan, M.D.
Audrey Khatchikian, Ph.D.
Laurie B. Kimmel, M.S.W.
Jason H. King, Ph.D.
Nancy Leigh King, M.S.W., L.C.S.W., L.C.A.S.
Kyle Kinne, M.S.C
Cassandra M. Klyman, M.D.
David R. Knapp, L.C.S.W.
Margaret Knerr, M.S.
Michael R. Knox, Ph.D.
Carolyn Koblin, M.S.
Valerie Kolbert, M.S., A.R.N.P.-B.C.
Heather Koontz, M.S.W.
Faye Koop, Ph.D., L.C.M.F.T.
Fern M. Kopakin, M.S.W., L.C.S.W.
Joel Kotin, M.D.

Sharlene K. Kraemer, M.S.E.
Marjorie Vego Krausz, M.A., Ed.D.
Nancy J. Krell, M.S.W.
Mindy E. Kronenberg, Ph.D.
Dwayne Kruse, M.S., M.F.T.
Ajay S. Kuchibhatla, M.D.
Shubha N. Kumar, M.D.
Helen H. Kyomen, M.D., M.S.
Rebecca M. Lachut, M.Ed., Ed.S.
Alexis Lake, M.S.S.
Ramaswamy Lakshmanan, M.D.
Brigitta Lalone, L.C.S.W.-R
John W. Lancaster, Ph.D.
Patience R. Land, L.I.C.S.W., M.S.W., M.P.A.
Amber Lange, M.A., Ph.D.
Jeff K. Larsen, M.A.
Nathan E. Lavid, M.D.
Michelle Leader, Ph.D.
Stephen E. Lee, M.D.

Cathryn L. Leff, Ph.D., L.M.F.T.
Rachael Kollar Leombruno, L.M.F.T.
Arlene I. Lev, M.S.W., L.C.S.W.-R
Gregory K. Lewis, M.A.-L.M.F.T.
Jane Hart Lewis, M.S.
Melissa S. Lewis, M.S.W., L.I.C.S.W.
Norman Gerald Lewis, F.R.A.N.Z.C.P.
Robin Joy Lewis, Ph.D.
Ryan Michael Ley, M.D.

Tammy R. Lias, M.A.
Russell F. Lim, M.D.
Jana Lincoln, M.D.
Ted Lindberg, L.M.S.W., L.M.F.T., M.S.W.
Peggy Solow Liss, M.S.W.
Andrea Loeb, Psy.D.
William David Lohr, M.D.
Mary L. Ludy, M.A., L.M.H.C., L.M.F.T.
Nathan Lundin, M.A., L.P.C.
Veena Luthra, M.D.
Patti Lyerly, L.C.S.W.
Denise E. Maas, M.A.
Silvia MacAllister, L.M.F.T.
Nicola MacCallum, M.S., M.F.C. Therapy
Colin N. MacKenzie, M.D.
Cynthia Mack-Ernsdorff, Ph.D.
John R. Madsen-Bibeau, M.S., M.Div
Christopher J. Maglio, Ph.D.
Deepak Mahajan, M.D.
Debra Majewski, M.A.
Harish Kumar Malhotra, M.D.
Pamela Marcus, R.N., M.S.
Mary P. Marshall, Ph.D.
Flora Lynne Martin, M.A., L.P.C., A.D.C.
Robert S. Martin, M.D.
Jennifer L. Martinez, M.S.
Ninfa Martinez-Aguilar, M.A., M.F.T.
Emily Martinsen, M.S.W.
Farhan A. Matin, M.D.
Janus Maybee, P.M.H.N.P.
Karen Mazarin-Stanek, M.A.

Eben L. McClenahan, M.D., M.S.
Jerlyn C. McCleod, M.D.
Susan E. McCue, M.S.W., L.C.S.W.
Kent D. McDonald, M.S.
Daniel McDonnell, M.S.N, P.M.H.-N.P.
Robert McElhose, Ph.D.
Lisa D. McGrath, Ph.D.
Mark McGrosky, M.S.W.
Katherine M. McKay, Ph.D.
Darren D. McKinnis, M.S.W.
Mona McNelis-Broadley, M.S.W., L.C.S.W.
Rick McQuistion, Ph.D.
Susan Joy Mendelsohn, Psy.D.
Barbara S. Menninga, M.Ed.
Hindi Mermelstein, M.D., F.A.P.M.
Rachel B. Michaelsen, M.S.W.
Thomas F. Micka, M.D.
Tonya Miles, Psy.D.
Matthew Miller, M.S.
Michael E. Miller, M.D.
Noel Miller, L.M.S.W., M.B.A., M.P.S.
Kalpana Miriyala, M.D.
Sandra Moenssens, M.S.
Erin Mokhtar, M.A.
Robert E. Montgomery, M.Ed.
Susan Moon, M.A.
Theresa K. Moon, M.D.
David B. Moore, B.A., M.Div., M.S.S.W., Ph.D.
Joanne M. Moore, M.S.
Peter I. M. Moran, M.B.B.Ch.
Anna Moriarty, M.P.S., L.P.C., L.M.H.C.

Richard Dean Morris, M.A.
Michael M. Morrison, M.A.
Carlton E. Munson, Ph.D.
Timothy A. Murphy, M.D.
Beth L. Murphy, Psy.D.
Melissa A. Myers, M.D.
Stefan Nawab, M.D.
Allyson Matney Neal, D.N.P.
Steven Nicholas, M.A.
Aurelian N. Niculescu, M.D.
Earl S. Nielsen, Ph.D.
Terry Oleson, Ph.D.
Julianne R. Oliver, B.S., M.S., Ph.D.
Robert O. Olsen, M.D.
Amy O'Neill, M.D.
Oscar H. Oo, Psy.D., A.B.P.P.
Laurie Orlando, J.D., M.A.
Jill Osborne, M.S., Ed.S.
Kimberly Overlie, M.S.
L. Kola Oyewumi, Ph.D.
Zachary J. Pacha, M.S.W.
Suzette R. Papadakis, M.S.
Amanda C. Parsons, M.A., L.P.C.C.
Lee R. Pate, B.A., M.A.
Eric L. Patterson, L.P.C.
Sherri Paulson, M.Ed., L.S.C.W.
Peter Dennis Pautz, B.A., M.S.W.
Jeanette Pelton, L.I.S.W.-S.U.P.V.
Malinda J. Perkins, M.S.W., L.C.S.W.
Eleanor F. Perlman, M.S.W.
Deborah K. Perry, M.S.W.

Amanda Peterman, L.M.F.T.
Shawn Pflugardt, Psy.D.
Robert J. Dean Phillips, M.S.
Laura Pieper, M.S.W., L.C.S.W.
Lori D. Pink, M.S.W., B.C.D
Michael G. Pipich, M.S., L.M.F.T.
Cynthia G. Pizzulli, M.S.W., Ph.D.
Kathy C. Points, M.A.
Marya E. Pollack, M.D., M.P.H.
Sanford E. Pomerantz, M.D.
Eva Ponder, M.S.W., Psy.D.
Ernest Poortinga, M.D.
David Post, M.D.
Laura L. Post, M.D., Ph.D., J.D.

Patrick W. Powell, Ed.D.
Beth M. Prewett, Psy.D.
Robert Price, D.C.C., M.Ed.
John Pruett, M.D.
Aneita S. Radov, M.A.
Dawn M. Raffa, Ph.D.
Kavitha Raja, M.D.
Ranjit Ram, M.D.
Mohamed Ibrahim Ramadan, M.D., M.S.
Christopher S. Randolph, M.D.
Nancy Rappaport, M.Ed.
John Moir Rauenhorst, M.D.
Laurel Jean Rebenstock, L.M.S.W.
Edwin Renaud, Ph.D.

Heather J. Rhodes, M.A.
Jennifer S. Ritchie-Goodline, Psy.D.
Daniel G. Roberts, M.A.
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Michelle Voegels, B.S.N., M.S.N., B.C.
Wess Vogt, M.D.
R. Christopher Votolato, Psy.D.

1003

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Dominique Walmsley, M.A.
Bhupinder Singh Waraich, M.D.
Joseph Ward, N.C.C., L.P.C. M.Ed.
Robert Ward, M.S.W.
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Michele Zimmerman, M.A., P.M.H.C.N.S.-B.C.
Judith A. Zink, M.A.

Vanderbilt University REDCap Team

Paul Harris, Ph.D.
Sudah Kashyap, B.E.
Brenda Minor
Jon Scherdin, M.A.
Rob Taylor, M.A.
Janey Wang, M.S.

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